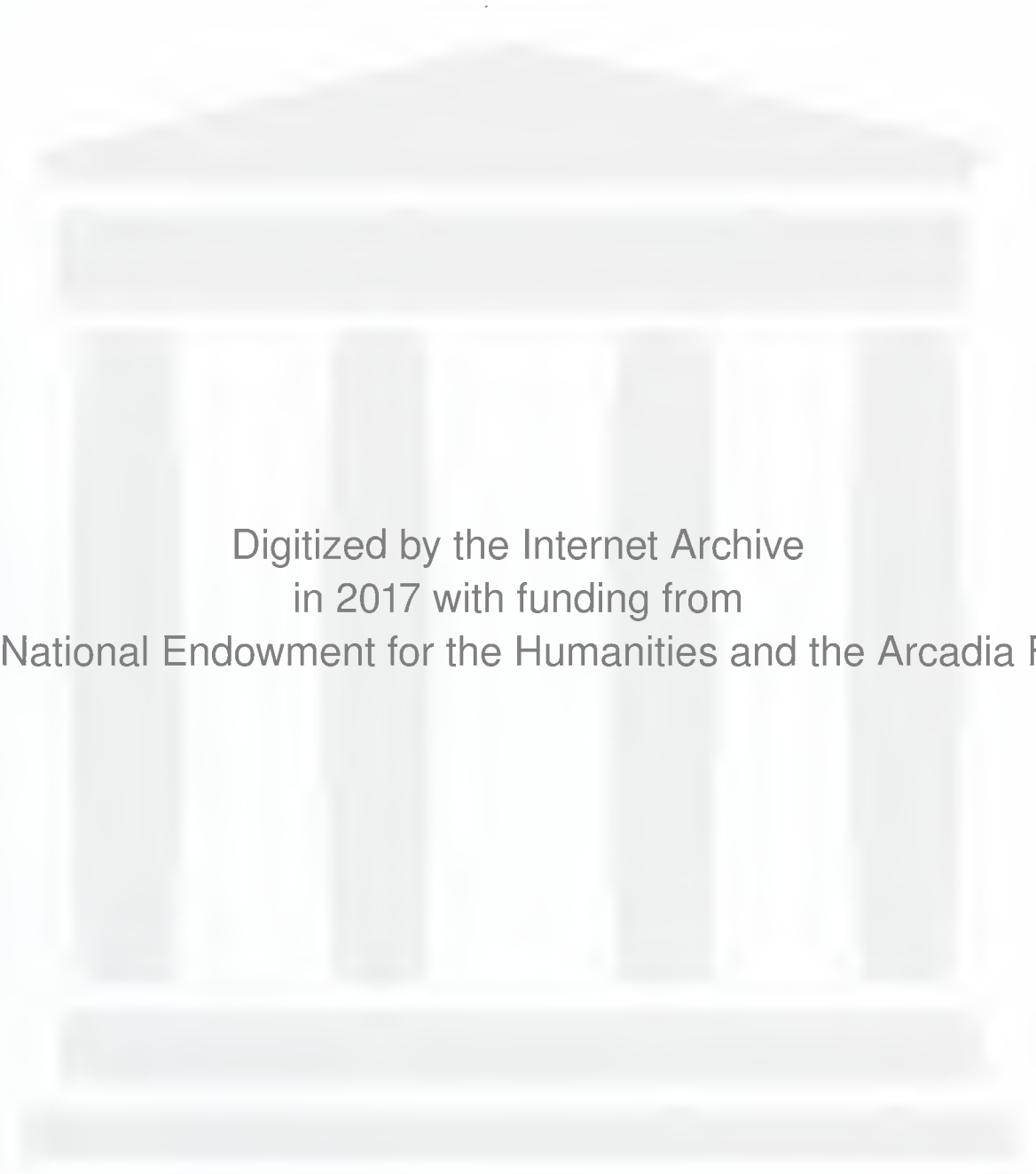

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Texas State Journal of Medicine

MAY 1966

TEXAS MEDICINE

IN THIS ISSUE

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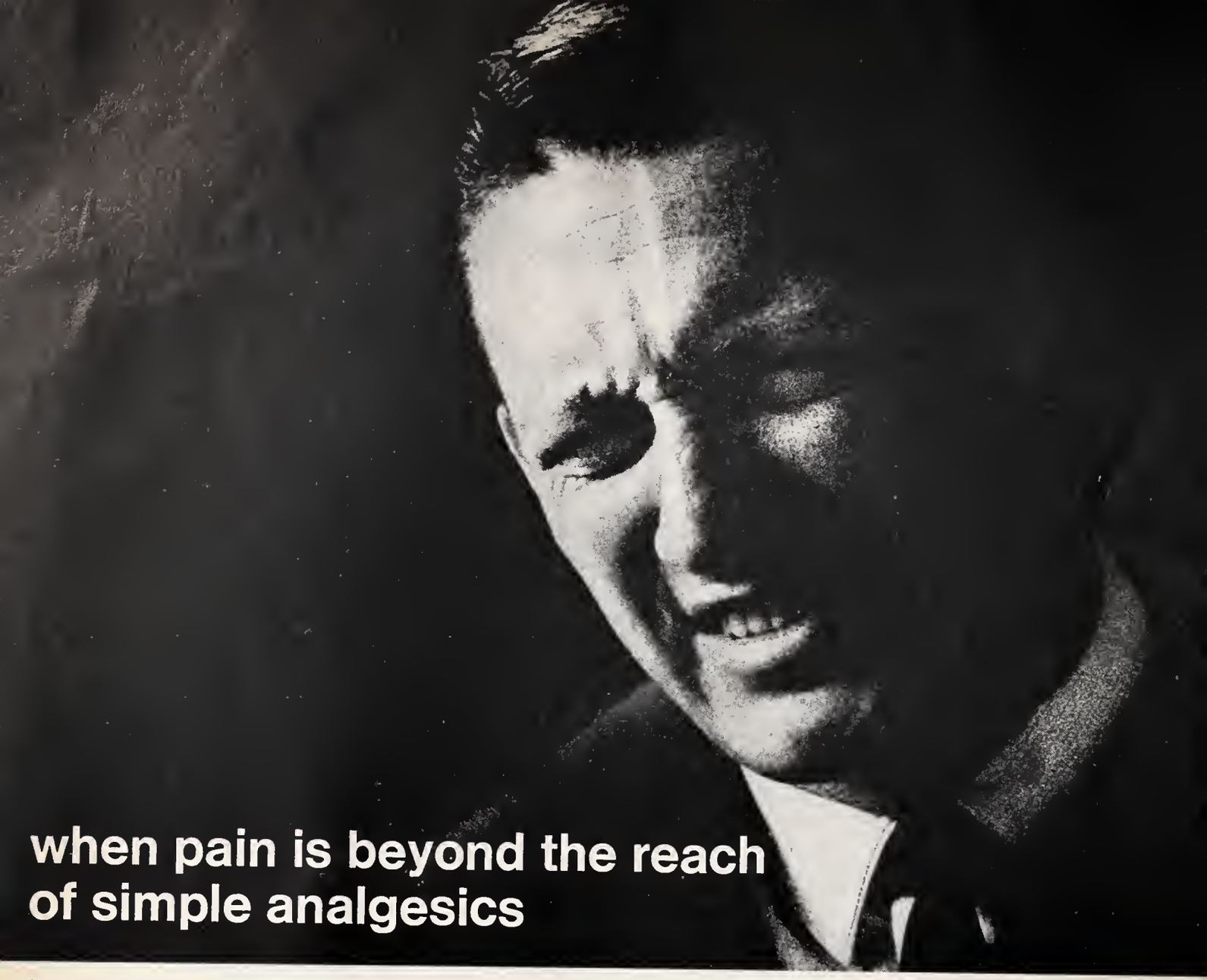

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1. Collective studies, Department of Medical Research, Winthrop Laboratories.

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48 TUMOR SEMINAR
Robert C. Horn, Jr.

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Case 4. Malignant lymphoma of stomach

Case 5. Rhabdomyosarcoma (mixed pattern)

Case 6. Granular cell myoblastoma

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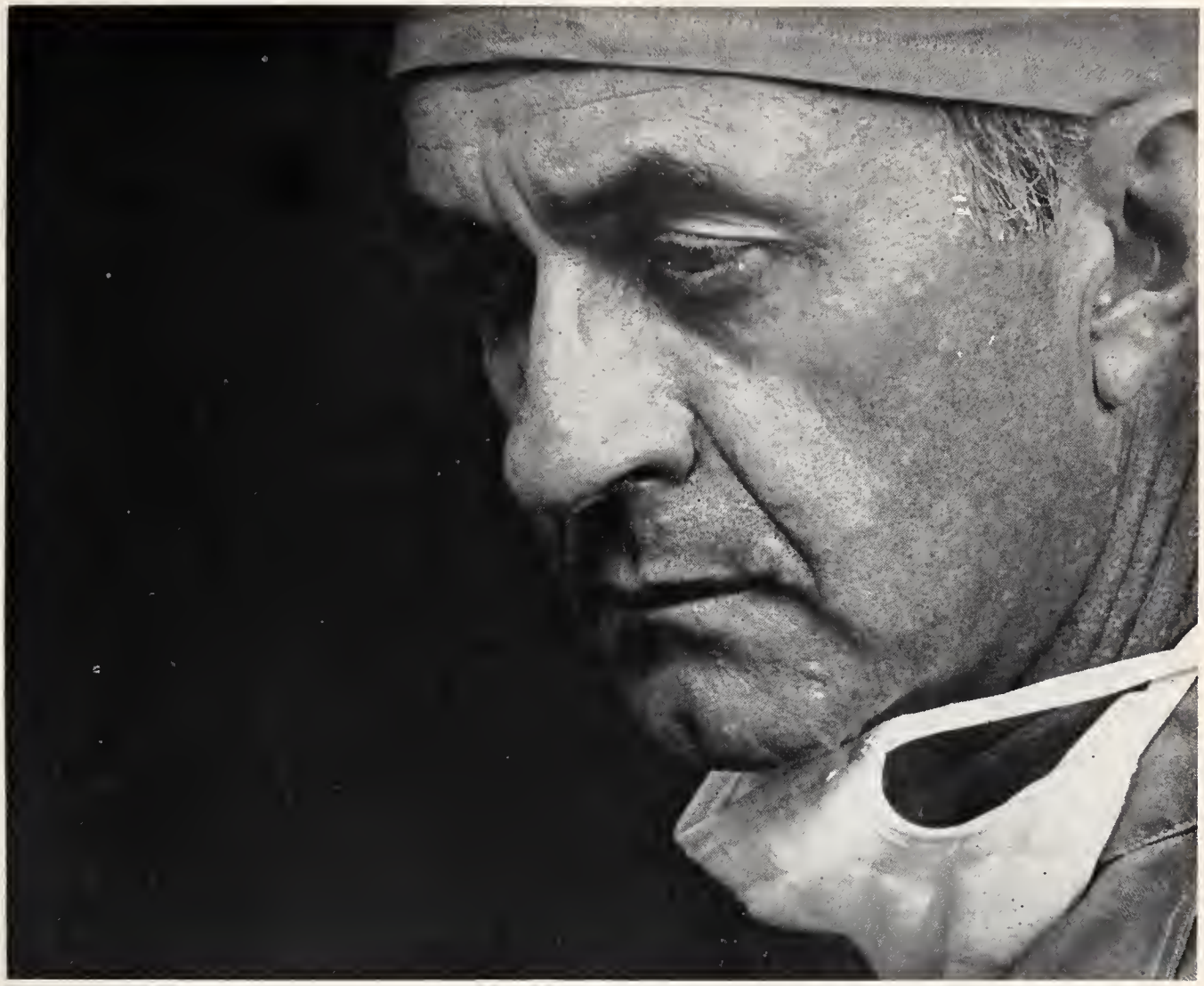

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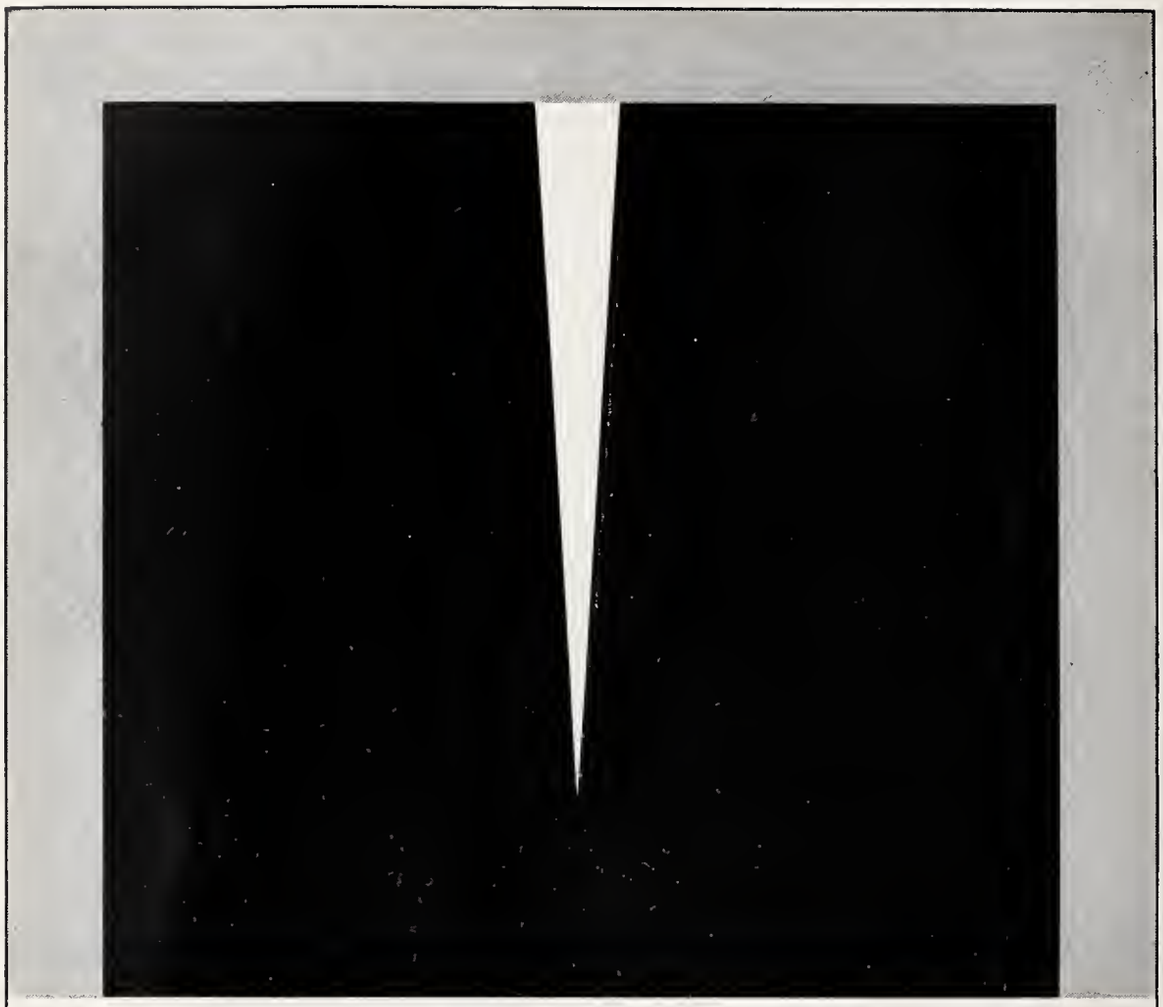

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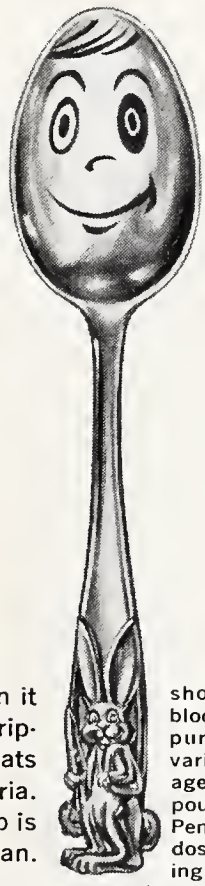

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Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

One instance each of pancytopenia and leukopenia, occurring when carisoprodol was administered with other drugs, has been reported, as has an instance of fixed drug eruption with carisoprodol and subsequent cross reaction to meprobamate. Rare allergic reactions, usually mild, have included one case each of anaphylactoid reaction with mild shock and angioneurotic edema with respiratory difficulty, both reversed with appropriate therapy. In cases of allergic or hypersensitivity reactions, carisoprodol should be discontinued and appropriate therapy initiated. Suicidal attempts may produce coma and/or mild shock and respiratory depression.

Dosage: Usual adult dose is one 350 mg. tablet three times daily and at bedtime.

Supplied: Two Strengths: 350 mg. white tablets and 250 mg. orange, two-piece capsules.

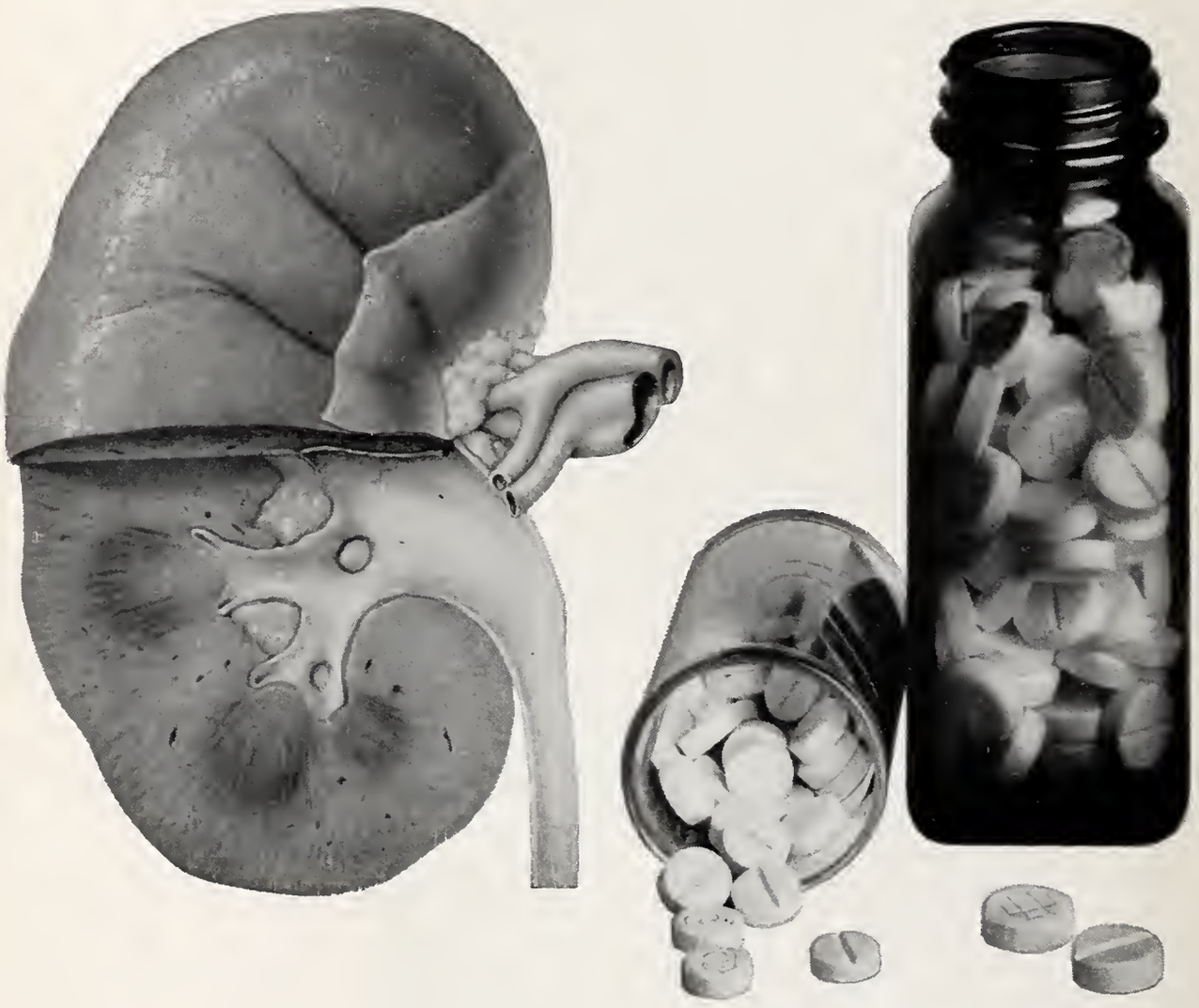
Before prescribing, consult package circular.

for the relief of low back sprains and strains

SOMA®
(CARISOPRODOL)

Wallace Laboratories, Cranbury, N.J.
26S01J

at Merck Sharp & Dohme...



understanding... precedes development

The development of chlorothiazide and probenecid were events of major importance, but perhaps even more important for the future was the Renal Research Program by which they were developed. When Merck Sharp & Dohme organized this program in 1943, it was expressing in action some of its basic beliefs about research:

- Many problems connected with renal structure and function were still undefined or unsolved. The Renal Research Program would begin its basic research in some of these problem areas.
- From knowledge thus acquired might come clues to the development of new therapeutic agents of significant value to the physician.

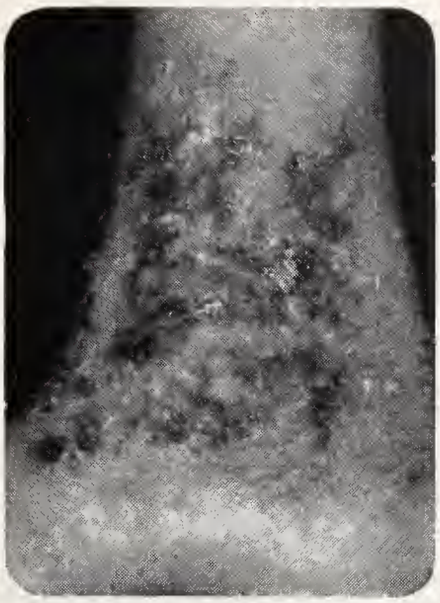
For example, the Renal Research Program put fifteen years into this search before chlorothiazide became available. But because these years had first led to a greater understanding of basic problems, the desired criteria for chlorothiazide existed before the drug was developed.

Along with other research teams at Merck Sharp & Dohme, the Renal Research Program continues to add new understanding of basic problems—understanding which will lead to important new therapeutic agents.

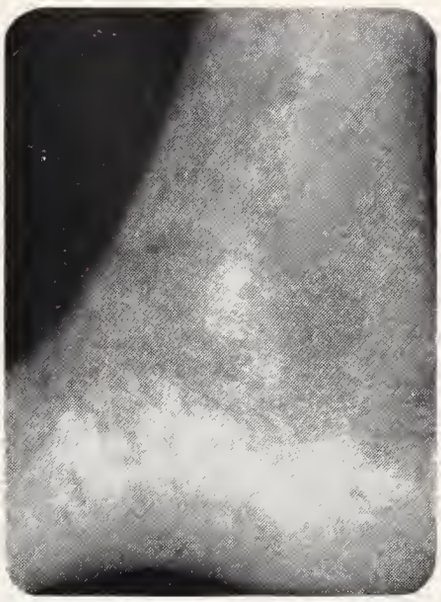
MERCK SHARP & DOHME Division of Merck & Co., Inc., West Point, Pa.

where today's theory is tomorrow's therapy

eczema: scourge of childhood



R. R., Age 11—Before treatment—atopic eczema of long standing



After treatment—with ARISTOCORT Topical Ointment 0.1% for two weeks

ARISTOCORT® Triamcinolone Acetonide Topicals have proved exceptionally effective in the control of various forms of childhood eczema: allergic, atopic, nummular, psoriatic, and mycotic.

In most cases responsive to topical ARISTOCORT, the 0.1% concentration is sufficiently potent. The 0.5% concentration provides enhanced topical activity for patients requiring additional potency for proper relief.

Administration and Dosage: Apply sparingly to the affected area 3 or 4 times daily. Some cases of psoriasis may be more effectively treated if the 0.1% Cream or Ointment is applied under an occlusive dressing.

Contraindications: Tuberculosis of the skin, herpes simplex, chicken pox and vaccinia.

Precautions and Side Effects: Do not use in the eyes or in the ear (if drum is perforated). A few individuals react unfavorably under certain conditions. If side effects are encountered, the drug should be discontinued and appropriate measures taken. Use on infected areas should be attended with caution and observation, bearing in mind the potential spreading of infection and the advisability of discontinuing therapy and/or initiating antibacterial measures. Generalized dermatological conditions may require systemic corticosteroid therapy. Steroid therapy, although responsible for remissions of dermatoses, especially of allergic origin cannot be expected to prevent recurrence. The use over extensive body areas, with or without occlusive nonpermeable dressings, may result in systemic absorption. Appropriate precautions should be taken. When occlusive nonpermeable dressings are used, miliaria, folliculitis and pyodermas will sometimes develop. Localized atrophy and striae have been reported with the use of steroids by the occlusive technique. When occlusive nonpermeable dressings are used, the physician should be aware of the hazards of suffocation and flammability. The safety of use on pregnant patients has not been firmly established. Thus, do not use in large amounts or for long periods of time on pregnant patients.

Packages: Tubes of 5 Gm. and 15 Gm.; ½ lb. jar.

PHOTOGRAPHS COURTESY OF M. M. NIERMAN, M.D.

Aristocort® Topical Ointment 0.1% and Cream 0.1%, 0.5%
Triamcinolone Acetonide

Also available in foam form and with neomycin.

Lederle LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

618-8

TOPICAL TREATMENT

TYPICAL RESULTS

PRIMARY PYODERMA

AFTER TREATMENT WITH
'NEOSPORIN' ANTIBIOTIC OINTMENT
AND SALINE COMPRESSES

with 'NEOSPORIN'® brand

Polymyxin B-Neomycin-Bacitracin OINTMENT

Each gram contains:

'Aerosporin'® brand Polymyxin B Sulfate ..... 5,000 Units

Zinc Bacitracin ..... 400 Units

Neomycin Sulfate (equivalent to 3.5 mg. Neomycin Base) ..... 5 mg.

Tubes of ½ oz. and 1 oz.

- clinically effective
- comprehensive bactericidal action against most Gram-negative and Gram-positive organisms, including Pseudomonas
- rarely sensitizes

For the eradication of infectious organisms in a wide range of dermatologic disorders: impetigo, ecthyma, pyodermas, sycosis vulgaris, paronychia, traumatic lesions, eczema, herpes and seborrheic dermatitis. Prophylactically, for protection against bacterial contamination in burns, skin grafts, incisions and other clean lesions, abrasions and minor cuts and wounds.

Caution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs.

Contraindication: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.

Complete literature available on request from Professional Services Dept. PML.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York

Lutrexin®

HW&D BRAND OF LUTUTRIN

3000 UNIT TABLETS

IN THE TREATMENT OF FUNCTIONAL DYSMENORRHEA AND SELECTED CASES OF PREMATURE LABOR AND 2ND AND 3RD TRIMESTER THREATENED ABORTION

In controlling abnormal uterine activity, LUTREXIN, the non-steroid "uterine relaxing factor" has been found to be the drug of choice by many clinicians.

No side effects have been reported, even when massive doses (25 tablets per day) were administered.

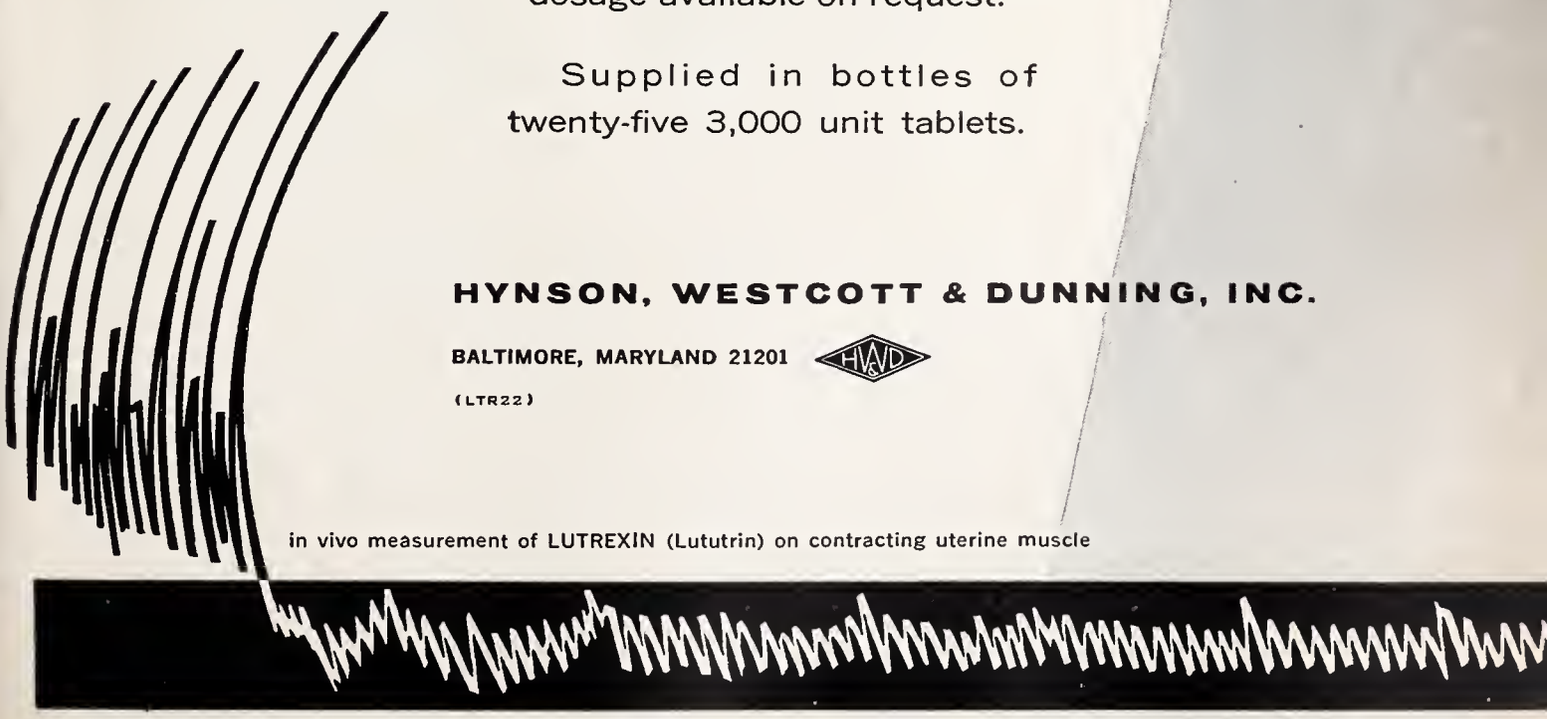
Literature on indications and dosage available on request.

Supplied in bottles of twenty-five 3,000 unit tablets.

HYNSON, WESTCOTT & DUNNING, INC.

BALTIMORE, MARYLAND 21201

(LTR22)



in vivo measurement of LUTREXIN (Lututrin) on contracting uterine muscle

Tareyton...with the taste worth fighting for

America's largest-selling charcoal-tip cigarette

©The American Tobacco Company

ECONOMY

When you prescribe or recommend Allbee with C, you can be sure your patient is getting a rational, specific multivitamin formulation at an economical price. The potent formula is sensible and simple. It contains therapeutic amounts of the water-soluble B and C vitamins. These vitamins are expended rapidly in the body and need to be replenished frequently. There are no extraneous factors, no frills in Allbee with C. It's the no-nonsense vitamin in the yellow and green capsule that always gives your patient his money's worth.

Each capsule contains: Thiamine mononitrate (B_1), 15 mg.; Riboflavin (B_2), 10 mg.; Pyridoxine hydrochloride (B_6), 5 mg.; Nicotinamide, 50 mg.; Calcium pantothenate, 10 mg.; Ascorbic acid (vitamin C), 300 mg.

A. H. ROBINS COMPANY, INC.,
RICHMOND, VIRGINIA 23220 A·H·ROBINS

—a good reason for AHR

ALLBEE® WITH C

THERE'S NOTHING
LIKE A VACATION*
FOR RELAXING
STRESS-INDUCED
SMOOTH MUSCLE
SPASM . . .

. . NOTHING, THAT IS,
EXCEPT THE SEDATIVE-ANTISPASMODIC
BENEFITS OF

DONNATAL®

There's nothing quite like a vacation to ease the pressures of the modern, "workingday" world. And for the patient who can't get away from it all, there's nothing quite like Donnatal to relax stress-induced smooth muscle spasm. For 31 years it has been the antispasmodic-sedative most often prescribed for relieving functional disturbances of tone and motility of the gastrointestinal tract.

belladonna alkaloids in optimally balanced ratio

In Donnatal, natural belladonna alkaloids are rationally balanced in a specific, fixed ratio that provides "the greatest efficacy with the smallest possible dose."[1] They avoid the clinical uncertainties of the variable tincture and extract of belladonna, and are considered superior in range of action to atropine alone.[2] Furthermore, they are generally recognized as being more effective than the synthetics for relieving visceral spasm.

phenobarbital for sedation

Years of clinical use have established phenobarbital as one of the most efficient and highly regarded sedatives. In fact, for general sedation it is the drug of choice.[3] In Donnatal, phenobarbital potentiates the spasmolytic effects of the belladonna alkaloids, lessening emotional tensions and checking the neurogenic impulses that trigger GI disorders.

more than 24 indications in PDR

Donnatal has withstood the test of time to become the classic sedative-antispasmodic because of its unsurpassed effectiveness, safety, economy, uniformity of composition, and dosage convenience. Its widespread acceptance and usage by the profession can also be attributed to its versatility in treating disorders characterized by smooth muscle spasm. There are more than two dozen distinct and separate indications for Donnatal listed in the current PDR.

IN EACH TABLET, CAPSULE, OR (5 cc.) OF ELIXIR

hyoscyamine sulfate	0.1037 mg.
atropine sulfate	0.0194 mg.
hyoscine hydrobromide	0.0065 mg.
phenobarbital	(1/4 gr.) 16.2 mg.

(warning: may be habit forming)

IN EACH EXTENTAB

hyoscyamine sulfate	0.3111 mg.
atropine sulfate	0.0582 mg.
hyoscine hydrobromide	0.0195 mg.
phenobarbital	(3/4 gr.) 48.6 mg.

(warning: may be habit forming)

BRIEF SUMMARY: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma, or urinary bladder neck obstruction. Contraindicated in acute glaucoma, advanced renal or hepatic disease, or a hypersensitivity to any of the ingredients.

REFERENCES: 1. Vollmer, H.: Arch. Neurol. and Psychiat., 43:1057, 1940. 2. Morrissey, J.H.: J. Urology, 57:635, 1947. 3. Krantz, J.C., Jr., and Carr, C.J.: Pharmacological Principles of Medical Practice, 2nd ed., Baltimore (1954), 552.

*This one at Westover, elegant Colonial Virginia plantation, located on the James River near Richmond. Built in the early 1730's by William Byrd II, founder of Richmond, it is now the home of Mrs. Bruce Crane Fisher.

A. H. ROBINS COMPANY, INC., RICHMOND, VA

A·H·ROBINS

An eminent role in medical practice

- Clinicians throughout the world consider meprobamate a therapeutic standard in the management of anxiety and tension.
- The high safety-efficacy ratio of 'Miltown' has been demonstrated by more than a decade of clinical use.

Miltown® (meprobamate)

Indications: 'Miltown' (meprobamate) is effective in relief of anxiety and tension states. Also as adjunctive therapy when anxiety may be a causative or otherwise disturbing factor. Although not a hypnotic, 'Miltown' fosters normal sleep through both its anti-anxiety and muscle-relaxant properties.

Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

Before prescribing, consult package circular.

WALLACE LABORATORIES
Cranbury, N.J.

CM-5761

An antibiotic of choice is one that works

TAO works

Susceptibility Results
Staphylococci [2,3,1]

# OF CULTURES	YEAR	% EFFECTIVE
6,725	1962	88.6%
5,440	1963	88.0%
10,384	1964	88.5%

β-Hemolytic Streptococci [2,3,1]

2,448	1962	89.5%
1,519	1963	95.2%
2,492°	1964	96.7%

The Product

In a world study of antibiotics *in vitro*[1], TAO had an overall effectiveness of 87.3%, higher than chloramphenicol and erythromycin, and significantly higher than tetracycline and penicillin.

The Plus... Consistent Performance

Yet antibiotics must not only work. They must work consistently. Here are the results from the largest study of microbial susceptibility ever undertaken. In 29,048 cultures of overt staphylococcal and β-hemolytic streptococcal infections, note the consistency of results with TAO.

TAO®
[triacetyloleandomycin]

ROERIG Rx

J. B. Roerig and Company, New York, New York 10017
Division, Chas. Pfizer & Co., Inc., Science for the World's Well-Being®

TAO Rx information

Indications: The bacterial spectrum includes: streptococci, staphylocci, pneumococci and gonococci. Recommended for acute, severe infections where adequate sensitivity testing has demonstrated susceptibility to this antibiotic and resistance to less toxic agents. **Contraindications and Precautions:** TAO (triacetyloleandomycin) is not recommended for prophylaxis or in the treatment of infectious processes which may require more than ten days continuous therapy. In view of the possible hepatotoxicity of this drug when therapy beyond ten days proves necessary, other less toxic agents, of course, should be used. If clinical judgement dictates continuation of therapy for longer periods, serial monitoring of liver profile is recommended, and the drug should be discontinued at the first evidence of any form of liver abnormality. It is contraindicated in pre-existing liver disease or dysfunction, and in individuals who have shown hypersensitivity to the drug. Although reactions of an allergic nature are infrequent and seldom severe, those of the anaphylactoid type have occurred on rare occasions. **References:** 1. Isenberg, Henry D.: Health Laboratory Science **2**.163-173 (July) 1965. 2. Fowler, J. Ralph et al: Clinical Medicine **70**.547 (Mar.) 1963. 3. Isenberg, Henry D.: Health Laboratory Science **1**:185-256 (July-Aug.) 1964.

Saion

Well, Doctor Cunningham, I was just telling Herbert I should talk to you about my cough. It comes from down here and...

Chances are the symptom recital may prove to be as difficult to control as the cough. If it's the useless, exhausting type of cough that often accompanies respiratory infection or allergy, you can provide prompt relief with Novahistine DH. Its decongestant-antitussive action controls frequency and intensity of cough spasms without abolishing cough reflex. And the fresh, grape flavor of Novahistine DH appeals to children and adults alike.

When your diagnosis is bronchitis, complicated by thick tenacious exudates, Novahistine Expectorant is particularly useful. It not only provides decongestive action and controls the cough, but also encourages expectoration, thus easing bronchial obstruction.

Use with caution in patients with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Ambulatory patients should be advised that drowsiness may result. Continuous dosage over an extended period is contraindicated since codeine phosphate may cause addiction.

Each 5 ml. teaspoonful of Novahistine DH contains codeine phosphate, 10 mg. (Warning: may be habit forming); phenylephrine hydrochloride, 10 mg.; chlorpheniramine maleate, 2 mg.; chloroform (approx.), 13.5 mg.; l-menthol, 1 mg. (Alcohol 5%). Each 5 ml. of Novahistine Expectorant contains the above ingredients and, in addition, glyceryl guaiacolate, 100 mg.

NOVAHISTINE® DH

NOVAHISTINE® EXPECTORANT

DOW **PITMAN-MOORE** Division of The Dow Chemical Company, Indianapolis

Sustained circulatory, respiratory and cerebral stimulation for the

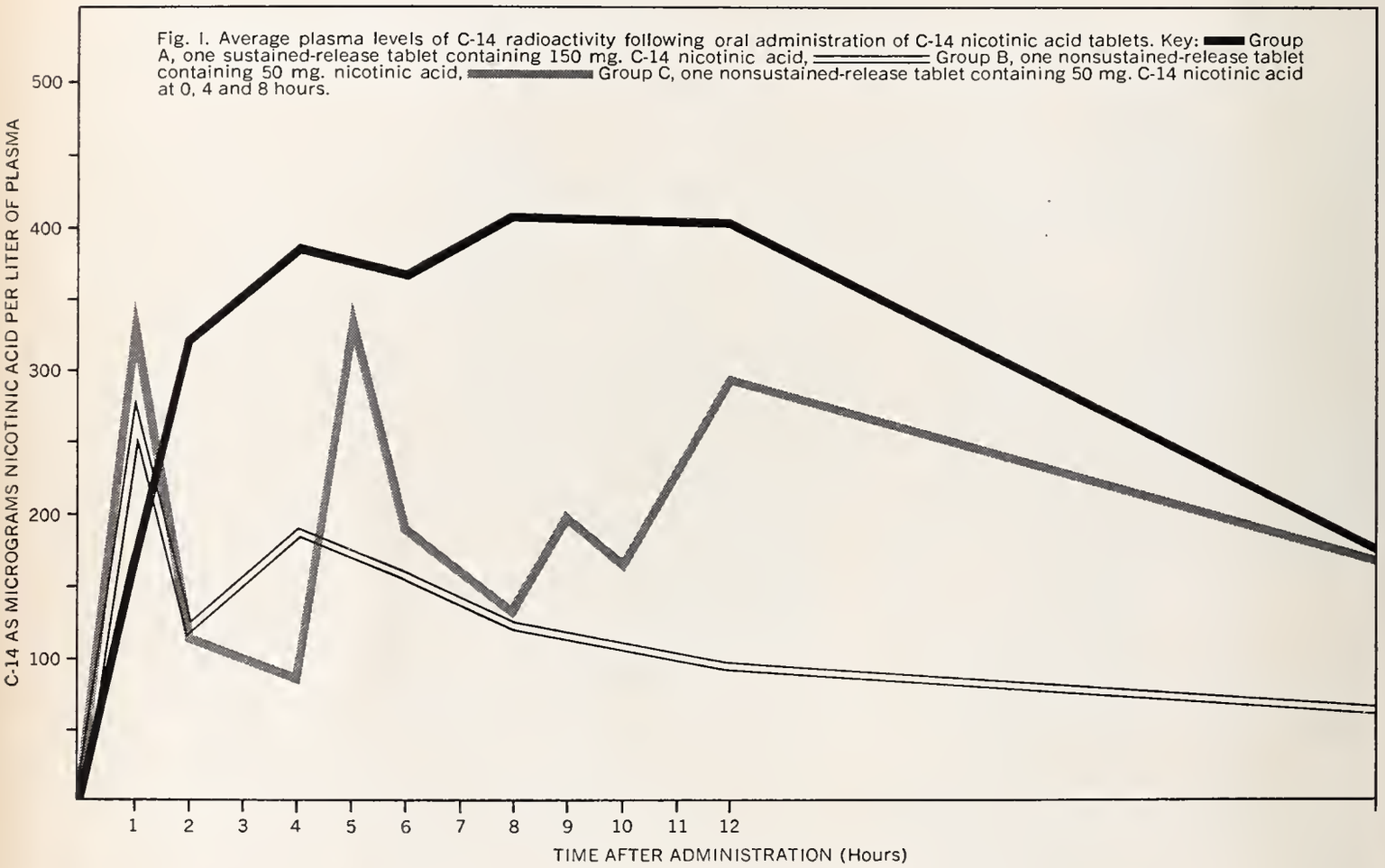


Fig. I. Average plasma levels of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: ▬ Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, ═══ Group B, one nonsustained-release tablet containing 50 mg. nicotinic acid, ▬▬ Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MICROGRAMS NICOTINIC ACID PER LITER OF PLASMA
500
400
300
200
100
1 2 3 4 5 6 7 8 9 10 11 12
TIME AFTER ADMINISTRATION (Hours)

(fewer absent doses by absent-minded patients)

Human volunteer subjects were administered Geroniazol TT tablets with the nicotinic acid component made radioactive with C-14. Plasma and urine samples were analyzed. (See Figures I and II) The radioactive tracer study substantiated the previous clinical evidence that the release of nicotinic acid from the Geroniazol TT tablet produced a gradual rise in plasma levels to a plateau for a total of 12 hours and more.

Such proven sustained activity makes the management of geriatric patients much easier by minimizing the possibility of neglected doses through absent-mindedness or senile confusion. Therapy *can* be continuous on a daily dose of only one Geroniazol TT tablet every 12 hours.

The gradual release of nicotinic acid in Geroniazol TT will provide the well-known peripheral vasodilatation needed in patients with deficient circulation and with a minimum amount (if any) of "flushing." Also, cerebrovascular circulation is complemented by pentylenetetrazol, long-established as a cerebral and respiratory stimulant.

Geroniazol TT improves the typical, unfortunate, signs of senile confusion. Patients become more alert,

ged and debilitated

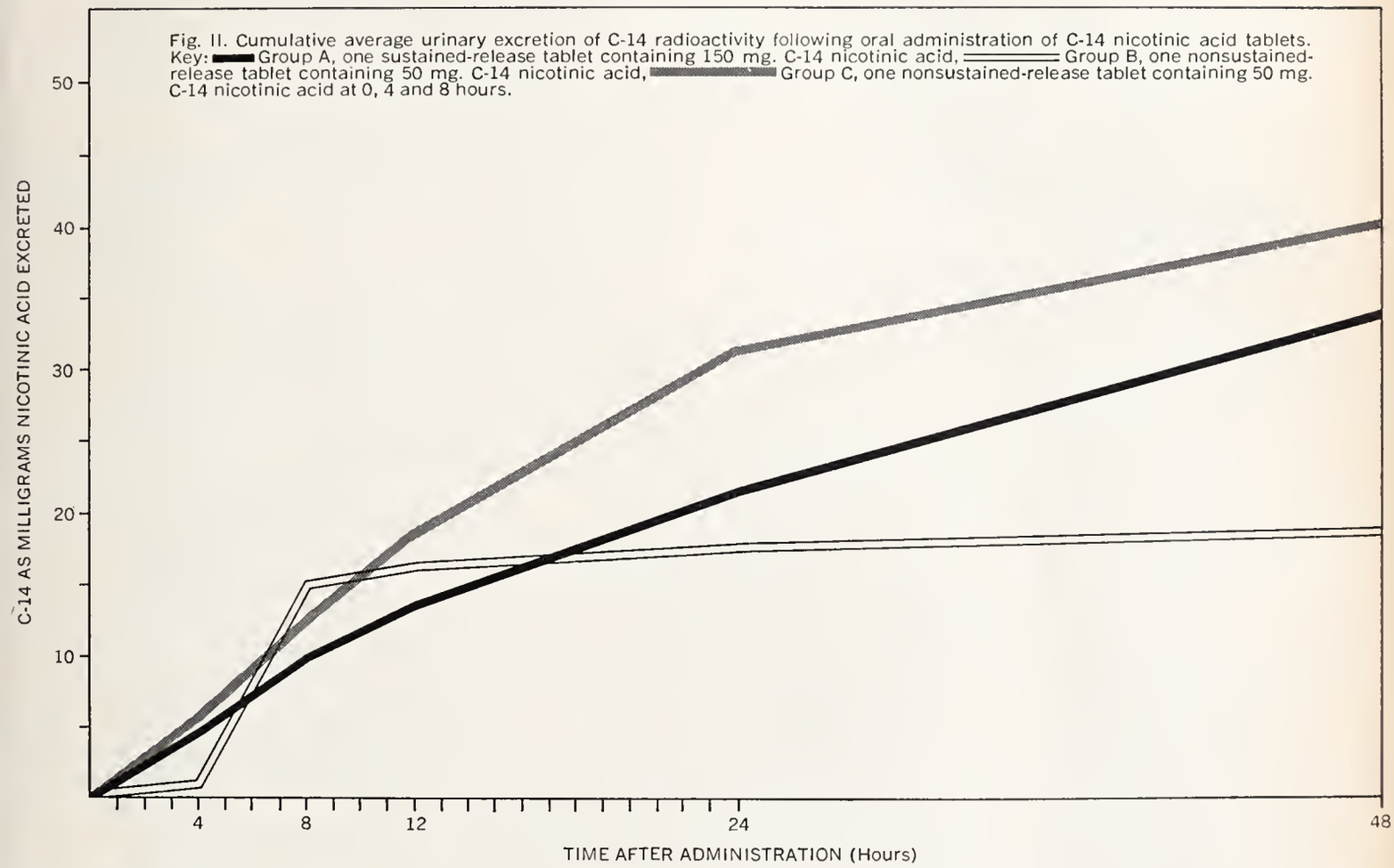


Fig. II. Cumulative average urinary excretion of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MILLIGRAMS NICOTINIC ACID EXCRETED
50
40
30
20
10
4
8
12
24
48
TIME AFTER ADMINISTRATION (Hours)

less confused and moody. Personal care, memory, emotional stability, social attention improve. Fatigue, apathy and irritability are reduced.

A prescription for 100 tablets of Geroniazol TT will permit your patients to enjoy the benefits of time-prolonged nicotinic acid/pentylenetetrazol therapy, at an economical price. Dosage is only one tablet every 12 hours.

Contraindications: There are no known contraindications.

Precautions: Exercise caution when treating patients with a low convulsive threshold.

Side Effects: Side effects are rarely encountered, however due to the vasodilatation effect of nicotinic acid, transitory mild nausea, flushing, tingling and pruritus are possible.

Dosage: One tablet every 12 hours.

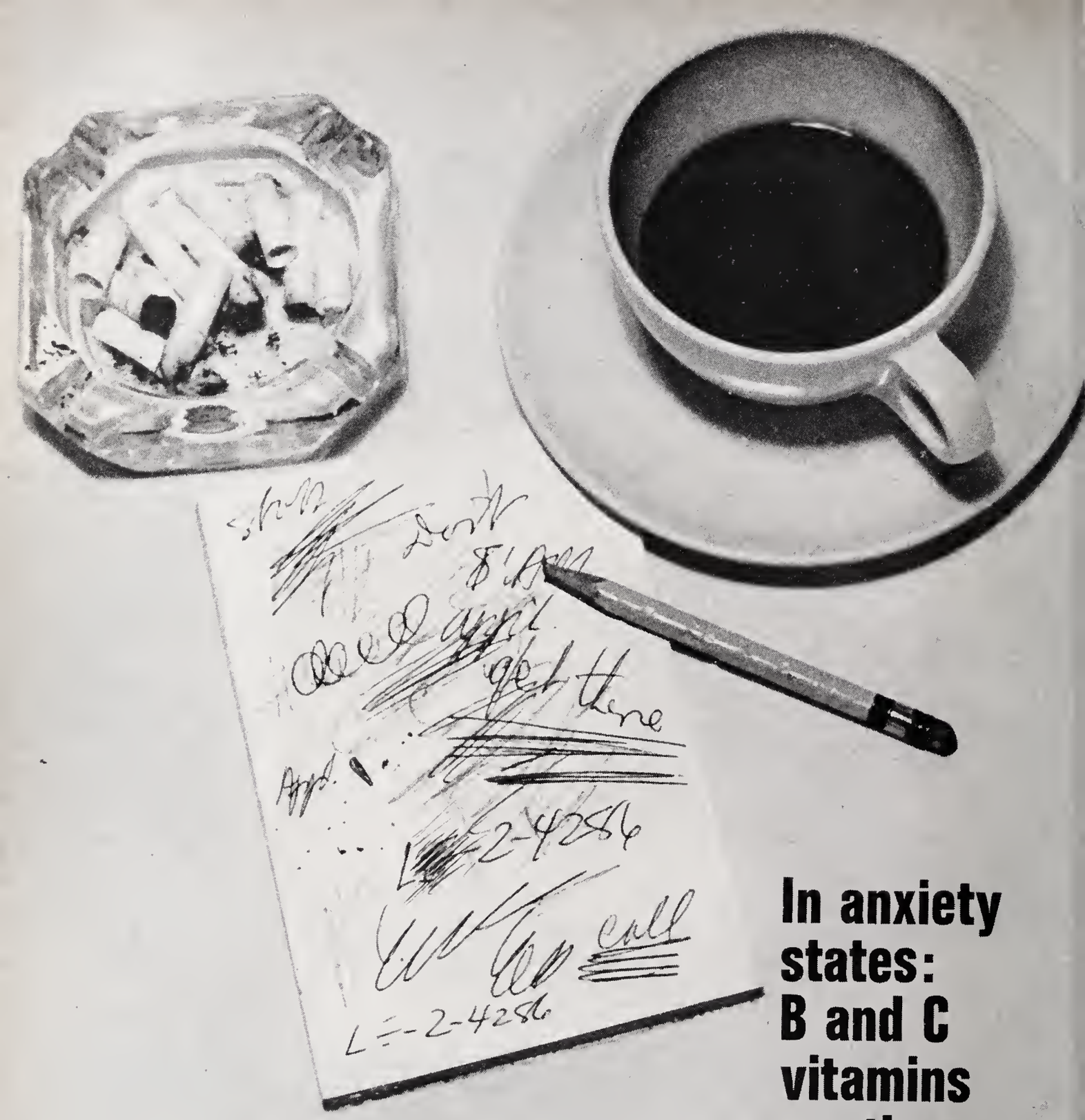
Supplied: Prescribe bottles of 100 tablets, to take advantage of recent price reduction.

References: 1. Report by Nuclear Science & Engineering Corp., Pittsburgh, Pa., in files of Philips Roxane Laboratories. 2. Connolly, R.: W. Virginia Med. J. *56*:263 (Aug.) 1960. 3. Curran, T. R., and Phelps, D. K.: Am. Pract. & Digest Treat. *11*:617 (July) 1960.

"First with the Retro-Steroids"
PHILIPS ROXANE LABORATORIES
Division of Philips Roxane, Inc., Columbus, Ohio
A Subsidiary of Philips Electronics and Pharmaceutical Industries Corp.

Geroniazol® TT

nicotinic acid 150 mg., pentylenetetrazol 300 mg.
Tempotrol® Time Controlled Tablet



In anxiety states: B and C vitamins are therapy

Stress formula vitamins are an important supportive measure in maintaining the nutritional status of the emotionally disturbed patient. With STRESSCAPS, B and C vitamins are present in therapeutic amounts to meet increased metabolic demands. Patients with anxiety, and many others undergoing physiologic stress, may benefit from vitamin therapy with STRESSCAPS.

STRESSCAPS®

Stress Formula Vitamins Lederle

FOR PROFESSIONAL RECOMMENDATION ONLY

Each capsule contains:

Vitamin B_1 (as Thiamine Mononitrate)	10 mg.
Vitamin B_2 (Riboflavin)	10 mg.
Niacinamide	100 mg.
Vitamin C (Ascorbic Acid)	300 mg.
Vitamin B_6 (Pyridoxine HCl)	2 mg.
Vitamin B_{12} Crystalline	4 mcgm.
Calcium Pantothenate	20 mg.

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, N. Y.

8241 4

spring 1966

DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M.D., Director of Medicine.

Duodenal mucosa with ulcer crater—
Approx. 80× Magnification.

Loosening epithelial cells of nasal mucosa during early stage of cold—Approx. 1800× Magnification.

this issue: partners in misery

Partners in misery: common cold and duodenal ulcer*

A century or two ago our forefathers believed that the common cold was caused by an excess of evil humors. Today we regard these baleful humors as a result of the cold rather than its cause. Only in the last ten years have some of the offending viruses been cultivated, and we now know many others can be transmitted to human volunteers although they cannot yet be grown in the laboratory. Various body defenses react to infection by a virus, and in the respiratory tract the lung is protected by the phagocytic properties of the pulmonary alveolar macrophage.[1]

Because of the brief history and large proportion of unidentified viruses, effective prophylactic vaccines have not yet appeared. A universal antiviral agent seems even more remote. Meanwhile, these viruses continue to produce epidemics of upper respiratory infection at seasonal intervals, particularly in spring, fall and winter, during which they propagate and distribute progeny indiscriminately. As a result, they are responsible for significant work loss. Since the immunity afforded is temporary, recurrent infections are common.

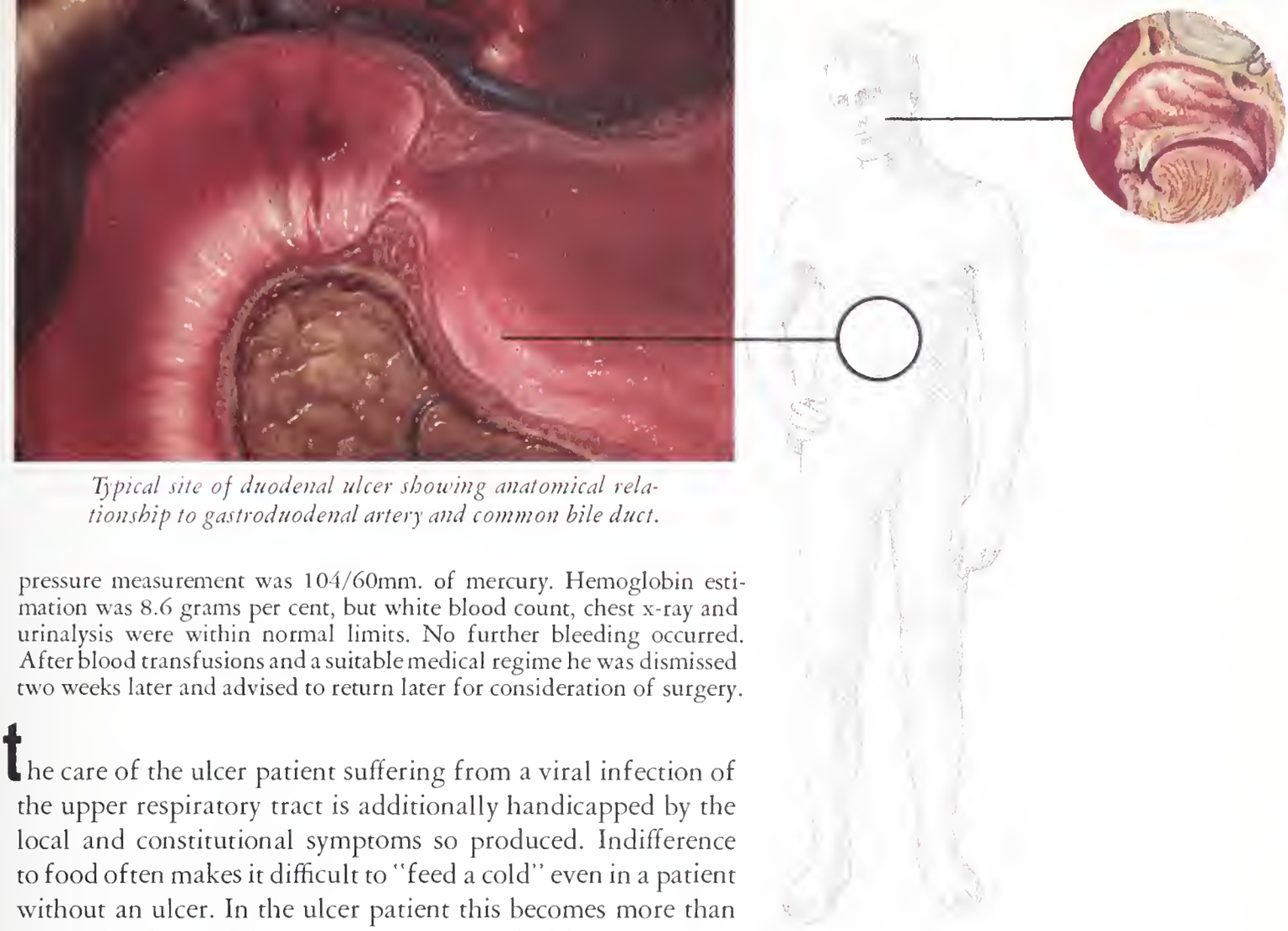
This recurrent morbidity in spring and fall is shared by duodenal ulcer, itself a cause of significant loss in wages and medical expense. Thirty years ago Emery and Monroe reported 1,279 recurrences of peptic ulcer of which 13 per cent could be attributed to upper respiratory infection, thus confirming a long-held clinical impression.[2] While the inflammation, congestion and secretion of the mucosa of the nasopharynx in the common cold, and the hyperemia and hypersecretion of the gastric mucosa during acute exacerbation of a duodenal ulcer may not be directly linked, it is probable that the miseries of the infection are reflected in the pain of the ulcer. The following case history illustrates this point.

Case report A 48 year old white male executive, suffering from hematemesis and melena for 12 hours was admitted to hospital on April 8, 1965. He gave a history of duodenal ulcer proved by x-ray studies 23 years previously. In the intervening years he suffered from typical epigastric pain occurring two hours postprandially, usually for two or three weeks each spring and fall. A hospital admission two years earlier had followed hematemesis of moderate amount.

The present admission was preceded by a recurrence of pain during a period of unusual business pressure. In addition he caught a cold one week before his entry to hospital and his already irregular eating habits were made more so by loss of taste and appetite, blocked nose and general malaise. During the final 36 hours he used aspirin to relieve his cold, taking 2 or 3 tablets with a glass of water on 6 or 7 occasions. Before retiring he drank a glass of hot toddy.

He awakened about 1 a.m. and vomited dark red blood and recently ingested food mixed with whisky. Melena followed and he was transferred to hospital. On admission he was pale, cold and sweating. Apart from epigastric tenderness and black stool on the examining finger after rectal examination, his general physical examination was not remarkable. Temperature was normal, pulse was thin and thready with a rate of 114 beats per minute, and blood

The commonly found "clover-leaf" roentgenographic abnormality of chronic duodenal ulcer due to spastic contractions and scarring.



Typical site of duodenal ulcer showing anatomical relationship to gastroduodenal artery and common bile duct.

pressure measurement was 104/60mm. of mercury. Hemoglobin estimation was 8.6 grams per cent, but white blood count, chest x-ray and urinalysis were within normal limits. No further bleeding occurred. After blood transfusions and a suitable medical regime he was dismissed two weeks later and advised to return later for consideration of surgery.

The care of the ulcer patient suffering from a viral infection of the upper respiratory tract is additionally handicapped by the local and constitutional symptoms so produced. Indifference to food often makes it difficult to "feed a cold" even in a patient without an ulcer. In the ulcer patient this becomes more than a homily, for malaise and apathy towards irksome routine produce a lack of interest in adhering to an ulcer program and result in its eventual failure if these symptoms are not relieved. The absence of curative treatment for these viral infections has spawned many a strange therapeutic offspring and some of the more generally accepted remedies contain a special risk for the ulcer patient.

To the gastric physiologist, the shot heard around the world was not fired at Concord but was discharged through the stomach of Alexis St. Martin. The worthy voyageur had a more than modest thirst and on several occasions William Beaumont observed through the resulting fistula the changes of gastritis which followed an immoderate ingestion of alcohol.[3] It is now well known that alcohol stimulates the production of gastric juice and the patient with an ulcer, even in remission, should not drink alcohol without first taking food, milk or antacid. The use of various alcoholic remedies for the common cold is to be strongly discouraged in this type of patient. Factors already present can produce an acute exacerbation of ulcer symptoms without the added stimulus of alcohol.

Headache and facial pain, sore throat and generalized aching are subjective discomforts of viral infections of the upper respiratory tract or their complications. Relief from them is usually sought in the family medicine cabinet, and of all the medications habitually stored there, salicylates are the most frequently used. Oral ingestion of salicylates has produced

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substernal pyrosis and sometimes epigastric pain in susceptible individuals. Bleeding from erosions of the gastric mucosa has followed their use and has been severe enough in some instances to cause hematemesis and melena.[4] It has been suggested that interference with the protective mucous layer of the stomach allows this to happen. There is no evidence that hypersecretion occurs, but the taking of aspirin has been followed by exacerbation of ulcer symptoms and occasionally by gastrointestinal bleeding.[5] The ulcer patient with a cold should take salicylates with an antacid, or preferably other means of symptomatic relief should be found.

Caffeine has two actions on gastric secretion. One directly stimulates production of acid and the other potentiates the out-pouring of gastric juice as a response to other stimuli.[6] In high doses to animals, it has produced erosive gastritis and peptic ulceration. Caffeine-containing beverages are discouraged for the ulcer subject and forbidden during an acute exacerbation.[7] Many cold remedies contain caffeine.

To sufferers from allergic rhinitis springtime brings symptoms of nasal obstruction, loss of taste and smell and malaise similar to those caused by viral infection but due instead to allergens which at that time of year are principally tree pollens. Relief from these symptoms can be obtained, though not wisely, by the use of steroid medication. Unfortunately, chronic sufferers from these allergies have used this approach with varying degrees of success. Steroid hormones increase gastric secretion and delay the healing of experimental ulcers.[8,9] Clinically their administration has been associated with reactivation of healed duodenal ulcers, and with bleeding and perforation which were not always preceded by typical ulcer distress. Because of these harmful effects, their use in the ulcer patient is best avoided for other than serious medical problems and then only with adequate antacid coverage.

Summing up It is immaterial by which pathways the malaise and lassitude, depression and irritability activate an ulcer, but it is the physician's responsibility to ensure that the medications he selects to relieve the symptoms of a cold do not further aggravate the ulcer.

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The Therapy of Coincidence

SOME YEARS AGO, two young women in Los Angeles died of pulmonary embolism within a short time of each other. It happened that both were using an oral contraceptive preparation at the time. Whatever sane comments may have been generated by this event, they were soon drowned out by the lay press ("Pills Kill!") and by panicky medical men whose training in logical thinking had clearly not introduced them to the commonest fallacy of all—that of *post hoc*. The rest is history. Case after case of thromboembolic disease in women using oral contraceptives crept out of the woodwork, to be told and retold in hospital corridors. Antagonists of contraception saw a golden opportunity to drive "the Pill" out of existence. Investigators began or were forced to undertake vast and expensive programs to study all the known clotting factors in patients taking these drugs, even though hematologists were by no means in accord over the meaning of the tests themselves. In all this uproar, few people listened to the lone voices which asked, "Could it all be coincidence?" and fewer still listened to the statisticians who reiterated that hallway collections of "anecdotal" data cannot possibly provide an answer to the question of causality versus coincidence.

Since thromboembolic phenomena "are associated with pregnancy" and since oral contraceptives embody hormones similar to those circulating in pregnancy, it seemed reasonable to infer that a predisposition to thromboembolic disorders should be produced. The truth, however, is not so simple. Thromboembolic phenomena are associated with the postpartum state, *not with the pregnancy itself*. The incidence during pregnancy is only about 180 per million, whereas the incidence associated with delivery and the postpartum state is 3,420 per million.[1]

Furthermore, idiopathic pulmonary embolism is not so rare a disease as it once might have been, and its incidence is rising. In England there were five times as many cases in 1961 as in 1952.[2] It is now the most common fatal adult pulmonary disease found at autopsy in general hospitals.[3] On this basis it seems logical to question whether the thromboembolic phenomena observed in women using oral contraceptives (and there are estimated to be more than 4,000,000 of them) are causally related to use of the drug, or whether they are occurring with the usual frequency, *coincidentally*, in this large population. It seems proper to demand an answer to this root question before condemning these drugs as dangerous and before demanding vast investigations of clotting factors.

The data which have been accumulated present a most interesting picture. Table 1 shows the incidence of thromboembolic phenomena reported to one drug company,[4] against an estimate of the spontaneous incidence of this disorder. In every year, the number of reported cases is *substantially less* than the number that might be expected by chance alone in a population of this size. It may be argued that thromboembolic phenomena are not reportable and that the true number of cases is underestimated. An underestimate by a factor of four seems unlikely, particularly in view of all the publicity this question has been accorded. One might even say that data compiled by a drug company could be biased. It is therefore most interesting to see that the same conclusion is reached from the figures compiled by the College of General Practitioners and General Register Office, H.M.S.O., London (Table 2). If even these figures are not convincing, one may turn to the incidence of fatal pulmonary embolism, surely a disorder which is not apt to go unnoticed or unreported. (Table 3). Again there is no evidence for a higher incidence in users of oral contraceptives than in non-users. From these figures and from the effectiveness of these drugs in preventing conception (and consequently the thromboembolic phenomena associated with delivery), one can actually make an excellent case for the use of oral contraceptives as prophylaxis against thromboembolic complications!

Statistics such as these have done little as yet to still the concern of the medical profession or lessen the exploitation of this sensationalistic material by the lay press. Since the *post hoc* fallacy rises from its ashes like the phoenix, one might anticipate that it will emerge perennially in other forms. And indeed it has: Walsh and his collaborators[5] have recently published another of these hallway collections, carefully entitled "Oral Contraceptives and Neuro-Ophthalmologic *Interest*" (italics mine). Most people will still fail to get the message that these cases are worth noting in the event that there *may* be some relationship here. Those scientifically interested in adverse reactions will note such a report, watch for further developments, and if it becomes timely, design appropriate studies to evaluate coincidence versus causality. The rest of us will undoubtedly be exposed to the gossip mill and to the pundits who expatiate on the significance of a "personally observed case."

A third round is now in the offing, with the recent report by Whitelaw and others[6]

TABLE 1.—*Idiopathic Venous Thromboembolism*

Year	Enovid Use (Woman-Yrs.)	Morbidity Predicted	Morbidity Reported
1957	20,000	13	1
1958	29,000	19	0
1959	34,000	23	2
1960	54,000	36	2
1961	297,000	198	53
1962	793,000	527	263
1963	1,482,000	986	118
Total		1,802	439

TABLE 2.—*Thromboembolic Disorders Reported in Association with Norethynodrel and Mestranol in the United Kingdom*

	Population at Risk (Expressed as Total Cycles of Experience)	Woman-Years of Experience Based on Conservative Estimate of 13 Cycles per Annum	Expected No. of Cases	Reported No. of Cases
1959-1962	698,281	46,552	102	27

TABLE 3.—*Fatal Pulmonary Embolism*

Year	Population* at Risk	Idiopathic† Predicted	Enovid Users
1961	500,000	4.8	4
1962	1,370,000	13.2	13
1963	2,280,000	22.0	17

*Females 15-44

†Based on average mortality rate of 9.66 per million nonpregnant women.

of menstrual irregularities and infertility following treatment with progestational steroids. For Whitelaw and others to designate these findings as side-effects is a little peculiar, since three of the 24 "normal" women were actually put on treatment *to regulate their cycles* and one of the women allegedly rendered infertile was a nulligravida.

Thus we see that panic, misinterpretation, or just plain ignorance still form the basis of judgments about the safety of our newer drugs. Clinicians are still not used to thinking in terms of the huge number of individuals taking a particular drug, and the opportunity this affords for coincidental events which may be mistaken for causal relationships. If physicians do not keep this inevitable possibility in mind, they may end up treating coincidences instead of patients—an occupation of doubtful utility and poor prognosis.

—JOSEPH W. GOLDZIEHER, M.D.,
San Antonio.

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Behind the Decision To Dissolve Texas 65

THE TEXAS 65 Health Insurance Association was organized in 1963 as a nonprofit, public service organization of 64 insurance companies doing business in Texas. The member companies agreed to underwrite the benefits of a health insurance program for the senior citizens of Texas. Through Texas 65, health insurance was made available to all citizens of the state, 65 years of age and over, regardless of health or age. Texas 65 was private enterprise's partial solution to the perplexing problem of health insurance for the aged.

Then came Medicare. The ultimate decision to discontinue Texas 65, when Medicare takes over July 1, was not a hasty one. The question was thoroughly studied and researched by the staff and officers of the Association. Similar organizations in other states were consulted, for they also faced the problem of what to do. The ultimate decision was unanimous. All states with 65 programs will cease their operation on July 1, 1966. All, however, will remain open to process claims incurred before that date.

Had the Medicare amendments to the Social Security Act covered only the costs of hospitalization for the elderly, then there would have remained some room for the 65 programs. They could have remained in operation to cover costs of physicians and surgeons fees and other related services. But the final version of Medicare legislation duplicated almost entirely those services offered by Texas 65 and similar plans in other states.

There is no reason of course to compete when what you are selling is being given away, or sold at a lower price. Especially is this true when your competition is the federal government.

The remaining areas, beyond those covered by Medicare, are so limited in scope as to make it foolish to continue the operation of Texas 65. Also, a number of member and nonmember companies who market health insurance to persons of all ages have indicated they will offer programs to supplement coverage by Medicare.

To make sure that the under-age 65 spouses of individuals who are insured by Texas 65 are not left without health insurance, Texas 65 is currently considering plans to continue their health insurance until they become eligible under Medicare. Details of this plan will be announced when completed.

Texas 65 felt a sincere obligation to announce its future plans well in advance of March 31 so that its members would have ample time to sign up before the first Medicare deadline. It also felt obligated to both its members, and the medical profession, to urge those over 65 to enroll for the voluntary part of Medicare which provides for certain doctor's fees.

Since Texas 65 began its operations in

November, 1963, it has paid doctors' and hospital bills in excess of $10,000,000.

Texas 65 has done what it set out to do—provide vital health insurance coverage to that segment of our population that otherwise might not have had health insurance protection. The program has just simply been replaced by Medicare.

DAVE SMITH, Austin,
Director of Public Information for Texas 65.

Computers Can Aid Physicians in Practice

THE DAY of the computer in the practice of medicine is here. The increasing acceptance of the computer at all levels in our profession and the understanding of its use are probably the greatest advances of this decade. There have been many advances in methodology in medicine and new techniques, but these apply to narrow areas. This instrument will make the doctor and the hospital more accurate and more efficient, and at the same time will continue to be indispensable as a research tool.

The computer can be used to the advantage of every physician and patient. It is not a substitute for the doctor; it cannot replace his judgment or his ability to act correctly and honorably, but it can serve as his companion, his helper, and relieve him of many tasks that take up his all-too-little time. The use of the computer in research is established, and its value unquestioned. It is just now breaking through into the practice of medicine, and its increased use is growing at a startling pace. Every hospital, whether teaching, community, or private, is either negotiating for or thinking about utilizing a computer. Computers will not only speed up and make more efficient the operation of the hospital, but will serve the staff in many ways. By standardizing nomenclature of history, physical findings, diagnoses, treatment, and results, a true evaluation of methods and procedures will be possible. Variations, even in minor degree, can be evaluated with unprejudiced accuracy and economy of time.

In this day of pressure to centralize treatment, we need means of studying the results of treatment at all levels. This will stimulate the facilities away from our large teaching centers toward improvement. By knowing what is being done, what results are obtained, and by comparing these with teaching institutions, computers may increase their pride of accomplishment. Small computers are available to small units, and arrangements would easily be made for compilation in the larger institutions.

As aids in diagnosing problem cases, computers can be of great help. They would not substitute for the physician but could remind him of diagnoses he may have failed to consider. As one computer enthusiast has said, "if you put trash into a computer, you get trash out"; however, correctly programmed, a computer could be most helpful.

Connecting computers in centers by means of communication facilities such as telegraph and telephone could bring to the smallest hamlet the best and quickest consultation. This could include analysis of laboratory studies, evaluation of electrocardiograms, suggestions of diagnoses, and suggestions of accepted methods of treatment. This information can only be used properly by a physician, and the more well trained and educated he is, the better he will use such information. Any doctor who feels he knows too much to use such an aid must have colossal conceit.

The shortage of paramedical personnel will become more acute in spite of crash programs to correct it. The assistance in this area by well planned use of computers staggers the imagination. The reduction of many procedures to one, the rapid storage of information available for instantaneous recall, and even the programming of procedures commonly requiring the time of one or more aids are all possible with computers, and they are unbelievably accurate. As an added benefit in this field, the release of paramedical personnel from boring and routine procedures will make them available for more important and stimulating work, and will thereby attract more and better people.

The computer's use in public health has already been proved. It has also been proved in research and in the practice of medicine and hospital management. Their acceptance by the profession as a whole is explosive. We do not need to know how they work or how to use them, but we do need to understand

what they can do for us and be prepared to work with those trained in their use. This does not mean that we are surrendering any of our prerogatives as doctors, it only means that we are accepting the advances of science and technology as we did when we abandoned the horse and buggy for the automobile.

—ALBERT W. HARTMAN, M.D., San Antonio.

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Insurance Companies Face Many Decisions on Medicare

THE ENACTMENT of Medicare is bound to have an important impact on the financing and provision of medical care. Certainly the insurance companies are faced with multiple decisions as to the effect this new law will have on policies in force and the issuance of policies sold now and in the future.

Insurance companies are confronted with such problems as policy language changes and policy approval by state insurance departments. Other matters to be considered include the avoidance of duplicating or overlapping the benefits to be provided under the government program; the status of the conversion privilege in group insurance; the status of guaranteed renewable individual insurance; the problem created by an insured person reaching age 65 but having a spouse or other dependent at some age less than 65; the problem of adverse selection on outstanding business; the adjustment of premium levels and other policyholder equities; and the effect upon the disability income business.

Insurance companies are also faced with the problem of deciding if and to what extent they wish to provide coverage that supplements Medicare benefits. Also, the companies will undoubtedly make a decision as to whether they want to provide coverage for those individuals over 65 who, for one reason or another, do not sign up for the voluntary supplemental medical insurance part of the Medicare program. It is safe to assume that no single answer to these questions will evolve from the considerations of the many insurance companies. However, the competitive interplay of the various insuring mechanisms will see that the public's best interests are served.

The potential influence of Medicare on the under 65 population points up the need for strengthening present channels of communication between the voluntary health insurance industry and the providers of medical care—particularly the medical profession. It seems obvious that the pattern of benefits developed as a result of Medicare will have some influence on the type of coverage provided for the under 65 population. Therefore, health insurance companies will, as they have in the past, continue to provide coverage that is responsive to public demand.

The effectiveness of existing health insurance coverage is exemplified by the approximately 50 million people in this country who have major medical type coverage. This coverage employs the concept of "usual and customary" fees for "reasonable and necessary" services and supports the concept that the individual physician should be given the widest possible range of freedom of judgment in the medical management of a patient's care. It is reasonable to expect that the public will continue to look to the voluntary health insurance industry to provide such broad forms of coverage at a reasonable cost.

If we accept the fact that the health insurance industry is providing effective coverage, and if we accept the fact that the American people are receiving the highest quality of medical care ever provided in this or any other country, then what additional responsibilities do we have? There is at least one, and that has to do with efficient use of the health-care dollar. We must assure our respective publics that steps are being taken to properly utilize the money they spend for insurance premiums and the costs of health care.

Although insurers provide insurance bene-

fits against the cost of medical care, it is an accepted fact that the doctors admit patients to the hospital, prescribe the type and frequency of service, perform the surgery, discharge the patient from the hospital, and prescribe continuity of care in the nursing home and home care environment. Therefore, the medical profession is in a unique position to provide leadership so that the problems involved in the inefficient use of the health-care dollar can be placed in proper perspective and resolved to the mutual satisfaction of all parties involved. It is the responsibility of the health insurance industry to work with medicine to attain this desirable goal.

The Medicare program has placed a great deal of emphasis either directly or indirectly on two mechanisms which can be responsive to conserving the health-care dollar—namely, review committees and hospital utilization committees. These mechanisms are not new innovations promulgated by the enactment of the law. They are, however, the results of voluntary joint discussions between the providers of medical care and the health insurance industry which have taken place over the years. The Health Insurance Council, representing approximately 90 percent of the health insurance business written by insurance companies in the United States, has been involved in joint discussions with organized medicine for at least the last five years. Such discussions should not cease but should be stepped up so that any programs involving review committees and hospital utilization activities that emerge will be a tribute to the voluntary system.

Mr. L. A. Orsini, director of the Health Insurance Council, succinctly set forth the challenge the voluntary system faces when he discussed "Medicare—Its Challenges and Responsibilities" in the November, 1965, issue of "Health Insurance Trends." The concluding paragraph of his letter to the medical profession states:

> The voluntary system of providing and financing health care has earned a unique position of public confidence. If we are to face our problem imaginatively, aggressively, seeking solutions which are consistent with the broad public interest, we can face the future with confidence. This is no time for defensive emotion, but there is an urgent need for accepting the challenges which confront us and demonstrating our willingness and ability to meet them constructively.

And so must we meet the challenges.

—R. B. DONOVAN,
First Vice President, United American Insurance Company, Dallas.

Cataract Removal In Elderly Persons

THIS ARTICLE is written as a protest against removal of cataracts in elderly people who are getting along well enough without operation. To the surgeon this may be a very small event, but to the patient it often is one of life's crises.

Consider the situation of the old person whose vision is failing, who cannot read the street signs so well, or who can no longer follow the flight of the golf ball. If it is discovered that he has cataracts and an operation follows, then he has to have an operation on the second eye or wear a contact lens; otherwise he loses binocular vision. If the eye is fitted with a cataract lens, the other eye must be blurred out or the patient will see double. In this case his last situation is worse than his first. Every patient does not get along well with a contact lens. If he is palsied, impatient, or neurotic the doctor is in for a hard time. Many nervous persons have great difficulty in adjusting to a cataract lens. It makes objects look larger and closer and restricts the field of vision. If these miseries are added to the others that accompany the aging process they are hard to bear and should not be inflicted upon a person who can still see well enough to do everything he wants to do.

At about the age of 40, changes take place in the crystalline lens. The fibers that are formed first in embryonic life—the fetal nucleus—are pushed to the center by the fibers that are formed later. These fibers are formed all through life from the lens epithelium at the equator and they pass forward and backward to join in complex suture lines beneath the lens capsule. These new fibers crowd the older ones together until eventually they become dehydrated, change color, and shrink. This is the mechanism that

keeps the lens from getting too big for the eye and cutting off the drainage angle, thus producing secondary glaucoma. Unfortunately, this does not always work, and many narrow angle glaucomas that develop in later life are due to an enlarged crystalline lens.

For the purpose of this discussion, cataracts in the elderly may be divided into two types, nuclear and cortical, although they are often mixed. Everyone who lives long enough has cataracts. Between the ages of 40 to 45, a person with normal eyes begins to use reading glasses. This is because the lens fibers are losing elasticity and can no longer mold the lens, hence the person's power of accommodation begins to fail. If the lens is examined carefully at this stage with the slit lamp microscope it will be noted that there is a slight discoloration of the lens nucleus. This increases with each year of life and often the refraction changes. If the lens does not flatten as the inner fibers become more discolored, or if the cortical fibers swell, myopia ensues and we have the "second sight" that we have often heard described by the admiring children of old folks who could read without glasses. The distance vision in their eyes may often be improved by myopic lenses and the near vision by a stronger plus lens. Many such persons read comfortably with the naked eye. I have observed many old people for years until they die with the cataracts unremoved. Some of these had been told years ago that they had cataracts which should be operated on.

My plea is that these old people deserve a careful examination instead of a hasty one. Many have degeneration of the retina in the macular region and cannot see well if operated on. Wide dilatation of the pupils will allow this to be detected. This will prevent a disappointed patient and an embarrassed doctor. Perhaps, now, I am old enough to appreciate what adding more miseries means to an elderly person.

—Everett L. Goar, M.D., Houston.

COMMENTS

Every ophthalmologist who does cataract surgery should read this timely article written by one of this country's great leaders in his field. If Dr. Goar's advice is followed, many elderly patients will be spared the unnecessary ordeal of a major operation—an operation that may leave a far greater annoyance than the preoperative dim vision.

—Everett R. Veirs, M.D., Temple.

Health Care Costs Rise; Doctors' Share Slides

Americans are increasing their spending for health care.

They still, however, spend more for tobacco than for hospital care, and more for cosmetics and haircuts than for doctor bills, reports the *AMA News*.

Spending for health care totaled $25.2 billion in 1964, according to figures of the U.S. Department of Commerce. That's an increase of 7.8 percent from the $23.4 billion spent in 1963.

Hospitals receive the largest share of health-care spending—about 30 cents of every dollar. The total spent for hospital care in 1964 was $7.6 billion. This compares with $7.8 billion spent on tobacco products.

Other portions of the health-care dollar are divided among drugs, 17 cents; dentists, 10 cents; health insurance, 7 cents; appliances, 4 cents; and miscellaneous expenses, 5 cents.

The remaining 27 cents goes to physicians. This percentage has declined slightly; 20 years ago, physicians received about 28 cents of every dollar spent on health care.

Expenditures for physicians' services totaled $6.8 billion in 1964, compared to $7 billion spent on personal items such as cosmetics, haircuts, and toiletries.

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Minimum Wage Boost Could Hurt Hospitals

With our coffee table conversation revolving almost exclusively around government regulations to implement Medicare, we may not take the time to delve into problems created by the pending hourly wage increase proposed by our present administration.

This labor-sponsored bill which is presently in committee is reputed to be ready soon for Senate and House action. There is said to be agreement for a raise in the minimum hour wage to $1.40 effective in January, 1967, and to $1.60 in January, 1968.

What impact will this have on the practice of medicine? Since medical bills and hospital bills are lumped together by the consuming public, the impact will be tremendous. Consider, for example, all hospital employees being covered by this minimum wage bill. Porters and maids will be raised to $1.40 per hour; orderlies and vocational nurses must be paid more than the minimum by reason of educational training and position responsibility; likewise, wages of office employees as well as those of the floor nurses and supervisors must be raised. Hence a chain reaction of wage increases throughout the entire hospital must take place.

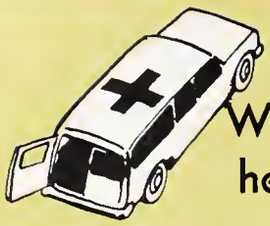
Hospital administrators estimate that a 350-bed general hospital will face wage increases of $500,000 per year at a minimum wage of $1.40 per hour. In terms of bed cost, this amounts to $7 per day. Already, in Texas, the average patient cost per day for a hospital bed is in excess of $47.

This is highly inflationary legislation and it denies the marginal worker a chance to earn a living. Hospitals, business, industry, and agriculture cannot afford such prices for labor, and, if the bill is enacted, many small businesses, restaurants, hotels, motels, and farms will cease to exist. Fewer jobs at higher pay, in money that will buy less, will be the end result. Even those whose wages are increased will find higher taxes and inflated prices. Those who cannot qualify for work will become permanent wards of the city, state, or federal government.

Industry has met this challenge by automation. We, too, must devise additional labor-saving methods in our hospitals; and we should oppose this legislation by conveying our views to congressmen and senators who represent us in the United States Congress. The ability of each individual, as well as his effectiveness and his contribution, in the nation's employment market of supply and demand, should determine wages.

James D. Murphy M.D.

President
Texas Medical Association



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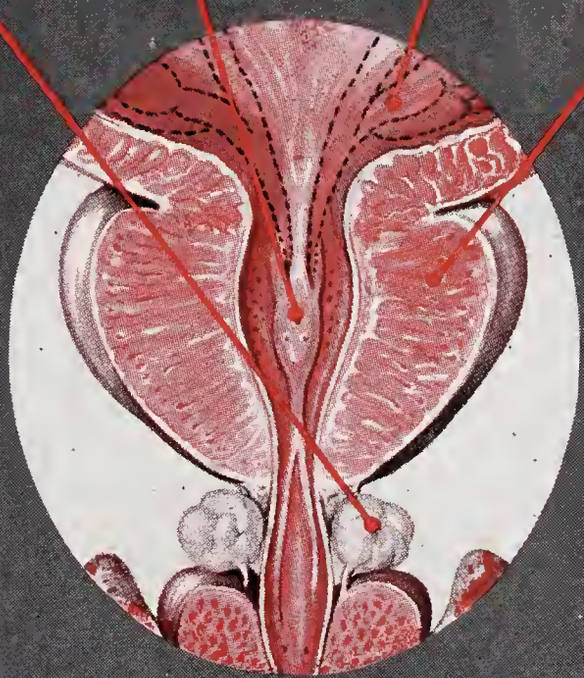
meet the authors

ROBERT C. HORN, JR., is chairman of the Department of Laboratories, Henry Ford Hospital, Detroit. Prior to 1955, he was professor of surgical pathology at the School of Medicine, University of Pennsylvania. Conductor, **Tumor Seminar, p. 48.**

JOSEPH W. GOLDZIEHER, a San Antonio endocrinologist, presents facts and statistics which have made him wary of charges that a direct relationship exists between contraceptive pills and thromboembolic disease. **The Therapy of Coincidence, p. 35.**

Flagyl destroys trichomonads

here & here & here & here



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Flagyl eliminates the difficulties and frustrations that have long attended the treatment of trichomonal infection.

These difficulties arose mainly from:

1) the failure of any previously known agent to destroy the protozoan in paravaginal crypts and glands;

2) the failure of any previously known agent to prevent reinfection by eradicating the disease in male consorts.

The introduction of Flagyl removed both of these long-standing deficiencies. Hundreds of published investigations in thousands of patients have confirmed the ability of Flagyl to cure trichomoniasis.

Correctly used, with due attention to repeat courses of treatment for resistant, deep-seated invasion and to the presumption of reinfection from male consorts, Flagyl has repeatedly produced a cure rate of up to 100 per cent in large series of patients.

Nothing cures trichomoniasis like Flagyl.

Dosage and Administration

In *women:* one 250-mg. oral tablet t.i.d. for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When the inserts are used one vaginal insert should be placed high in the vaginal vault each day for ten days, and concurrently two oral tablets should be taken daily.

In *men:* in whom trichomonads have been demonstrated, one 250-mg. oral tablet b.i.d. for ten days.

Contraindications

Pregnancy; disease of the central nervous system; evidence or history of blood dyscrasia.

Precautions and Side Effects

Complete blood cell counts should be made before and after therapy, especially if a second course is necessary.

Infrequent and minor side effects include: nausea, unpleasant taste, furry tongue, headache, darkened urine, diarrhea, dizziness, dryness of mouth or vagina, skin rash, dysuria, depression, insomnia, edema. Elimination of trichomonads may aggravate moniliasis.

Dosage Forms

Oral—250-mg.tablets/Vaginal—500-mg.inserts

SEARLE

Research in the Service of Medicine

TUMOR SEMINAR

Case 1/Tumor Seminar

Diagnosis. — Botryoid rhabdomyosarcoma of maxillary sinus.

Contributors.—Drs. A. M. Richmond, C. J. Farinacci, and H. C. Ford, Nix Clinical Laboratory, San Antonio.

History.—A 38-year-old white woman with a history of sinusitis, rhinitis, and hay fever, had a nasal polypectomy. Three weeks later, 43 gm. of sticky, mucinous portions of tissue, some with polypoid configuration, were removed from the right maxillary sinus.

DR. HORN: One section, representative of much of the tissue from this case, has normal respiratory epithelium at the top, and below, an edematous, relatively acellular structure much like a nasal polyp.

Submitted Diagnoses.—Fibromyxoma, 12; other fibromas, 3; myxoma, 2; embryonal rhabdomyosarcoma, 3; rhabdomyosarcoma, 2; myosarcoma, 1; nasal polyp, 5; nasal glioma, 1; scleroma, 1; chronic sinusitis, 1.

DR. HORN: Most of the diagnoses are in the fibromyxoma or fibroma, or myxoma group; relatively few pinpointed this as a rhabdomyosarcoma.

Portions of this lesion are much more cellular than the extremely edematous, superficial parts. The cells are fairly characteristic in having long, bipolar, tapering processes, and the cytoplasm is extremely eosinophilic. Individual cells show shrinkage of the cytoplasm away from the cell border, which is characteristic of many skeletal muscle tumors. Cross-striations are just barely visible in one section, but I believe they are definite. Even if they were not, I would still call this a polypoid rhabdomyosarcoma. The picture is typical, although the patient is much too old for the typical polypoid, botryoid, or embryonal rhabdomyosarcoma. To me, it simply demonstrates that older persons may have this type of tumor.

Much of the PAS-positive material in the tumor cell cytoplasm is digestible by amylase and is presumably glycogen, which supports the diagnosis but by no means proves it. Many rhabdomyosarcomas mimic fetal skeletal muscle, and cells may resemble normal muscle at the hollow-tube stage with the nuclei lying within the tubes. These tumor cells also can mimic adult rather than fetal skeletal muscle, and this is a more typical presentation than the seminar case.

One of the cases submitted for possible use in the seminar was a tumor of the orbit of a child. The orbit is a common location for these tumors, and they are common in small children. In another case submitted, the tissue was necrotic and had little viable tumor; for that reason I did not use it. It was a lesion of the flank, was thought to be an abscess, and was incised and drained. This has happened with many rhabdomyosarcomas in infants.

DR. GEORGE C. MANI,* San Antonio: Despite the paucity of correct diagnoses, I know that Dr. Horn welcomes discussion.

DR. RICHMOND: I can give a short follow-up on this case. About a year ago, a modified Caldwell-Luc operation was performed with wide exposure to the lip and alongside the nose, and a tumor with a rather small base was found arising from the antrum on its inner surface. The bone was necrotic posteriorly, and the tumor had extended into the nasopharynx. It was resected completely, and since then the patient has had an uneventful course.

Dr. Horn, would you care to discuss the course of the rhabdomyosarcoma, embryonal type, in this area, in relation to what a myxoma might be expected to do?

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*President of the San Antonio Society of Pathologists in 1964, and moderator of the Tumor Seminar.

Proceedings of the 21st Annual Tumor Seminar presented by the San Antonio Society of Pathologists at Brooke Army Medical Center, Fort Sam Houston, Texas, on Dec. 5, 1964. The conductor was Dr. Robert C. Horn, Jr., Director of the Department of Laboratories, Henry Ford Hospital, Detroit.

DR. HORN: I had four such cases of botryoid or polypoid rhabdomyosarcomas; one patient was a 16-year-old boy and the others were considerably younger. The 16-year-old boy died within a few months with intracranial extension of tumor and distant metastases. One of the children died of the tumor, but I do not know the details. The third patient was a 2½-year-old boy and he is well after 16 months, with surgical and x-ray treatment. I have no follow-up on the other case.

Incidentally, the published cases have had almost a universally hopeless prognosis, but more and more people tell me of patients who are surviving for various intervals—usually not five years—and it may be that the prognosis is not as hopeless as we have believed it to be.

I think the lung metastases, which are the most common, are blood borne, and they have been widespread throughout the lungs. In one of these cases there were other visceral metastases.

1. Farinacci, C. J., et al.: Sarcoma Botryoides (A Form of Embryonal Rhabdomyosarcoma) of Common Bile Duct; Report of Two Cases, Cancer 9:408 (March-April) 1956.
2. Horn, R. C., Jr., et al.: Rhabdomyosarcoma (Sarcoma Botryoides) of the Common Bile Duct; Report of Case, Cancer 8:469 (May-June) 1955.

Case 2/Tumor Seminar

Diagnosis.—Metastatic malignant melanoma.

Contributors.—Drs. A. M. Richmond, C. J. Farinacci, and H. C. Ford, Nix Clinical Laboratory, San Antonio.

History.—A 36-year-old white man, in good health, noticed a gradually enlarging painless mass in the right axilla. The chest x-ray showed no abnormalities, but at sur-

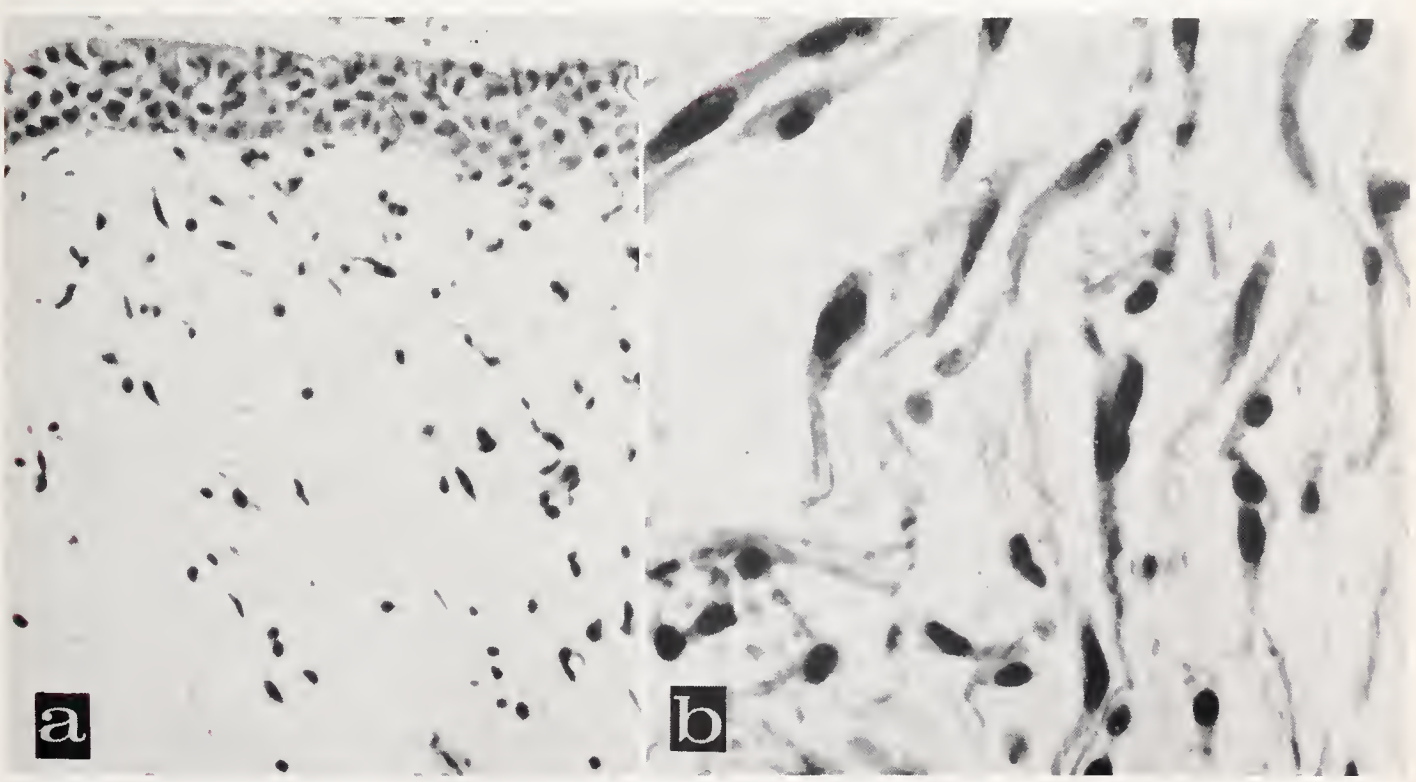


Fig. 1. Case 1. Botryoid (polypoid embryonal) rhabdomyosarcoma. a. Note edema and poor cellularity just beneath the mucous membrane. X 169.5. b. Scattered, nonstriated rhabdomyoblasts. X 440.7.

gery a globular mass of rubbery tissue surrounded by fat, measuring 10 by 6 cm., was removed. There was no attachment to skin, muscle, or nerves. Cut sections had a variegated appearance, ranging from gray-pink to areas of hemorrhage and necrosis. There were similar satellite lesions about the main mass.

DR. HORN: This case was presented to me as a problem in differentiation between metastatic malignant melanoma and alveolar rhabdomyosarcoma. Here is a fine example of an alveolar pattern, the cells being segregated in groups by a vascular, rather delicate stroma, which lines the alveolar spaces. A solid portion of the same tumor shows no suggestion of an alveolar pattern, or any pattern, for that matter. In one group of cells, the giant cell is obvious, but the others show margination of chromatin and prominent nucleoli, the so-called bird's eye nucleus which is common in malignant melanomas.

Submitted Diagnoses.—Metastatic melanoma, 11; melanoma, 10; alveolar soft-part sarcoma, 7; rhabdomyosarcoma, 2; granular-cell myoblastoma, 1; liposarcoma, 1; sebaceous adenocarcinoma, 1; metastatic squamous cell carcinoma in lymph node, 1.

DR. HORN: Melanoma, or metastatic melanoma (and I presume the diagnosis of melanoma means the same thing), is the prevailing diagnosis. Some of you recognized the alveolar pattern and were carried away with it.

After I discontinued the search for pigment in this tumor, I found some. It was difficult to photograph. An iron-uptake preparation, which I did not have until after I found the pigment in section stained with hematoxylin and eosin, is completely convincing. On the left is a bit of normal epidermis, used as a control for the iron uptake. I think this is definitely a metastatic malignant melanoma.

I once mixed up rhabdomyosarcoma and malignant melanoma, and am still not completely convinced of the answer in that case. The tumor was on the distal part of a man's thumb. When I first went to Henry Ford Hospital, the diagnosis was being debated. I went into the residents' conference and, fortuitously, saw under the microscope a cell with perfectly good cross-striations, which I showed to the residents at that time. We reported it as a rhabdomyosarcoma. Not long afterward metastatic nodes developed in the patient's axilla; these were removed elsewhere and were perfectly classical examples of malignant melanoma. I cannot explain the paradox, unless I saw a slide on another case, because those cross-striations were unequivocal, although I was never able to find them again. I assume that I made a misdiagnosis, but I do not have complete information about the patient.

DR. FARINACCI: This patient died shortly after his operation, and he had pulmonary metastases. There was no autopsy. The physician who had cared for this man for most of his life said that he never had any skin lesions or moles removed.

In the first slide that we had, we could not find the pigment at all, and we were strongly in favor of the alveolar cell type of rhabdomyosarcoma. Dr. Horn reminded us that rhabdomyosarcomas usually do not metastasize to the lymph nodes, but are vascular. In preparing these slides for the seminar, the first slide I picked up had one beautiful area of melanin pigment, so there is no question about it as far as we are concerned. Unfortunately, we do not know the primary site.

DR. MANI: Dr. Horn, the conductor of an earlier seminar, in discussing Hodgkin's disease, told us that he had "Reed-Sternberg cell hunters," and I wonder if you have "melanin versus striation hunters."

DR. HORN: I don't hunt for cross-striations as hard as I used to. Finding them is merely a matter of time and intensity of effort, and I know now that some rhabdomyosarcomas can't be expected to have cross-striations if they are mimicking fetal skeletal muscle, since skeletal muscle does not develop cross-striations until a fetal age of 10 or 11 weeks.

DR. J. J. ANDUJAR, Fort Worth: Dr. Farinacci, is there much concern about the fact that there is no skin primary on a metastatic melanoma?

DR. FARINACCI: No, but we like to find the primary if we can. I wonder also about the possibility of this being an orbital melanoma, but we have no report on that. We as-

sume that there wasn't any, but we cannot tell for sure.

DR. HORN: When you don't find the primaries, where do you assume they are?

DR. ANDUJAR: I was thinking about the orbit since this was a lymph node metastasis, but they also can occur in many places other than the skin.

DR. HORN: The ferrous ion uptake reaction is supposedly specific for melanin pigment. I was introduced to it by the Ann Arbor group. It is a Lillie technique, and is described in the third edition of "Histopathologic Technic and Practical Histochemistry."

Case 3/Tumor Seminar

Diagnosis.—Leiomyoma (round-cell variant) of jejunum.

Contributors.—Dr. D. L. Galindo and H. M. Perches, Robert B. Green Memorial Hospital, San Antonio.

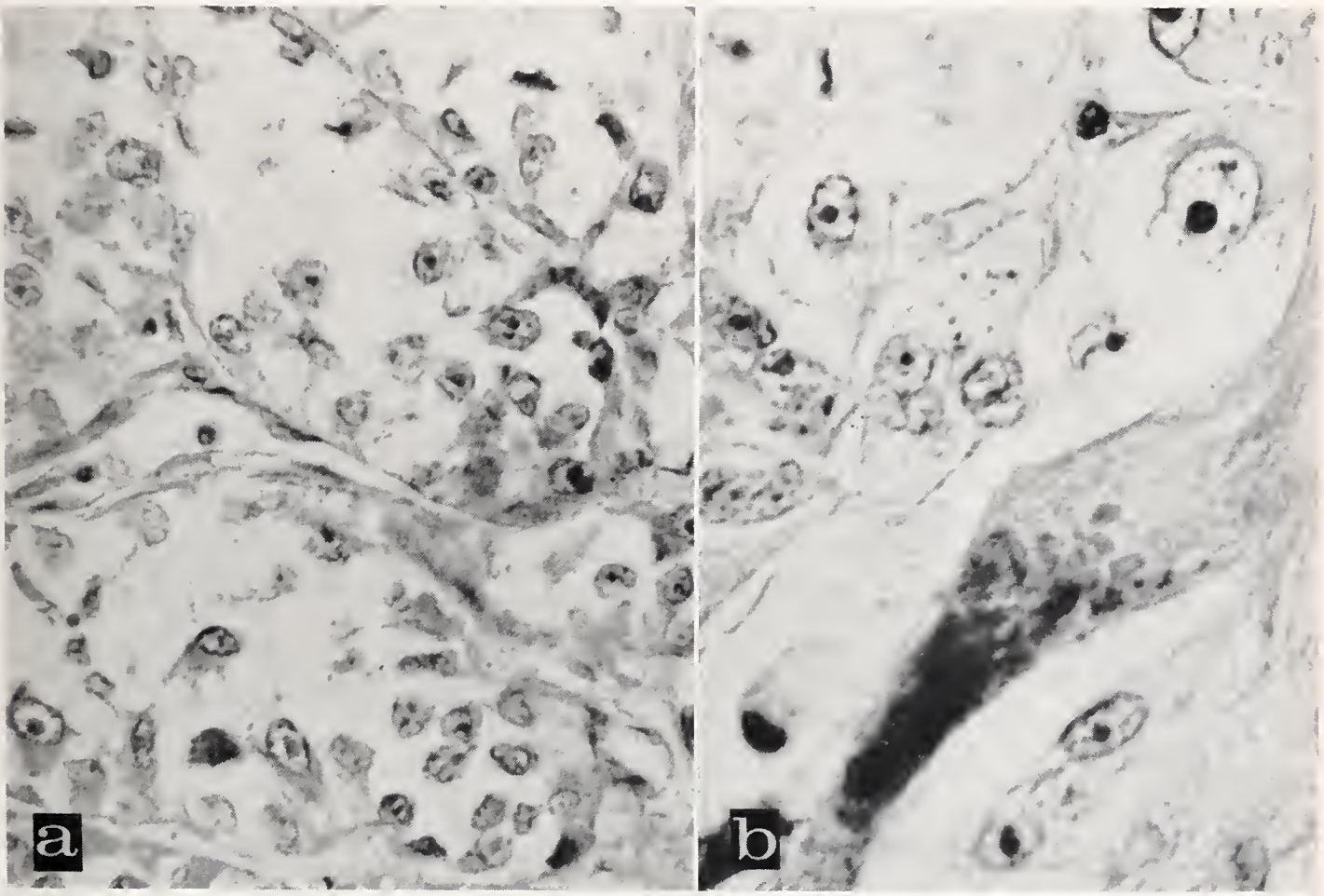


Fig. 2. Case 2. Metastatic malignant melanoma. a. Suggestion of alveolar pattern. X 267.5. b. X 417.3.

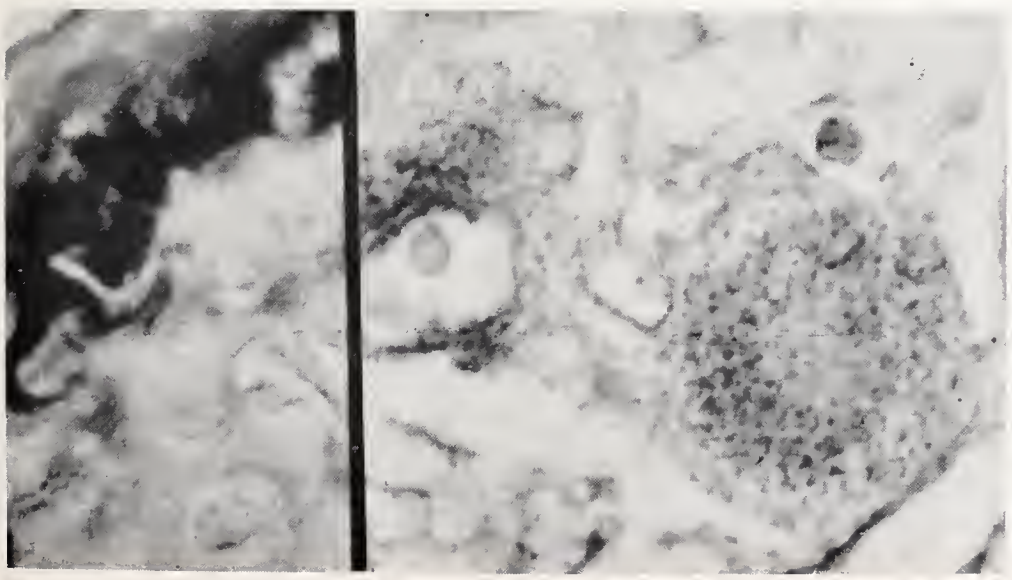


Fig. 3. Case 2. Ferrous ion uptake preparation demonstrating melanin pigmentation. X 1159.2. Left, normal basal epidermis serves as control.

History.—A 73-year-old man with diffuse gastrointestinal bleeding, but no vomiting, had a large mass in the greater omentum, connected with another in the jejunum. The irregular, nodular, semifirm, rubbery, gray-brown-yellow mass measured 12 by 9 by 7 cm. On section it was composed of outer nodules of lobular, fleshy, white-gray-yellow tissue blending with necrotic brown-red central areas.

DR. HORN: That certainly describes a malignant tumor. Much of the lesion is composed of rounded or polygonal cells, having no special pattern, and resembling no normal tissue. In some areas, the cells are not only bunched, but take on considerable pleomorphism and hyperchromatism. Other sections show a tendency to spindling of the cells and to arrangement in interlacing bundles, which definitely suggest smooth muscle to me in an area showing some necrosis, which also is characteristic of smooth muscle tumors.

Submitted Diagnoses. — Leiomyosarcoma, 19; hemangiopericytoma, 3; fibrosarcoma, 2; leiomyoma of small intestine, 2; fibrous mesothelioma, 2; lymphangioendothelioma, 1; malignant hemangioendothelioma, 1; malignant schwannoma, 1; liposarcoma, 1.

DR. HORN: Most of you recognized this as a smooth muscle tumor. Hemangiopericytoma may be closely related to the smooth muscle tumors; I think most "schwannomas" of the gastrointestinal tract are probably smooth muscle tumors. My diagnosis was leiomyoma of the small intestine. We have described a tumor of the omentum, but since the patient was bleeding from the gastrointestinal tract, with a hemoglobin of 6 gm. per 100 ml., we must assume that it is a lesion of the small intestine with a large component extending into the mesentery or omentum. This brings up the problem of malignancy in smooth muscle tumors as well as this peculiar round-cell or polygonal-cell appearance. In 1942, I tried to convince Dr. A. P. Stout that a round-cell tumor was a smooth muscle tumor. It was malignant, because it had metastasized from the stomach to the liver of a 14-year-old girl; but Dr. Stout disagreed. Recently, however, he wrote an article on "bizarre smooth muscle tumors." They are essentially round-celled tumors; he emphasized a clear area around the nucleus, and pointed out that these were, indeed, smooth muscle tumors. I think this one fits in that group. The necrosis, large size, pleomorphism, and cellularity have not been reliable guides to malignancy of smooth muscle tumors. The only reliable guide, and you can't always depend

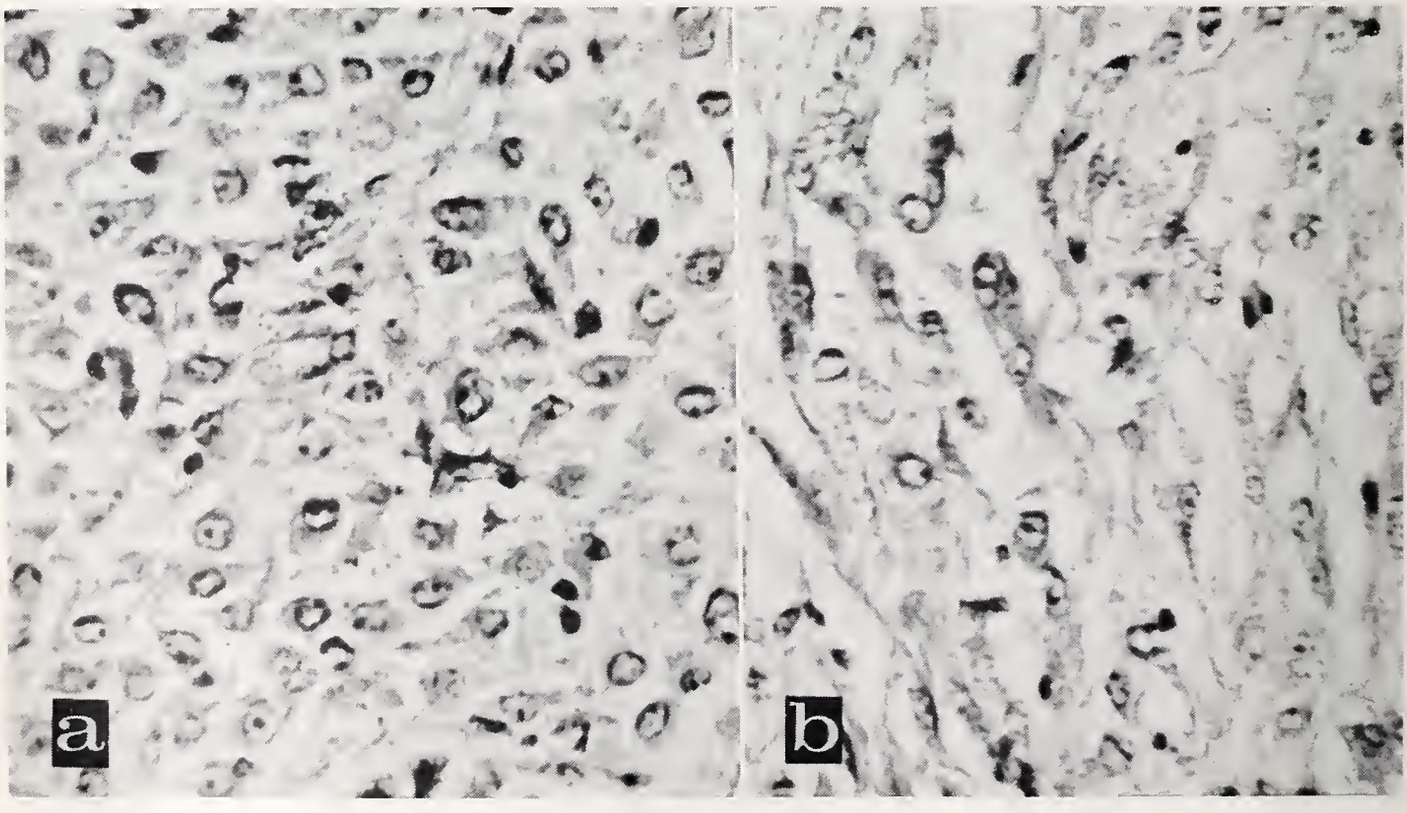


Fig. 4. Case 3. Leiomyoma (round-cell variant) of jejunum. a. Dominant round cell pattern. b. Spindle-celled area. Both X 295.

upon that, is the presence of large or appreciable numbers of mitotic figures. I did not find any in this tumor, and therefore I would call it benign.

Occasionally well-differentiated smooth muscle tumors without mitotic figures do metastasize, just to embarrass the pathologist.

DR. MANI: These tumors are often hard to classify, and I imagine this case will evoke some comments.

DR. GALINDO: This patient died as a result of peritonitis which developed from a wound infection following the operation. The tumor in the omentum was large, and was firmly connected to the tumor in the small intestine. At autopsy we found no other tumors, and the gross and microscopic examinations did not reveal any areas of metastases or any involved lymph nodes. The patient had terminal bronchopneumonia.

DR. HORN: Although necrosis is not an indication of malignancy, it is characteristic of smooth muscle tumors and causes them to bleed, usually into the lumen. One patient presented as an abdominal emergency; she had a smooth muscle tumor of the bowel, but it perforated the other way and bled into the peritoneal cavity.

1. Stout, A. P.: Bizarre Smooth Muscle Tumors of the Stomach, Cancer 15:400 (March-April) 1962.

Case 4/Tumor Seminar

Diagnosis.—Malignant lymphoma of stomach.

Contributors.—Drs. A. O. Severance, H. A. Van Auken, and M. W. Delmer, Baptist Memorial Hospital, San Antonio.

History.—A 67-year-old woman had a peptic ulcer on the posterior wall of the antrum. It failed to heal after conservative therapy. Gastrectomy revealed diffuse thickening of the distal 8 cm. of the stomach with a focal area of small ulceration. The surgeon's impression was linitis plastica. Normal lymph nodes accompanied the specimen.

DR. HORN: There is a give-away in this history. When the surgeon thinks he is dealing with a linitis plastica, I always make a diagnosis of lymphosarcoma.

Submitted Diagnoses.—Pseudolymphoma, 14; reactive lymphoid hyperplasia, 6; chronic gastritis, 5; malignant lymphoma, 7; hemangiopericytoma, 1.

DR. HORN: I suppose reactive lymphoid hyperplasia is the same as pseudolymphoma, and therefore we have, three to one, pseudolymphoma or inflammatory lesion over malignant lymphoma. The real problem is whether we are dealing with an inflammatory lesion or a malignant tumor. At the edge of a shallow ulcer, the mucous membrane is barely penetrated by the tumor or by the ulceration. In the cellular infiltrate, there are areas suggestive of reactive or

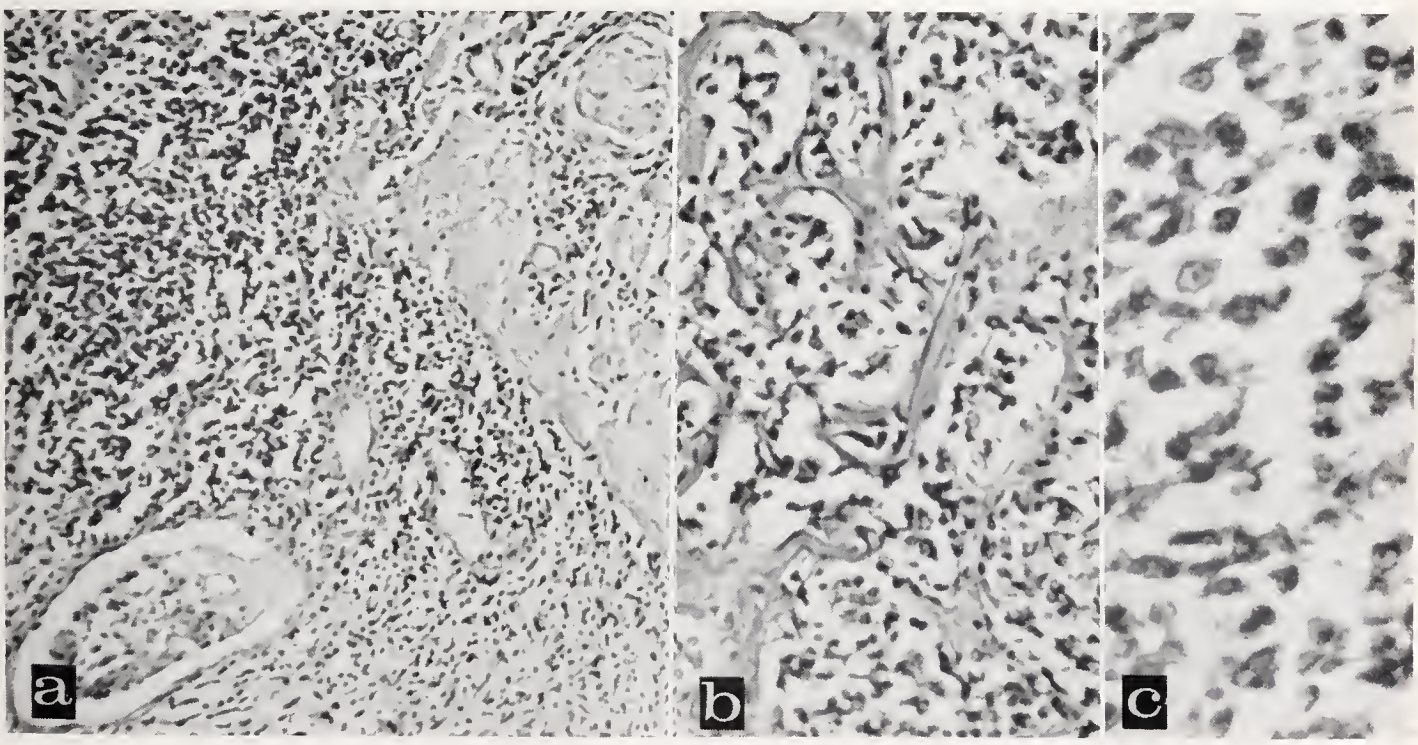


Fig. 5. Case 4. Malignant lymphoma of stomach. a. X 82.8. b. X 138. c. X 358.8.

germinal centers, but they are not striking and maybe they are nothing more than foci where the cells are packed a little bit tighter than elsewhere. In some portions of the lesion there is hyaline fibrosis. The higher magnification shows uniformity of cell size and shape. They are essentially small cells, and are of the order of lymphocytes. After long and hard study, I diagnosed this as malignant lymphoma. Jacobs compared pseudolymphomas, or lymphoid inflammatory lesions, with malignant lymphomas. He said the distinction can be easy, and maybe it is if you have a group of cases and can study them after the fact. The distinction in a given case is not easy. The presence of reaction centers would favor the diagnosis of pseudolymphoma, but in this case the reaction centers are neither conspicuous nor definite. The size of this lesion would favor its being a tumor. The superficiality and relatively inconsequential appearance of the ulcer would suggest that it is secondary, or an ulceration of a tumor rather than a primary peptic ulcer with secondary inflammation. The age of the patient in this instance would favor malignant lymphoma. I cannot determine whether there is real venous invasion or not, but some of the tumor cells, or reactive cells if that is what they are, are within vessels, and there is perineural involvement as well. These are seen fairly extensively in the lesion. The muscle is widely involved in this case, although not massively; the tongues of the infiltrative process are going out into the muscular coat, whereas the bulk of the tumor is in the mucous membrane and submucosa. The uniformity of cell type favors the interpretation of malignant lymphoma; on the other hand, the lymphocytic type of lymphoma is not common among the gastrointestinal lymphomas. They are usually the reticulum cell type, or, more often, pleomorphic or polymorphic.

In inflammatory lesions, there is often the impression of ulceration, although it may not be deep, and of the presence of an ulcer, rather than the ulceration of the tumor mass. This was my impression in this case. A superficial infiltrate, and the presence of reaction centers, are considerably more convincing. Inflammatory lesions may produce a variable cellular infiltration of all sorts of cells, including plasma cells, eosinophils, and polymorphonuclear leukocytes in addition to larger reticulum cells. In our case of malignant lymphoma, a lymphocytic type of malignant lymphoma, there are other cells in the infiltrate, but mostly they are in the superficial part of the mucous membrane, where they may be expected in any case.

DR. SEVERANCE: The patient is well and healthy, but it has been only about a year since the operation.

We looked at this with a jaundiced eye, and we wondered (we like to make lymphosarcoma diagnoses, too); but we saw the small cells, germinal centers, and lymphoid follicles, and we were undecided. Then we read the article that you referred to, and we diagnosed pseudolymphoma.

DR. HORN: There is a primary lymphoma of the stomach. Those of the rest of the gastrointestinal tract, in my experience, have all turned out to be part of disseminated lymphoma.

DR. DELMER: In 1960, Dr. Elson Helwig presented a case which he called a pseudolymphoma, and which most of us thought was a true lymphosarcoma. At that time he said he had 130 or 160 cases which he was going to publish. Since they did not appear in print, I felt that maybe some of them were less pseudo- than lymphoma. He called this case a pseudolymphoma, but he said that there were some gray zones.

DR. G. D. LUNDBERG, Fort Bliss: Dr. Helwig said that he was still going to publish this series, and that his feeling was still the same.

DR. HORN: This is the story of our lives; we see malignant lymphomas of the stomach, and some of them don't behave in malignant fashion, and we recognize pseudolymphomas. Soon we have too many pseudolymphomas, and maybe metastasizing ones. We have done this with keratoacanthomas, and with adenosis of the breast, and maybe we are in danger of doing it with the stomach, too.

DR. L. LOWBEER, Tulsa: I believe these articles which have been talked about, or published, have played havoc with the diagnosis of lymphosarcoma of the gastrointestinal tract. One of the arguments for calling this a pseudolymphoma is the longevity of patients with these lesions. This is no argument whatsover. For that same reason, the follicular lymphoma—Brill-Symmers disease—for years had been considered as a benign disease until it was found that every one of the patients had died from it,

although it sometimes took 15 or 20 years. If this publication will be delayed for 15 or 20 years, there won't be anything to publish, and many cases which have already backfired will not be in the series. If this infiltration extends into the muscle layer, one cannot conceivably talk about benignity. If there is some reaction at the periphery, this is also no argument, since reactive lesions can be found in the vicinity of malignant ones, and even reactive lymphoid lesions in the vicinity of malignant lymphoid lesions. The criteria has to be far more strict. I believe that pseudolymphomas occur, but are more rare than the real ones.

1. Jacobs, D. S.: Primary Gastric Malignant Lymphoma and Pseudolymphoma, Amer. J. Clin. Path. 40:379 (Oct.) 1963.
2. McNeer, G., and Berg, J. W.: The Clinical Behavior and Management of Primary Malignant Lymphoma of the Stomach, Surgery 46:829 (Nov.) 1959.

Case 5/Tumor Seminar

Diagnosis.—Rhabdomyosarcoma (mixed pattern).

Contributor.—Captain G. E. Branam, MC, Wilford Hall U.S. Air Force Hospital, Lackland Air Force Base.

History.—A 28-year-old white man was treated with bed rest and anticoagulants for thrombophlebitis. Three to four months later a mass developed in the right calf. A fairly well-encapsulated nodular mass measuring 14 by 7 by 4.5 cm. was excised. The cross section was tan-yellow with focal areas of mucoid degeneration. The tissue was firm but not hard.

DR. HORN: I did not know about the thrombophlebitis. My diagnosis was rhabdomyosarcoma. I think the tumor has some of the features of the alveolar, embryonal, and pleomorphic types, and it is difficult to classify.

Some lining-up of cells might suggest an alveolar pattern, but this is not well developed. In one lobule, the tumor cells are finely vacuolated, suggesting that this might be an area of high glycogen content. Incidentally, this tumor was PAS-positive, but not nearly to the extent of the first case. There is an area where the cells are polygonal or rounded, show little differentiation, and simulate the embryonal rhabdomyosarcoma, showing not the small spindle-shaped cells, but rather the larger, rounded cells. They do have considerable bright, eosinophilic cytoplasm. In an area of the same tumor, the cells are spindled. They suggest the pleomorphic adult rhabdomyosarcoma more than they do the embryonal.

Submitted Diagnoses. — Rhabdomyosarcoma, 7; embryonal rhabdomyosarcoma, 6; alveolar soft-part sarcoma, 3; hemangiopericytoma, 5; malignant hemangiopericytoma, 2; clear-cell hemangioendothelioma, 1; liposarcoma, 3; metastatic carcinoma, 2; syno-

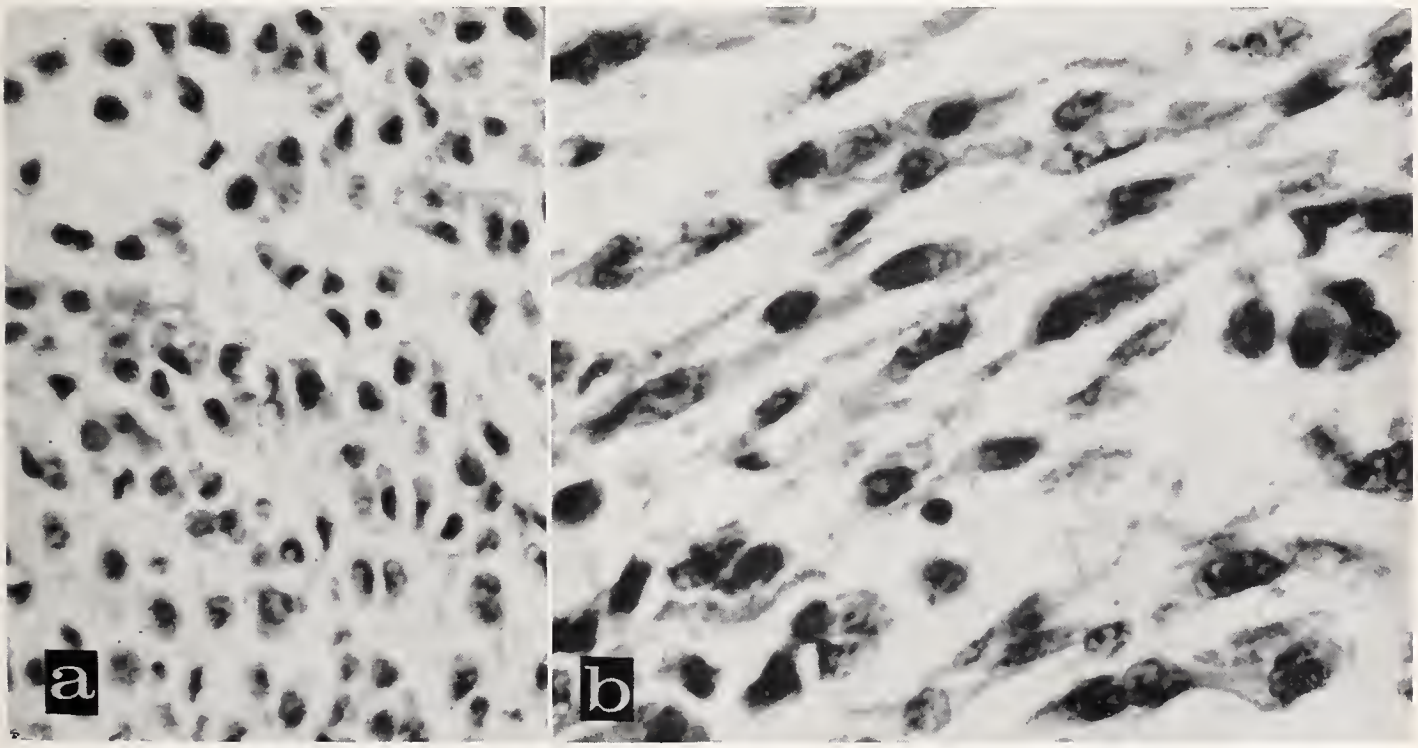


Fig. 6. Case 5. Rhabdomyosorcomo. a. Suggestion of alveolar pattern. X 270. b. Rhabdomyoblosts with abundant eosinophilic cytoplasm. X 421.2.

vial sarcoma, 1; malignant histiocytoma, 1; glomus, tumor, histologically malignant, 1.

DR. HORN: Metastatic carcinoma can be simulated by alveolar rhabdomyosarcomas; it seems a ridiculous differential problem, but it does occur.

Dr. Stout has taken exception to the term "alveolar," and insists on labeling these tumors "embryonal" rhabdomyosarcomas. I have no brief for the term "alveolar"; whether or not it is recognized as a separate type is not really important. The behavior is much like the embryonal rhabdomyosarcomas, and the main point is that it is important to recognize tumors with this pattern as rhabdomyosarcomas. If we do that, their subclassification is unimportant.

A completely undifferentiated tumor from the pleural cavity or the chest wall of an 18- or 20-year-old girl shows embryonal skeletal muscle in a completely indifferent stage. This is in the embryo of about seven or eight weeks. This structure looks like a greatly elongated muscle fiber. Autopsy was performed, and the tumor extended into the mediastinum, and from the mediastinum up into the neck organs. I do not believe she had distant metastases. Where the tumor invaded the thyroid gland, it showed some differentiation, large cells with multiple nuclei, and a lot of good, pink cytoplasm. Portions of proven rhabdomyosarcomas show no differentiation whatsoever.

DR. J. GUILLEBEAU, Wilford Hall Hospital: I have a follow-up on the patient in the seminar case. About one year later he was still living, but had multiple pulmonary lesions.

1. Enterline, H. T., and Horn, R. C., Jr.:Alveolar Rhabdomyosarcoma; A Distinctive Tumor Type, Amer. J. Clin. Path. 29:356 (April) 1958.

2. Patton, R. B., and Horn, R. C., Jr.: Rhabdomyosarcoma: Clinical and Pathological Features and Comparison with Human Fetal and Embryonal Skeletal Muscle, Surgery 52:572 (Oct.) 1962.

3. Riopelle, J. L., and Theriault, J. P.: An Unknown Type of Soft Part Sarcoma: Alveolar Rhabdomyosarcoma, Ann. Anat. Path. 1:88 (Jan.-March) 1956.

Case 6/Tumor Seminar

Diagnosis.—Granular cell myoblastoma.

Contributor.—Captain W. R. Beach, MC, Fourth U.S. Army Medical Laboratory, Fort Sam Houston.

History. — A 23-year-old asymptomatic man had a 3 by 5 cm. mass palpable at the base of the penis. Minimal compression of the membranous portion of the urethra was noted on cystoureterograms. At surgery the mass was hard, firmly adherent to the periurethral tissue, and multinodular. The tumor surrounded the urethra and extended bilaterally to involve both lateral crura. The urethral lumen was not involved.

DR. HORN: This lesion is striking in that a tremendous amount of dense, collagenous tissue makes up its greater portion. Scattered through it, however, are small nests, and sometimes clusters of nests of large cells with granular cytoplasm and relatively small nuclei.

Submitted Diagnoses.—Granular cell myoblastoma, 29; Peyronie's disease of penis, 3.

DR. HORN: Some of the granular cell myoblastomas present as extensive fibrous masses, with the granular cell elements widely scattered. Perhaps these elements were not in the sections from which the diagnoses of Peyronie's disease were made.

Granular cell myoblastoma has nothing in common with fetal muscle. In the hollow-tube stage, with the fibers cut in cross section, one can see the fibrils packed around the periphery of most of these fibers, but they are not yet filled up the center. Next, myofibrils gradually fill the hollow space in the tube, and then the fiber is essentially filled. The next stage is the migration of the nuclei to the periphery, and then the gradual enlargement of the cell, the individual fiber, until it reaches adult appearance. There is no reason to think of the granular cell myoblastoma as being myoblastic in terms of any resemblance to fetal muscle.

A tumor was sent to me in great glee by Dr. John Fust of Fust and Custer; they were the first to suggest a possible neurogenous origin of granular cell myoblastoma. This was a whole tumor, and it certainly had the appearance and configuration of a nerve. A section from within the seminar lesion had the configuration of a nerve, with accumulation of granular cells within what appeared to be the nerve sheath. A still higher magnification showed morphologic identity with the granular cell myoblastoma. There is good histochemical evidence from several different sources allying the granular cell myoblastoma with nerve tissue, or with a neurogenous origin. I think Fisher's electron microscope studies are convincing in tracing of the schwannian origin of the granular cell "myoblastoma." His

article was in *Cancer;* it was disappointing to me because it was less convincing than the original electron micrographs. I have wondered whether there might not be two granular cell myoblastomas, one of nerve sheath origin, and growing very much like neurofibromas do, and one of some other origin, possibly, but certainly not definitely, muscle.

CAPT. H. R. McDANIEL, Fourth U.S. Army Medical Laboratory: Our diagnosis was the same as Dr. Horn's. Six weeks following removal of the tumor the patient had a severe stricture which was believed to be extra-urethral in origin. The stricture was treated by dilation, and the patient was discharged from the Army. He was last seen at a Veterans Administration hospital, nine months postoperatively. There was no evidence of recurrence and no stricture.

DR. W. G. SPRAGUE, Lackland Air Force Base: I could add a little fuel to the granular cell problem. In oral pathology we have a granular-cell variant of ameloblastoma; to my knowledge it has not been investigated histochemically because there is insufficient material. About ten years ago I first heard of the granular-cell variant of ameloblastoma; this is an interesting variant that certainly would add confusion to the problem.

DR. HORN: There is also congenital epulis, which has been called a granular cell myoblastoma, and which many people believe is a distinctive lesion.

DR. E. DeCOURSEY, San Antonio: When you think of everything in the body coming from two cells, you can imagine anything coming out one way or other.

DR. L. B. STARR, Fort Benning, Ga.: We had a case of a young soldier who had an appendectomy. At that time there was nothing noted in the skin overlying the abdomen in the right lower quadrant. There were no sequelae; however, four or five months after the operation a pea-sized lesion developed in the scar, and in about six more months it enlarged to 0.5 cm. When removed it was a classical granular cell myoblastoma. This lesion was completely within the scar.

DR. HORN: One wonders if many different lesions look alike.

DR. RICHMOND: Is there an adequate explanation for the fact that many of these occur in the midline, or close to it?

DR. HORN: I didn't realize that they did. Is that a fact?

DR. GALINDO: I saw a case in which multiple lesions involved the inguinal region as well as the breast and along the axillary line; we thought this was granular cell myoblastoma of the malignant type.

DR. HORN: Did you interpret those as metastases?

DR. GALINDO: I don't remember how we interpreted it, but the masses in the breast were large and malignant and the cells were irregular. The granular cells were evident, but diffuse.

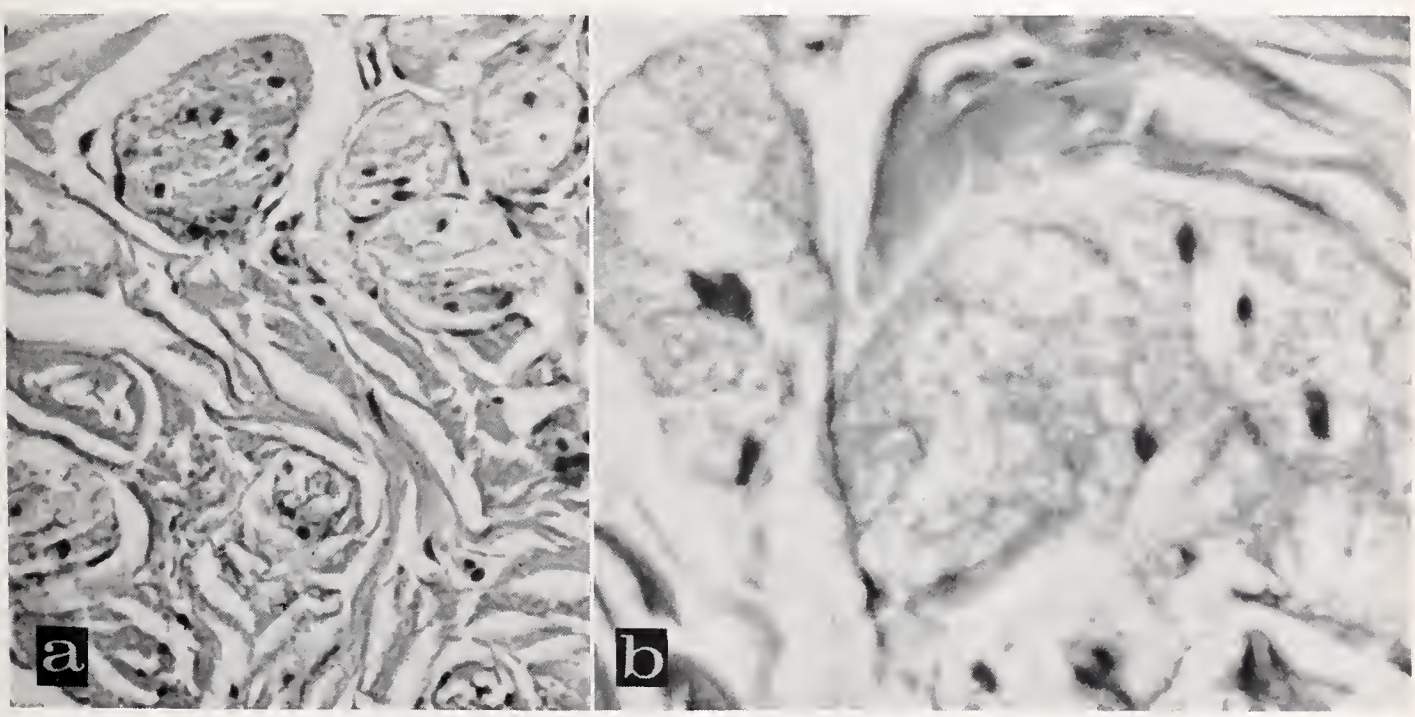


Fig. 7. Case 6. Granular cell myoblastoma. a. Fibrosis. X 154.5. b. Detail. X 334.8.

DR. HORN: In the few cases of multiple skin tumors that I have seen, the individual tumors seemed to grow like neurofibromas. This fits with the possibility of their neurogenous origin.

DR. LUNDBERG: Dr. Horn, do you think the so-called malignant granular cell myoblastoma is related to the benign entity we are discussing?

DR. HORN: There are two malignant granular cell myoblastomas; one looks exactly like this, and rarely does metastasize. I have seen a few in inguinal lymph nodes. The other one has been called malignant organoid granular cell myoblastoma, alveolar soft part sarcoma, and paraganglioma. But it is all the same tumor, as far as I am concerned.

1. Fisher, E. R., and Wechsler, H.: Granular Cell Myoblastoma—A Misnomer; Electron Microscopic and Histochemical Evidence Concerning Its Schwann Cell Derivation and Nature (Granular Cell Schwannoma), Cancer 15:936 (Sept.-Oct.) 1962.
2. Fust, J. A., and Custer, R. P.: On the Neurogenesis of So-Called Granular Cell Myoblastoma, Amer. J. Clin. Path. 19:522 (June) 1949.

Case 7/Tumor Seminar

Diagnosis.—Localized fibrous mesothelioma of pleura.

Contributors.—Drs. A. O. Severance, H. A. Van Auken, and M. W. Delmer, Baptist Memorial Hospital, San Antonio.

History.—A 44-year-old asymptomatic white woman had a lesion at the periphery of the lung. The firm, fibrous tumor weighed 138 gm., measured 9.5 by 6 by 5 cm., and was covered by pleura.

DR. HORN: Much of this tumor is fibrous, with a good deal of collagen; it is relatively poor in cells, and the cells are essentially small, dark spindles. There is no specific arrangement. This has been described as "a disordered tangle of fibers." Throughout, there are a few small cysts, or gland-like spaces. It is hard to see the cells that are lining them, but they do not look like endothelial cells. I presume they are epithelial or epithelioid. These two kinds of tissue together make up the fibrous mesothelioma.

Submitted Diagnoses.—Fibrous mesothelioma of pleura, 15; neurofibroma, 6; pulmonary fibroleiomyoma, 3; leiomyoma, 3; fibroma, 2; fibroxanthoma, 1; hamartoma, 1; neurilemoma, 1.

DR. HORN: Fibrous mesothelioma is correct; in the older literature, it has been called fibroma and fibrosarcoma. Perhaps some of the fibroxanthomas of the lung are really fibrous mesotheliomas, but I do not believe there is any smooth-muscle component in this lesion. Some fibrous tumors have been associated with hypoglycemia, and the hypoglycemia has been cured by removal of the tumor. An epithelium-like appearance characterizes the typical fibrous mesothelioma. I have seen another tumor, from the retroperitoneum, which was associated not only with hypoglycemia, but also with virilism. This was a metastasizing tumor and ultimately killed the patient. The point is that this has been reported as hemangiopericytoma. I cannot see the difference between this and the tumors that have been called fibrous mesotheliomas, and I do not know why this can't be a peritoneal mesothelioma. There is a solid, more or less epithelioid-appearing portion of the tumor, and a spindle area. It shows considerable vascularity, but there is no reason why many of these tumors should not have a goodly number of vessels. I wonder if all of the spindle-cell tumors with hypoglycemia are not in reality the same tumor.

We can categorize the mesotheliomas as fibrous or tubular or papillary, localized or diffuse; and benign or malignant. This makes quite a combination when we realize how relatively infrequent mesotheliomas are. In the pleura, localized fibrous mesothelioma is the most common. Occasionally the fibrous tumors may be much more cellular than this one, and may be malignant. This tumor apparently was not within the lung substance, but the few malignant ones have more often been within the lung itself. This I would interpret as a benign tumor.

DR. SEVERANCE: This patient was seen recently, and roentgenograms of the chest were normal. The patient felt fine.

DR. MANI: Dr. Horn, one of the diagnoses was a fibroxanthoma, and you said that was a variant of a fibroma. Would you explain this point more fully?

DR. HORN: I was thinking of the so-called pulmonary pseudotumor, or postinflammatory pseudotumor, which I suspect is the same thing as a fibrous mesothelioma. I have never recognized it as a distinctive lesion, and I wonder if the fibroxanthoma might not be the same thing. I don't know.

DR. ANDUJAR: Have you had any experience with classifying mesotheliomas and predicting their biological behavior by the presence of abnormal mitoses? Silver, Cox and others have stressed the importance of triaster and quadripolar mitoses. Do you have any feelings about predicting what degree of malignancy any specific mesothelioma will have?

DR. HORN: I have not seen enough mesotheliomas to think of them in such terms. Generally speaking, abnormal mitoses make me leery of any tumor.

DR. ANDUJAR: Some of these tumors will show abnormal mitoses which we usually associate with a high degree of anaplasia, and yet they behave in a benign fashion, and therefore are the only tumors with which this is the case.

DR. HORN: I have no experience with that.

DR. DeCOURSEY: I would like to invite attention to the biology of mitoses, and ask whether if one sees many, many abnormal mitoses, won't this tumor act benign, and shouldn't it, because if there are so many abnormal mitoses there will not be many offspring cells?

DR. HORN: Presumably, the abnormal mitoses will not give rise to cells that will harm the individual, but doesn't the tumor with many abnormal mitoses have other anaplastic cells that are going to reproduce their kind by normal mitoses?

DR. ANDUJAR: I would not want to take issue with such a distinguished pathologist as Dr. DeCoursey, but in every tumor excepting mesothelioma, abnormal mitoses are almost *ipso facto* the sign of a high degree of anaplasia, because it is true that the quadripolar, the triaster, the wild mitoses do not reproduce themselves, but along with them are tremendous numbers of normal mitoses which will reproduce. Therefore, if you see them you automatically think of a malignant tumor; but in mesothelioma there can be some fields showing abnormal mitoses. Yet, this is a benign tumor biologically.

DR. HORN: I didn't appreciate mitoses at all in this case, but I did not specifically look for them.

Dr. RICHMOND: Not infrequently we see bizarre mitoses in fibroxanthomas, particularly about the ear; we also see them in pseudosarcomas of the larynx.

DR. HORN: I have thought that some fibroxanthomas with abnormal mitoses were malignant, but they did not behave that way.

DR. DeCOURSEY: Are we speaking, then, of quantitation: what we do with chemicals, with irradiation, is to make many, many more abnormal mitoses so we will kill the tumor.

DR. S. C. McCLURE, San Antonio: I wish you would discuss the hypoglycemia associated with tumor.

DR. HORN: Dr. Lowbeer has the classic review of tumors associated with hypoglycemia.

DR. LOWBEER: I reviewed about 26 of these cases and all belonged, microscopically, to the type which Dr. Horn has described. All of them also were huge, weighing any-

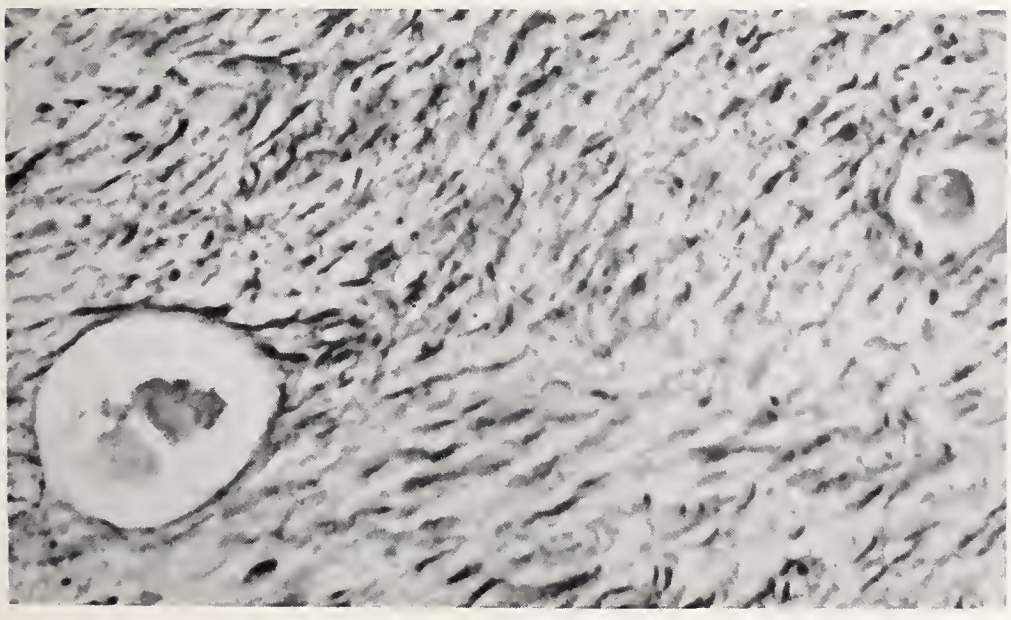


Fig. 8. Cose 7. Locolized fibrous mesathelioma af pleuro. It is composed of heavily collagenized fibraus tissue, locking ony particular arientotion, but enclasing gland-like spoces. X 157.5.

where between 1,000 and 7,000 gm., and virtually all were either above or below the diaphragm, and either pleural or retroperitoneal. In three of these cases (and in more since then) insulin assays were positive for an insulin-like substance. Beyond that, however, nothing is known. The histologic diagnoses are variable but generally they are all spindle-celled tumor, and all of them are huge. There are two other types of tumors associated with hypoglycemia: primary malignant tumors of the adrenal glands and of the liver. These also are large. Therefore, if this tumor does produce an insulin-like substance, it is very little per weight unit of tumor. The tumors have to be extremely large to produce these symptoms. The proof of this is that if they recur, the symptoms which disappeared after removal recur also. All the symptoms recur in the metastases thereof. About 75 percent of these have been malignant.

1. Benoit, H. W., Jr., and Ackerman, L. V.: Solitary Pleural Mesotheliomas, J. Thor. Surg. 25:346 (April) 1953.
2. Stout, A. P.: Mesotheliomas of the Pleura and Peritoneum, J. Tenn. Med. Ass. 44:409 (Oct.) 1951.
3. Stout, A. P., and Himadi, G. M.: Solitary (Localized) Mesothelioma of the Pleura, Ann. Surg. 133:50 (Jan.) 1951.

Case 8/Tumor Seminar

Diagnosis. — Malignant neurilemoma of sciatic nerve arising in neurofibroma in von Recklinghausen's disease.

Contributors.—Drs. A. O. Severance, H. A. Van Auken, and M. W. Delmer, Baptist Memorial Hospital, San Antonio.

History.—A 41-year-old white man with von Recklinghausen's disease complained of pain and swelling in his left thigh for six months at a site from which a large, sciatic neurofibroma had been removed five years previously. Biopsy of the tumor was followed by hip disarticulation. There were multiple neurofibromas of the skin, and involving the sciatic nerve was a fusiform tumor mass 10.5 by 4 by 3.5 cm. The cut surface was mottled dark brown to gray to orange, and the nerve seemed to be incorporated in the tumor.

DR. HORN: The amputation specimen shows neurofibromatous change in the periphery of the sciatic nerve. It is a characteristic appearance. Essentially it looks as though the nerve trunk is edematous. The biopsy specimen shows a cellular tumor which tends to be spindle-celled in some places and round-celled in others, and is not characteristic of anything, to my eye. In another area from the biopsy, we see almost all round cells, with a little spindling. In one area there is almost a cartilaginous appearance. In the amputation specimen, the major trunk of the sciatic nerve is described as being neurofibromatous, and in the sections it looks so; but in the center of portions of the tumor there is a cellular appearance—round cells essentially, with a great deal of pleomorphism, hyperchromatism, mitotic activity, and again, no specific pattern and no way to relate this to any special kind of tumor.

Submitted Diagnoses.—Malignant schwannoma, 16; neurofibrosarcoma, 6; malignant neuroepithelioma, 3; neurogenic sarcoma, 1; cellular neurilemoma, 1; liposarcoma, 1; fascinating leiomyoma, 1; ependymoblastoma, 1; hemangiopericytoma, 1; hemangioendothelioma, 1; neuroepithelioma, 1.

DR. HORN: The plurality have called this a malignant schwannoma; neurofibrosarcoma presumably means the same thing. If we restrict the term sarcoma to tumors of connective tissues, or to mesenchymal tissues, and regard the nerve sheath elements as being of ectodermal origin, then the term sarcoma is incorrect, although many people use it for this tumor. Neurogenic sarcoma, again, is the same thinking. Perhaps this is a neuroepithelioma; I had not thought of the round, almost pavement-like, cells as being epithelial, but perhaps they are. I find it difficult to believe that this is simply a cellular neurilemoma and not a malignant tumor. I agree with the majority; I think this is a malignant neurilemoma, or if you prefer, malignant schwannoma, arising in a neurofibroma of von Recklinghausen's disease. I have no specific way to recognize this as neurilemoma, and my principal reason for doing so is that I see what I think is a malignant tumor in a neurofibroma in a patient with von Recklinghausen's disease, and I conclude that it has to be a malignant neurilemoma. Tissue removed from this patient earlier showed typical neurofibroma without evidence of malignancy; it had areas of spindle cells and of edematous appearance, and was quite cellular. However, this is seen in some neurofibromas and is not necessarily evidence of malignancy. Another section showed considerable amounts of collagen forming wavy fibers or bundles of fibers.

Two publications from the Mayo Clinic have dealt with malignant neurilemomas; one series was in patients without von Recklinghausen's disease, and the other was in patients with von Recklinghausen's disease. Almost half of the malignant tumors in the patients with von Recklinghausen's disease were not considered neurilemomas. Some were called probable rhabdomyosarcomas, and one liposarcoma, and several were unclassifiable. I have not had this experience, but perhaps I have been too generous with the diagnosis of malignant neurilemoma, simply on the basis of established von Recklinghausen's disease or neurofibroma. I do not know the incidence of malignancy in neurofibromatosis because we have no clear idea of the incidence of neurofibromatosis in the population at large. If we consider only the hospital populations, and numbers of people with malignant tumors, I think we get a distorted idea of the incidence of malignancy in this disease.

DR. SEVERANCE: Recently, this patient was seen by the doctor who removed this specimen. So far, the patient is well and has no evidence of recurrence or metastasis.

DR. HORN: He has other neurofibromas?

DR. SEVERANCE: Yes, he has multiple neurofibromas all over his body. Five years ago, when this tumor came to my attention, I diagnosed it as a cellular neurofibroma. Maybe an occasional mitosis was there, but I dismissed that because the periphery of the lesion indicated a simple, ordinary neurofibroma. When I saw it five years later looking cellular and malignant and was told that it occurred in approximately the same site, I reexamined that first slide with another point of view—did I miss a malignant change in the neurofibroma? I sent it to Dr. Stout, who concurred that the original neurofibroma was benign. He did call this succeeding tumor a malignant schwannoma. As for the rather round cells which attracted our attention, they remind me of gemistocytes in a glioma, such as we see in the brain. All I can say is that there are additional neurofibromas in this same sciatic nerve, both above and below the malignant tumor, but they look like ordinary neurofibromas.

DR. HORN: I agree with Dr. Stout; I don't think you missed a malignant tumor the first time around.

DR. DeCOURSEY: When I cut a cylinder of bologna, straight across, it is round; and when I cut it lengthwise, it is spindle-shaped. Are these round cells really round, or just in cross section?

DR. HORN: I don't know. They look

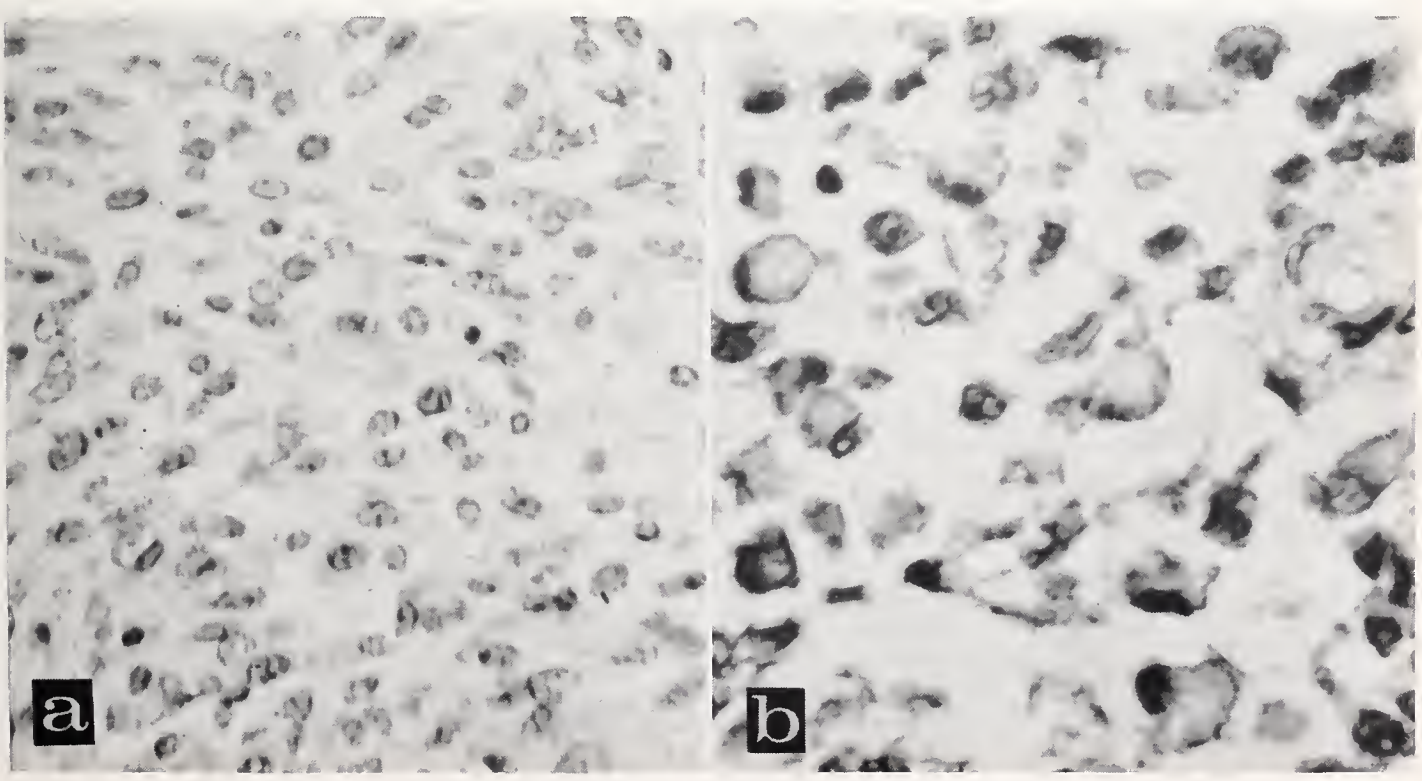


Fig. 9. Case 8. Malignant neurilemoma arising in neurafibrama of sciatic nerve (van Recklinghausen's disease). a. X 280. b. X 436.8.

round, but they may be cut in cross section.

DR. SEVERANCE: They aren't bologna.

1. D'Agostino, A. N., et al.: Primary Malignant Neoplasms of Nerves (Malignant Neurilemomas) in Patients Without Manifestations of Multiple Neurofibromatosis (von Recklinghausen's Disease), Cancer 16:1003 (Aug.) 1963.
2. D'Agostino, A. N., et al.: Sarcomas of the Peripheral Nerves and Somatic Soft Tissues Associated with Multiple Neurofibromatosis (von Recklinghausen's Disease), Cancer 16:1015 (Aug.) 1963.

Case 9/Tumor Seminar

Diagnosis.—Spindle cell epidermoid carcinoma of floor of mouth.

Contributors.—Drs. A. O. Severance, H. A. Van Auken, and M. W. Delmer, Baptist Memorial Hospital, San Antonio.

History.—A 64-year-old white woman had a polypoid tumor filling the floor of the mouth. Two years previously, after a diagnosis of carcinoma of the tongue, radium needles had been implanted but were promptly removed by the patient, and no further treatment had been given prior to excision of the polypoid masses.

Submitted Diagnoses. — Epidermoid carcinoma with pseudosarcomatous metaplasia, 13; epidermoid carcinoma and fibrosarcoma, 2; epidermoid carcinoma and rhabdomyosarcoma, 1; epidermoid carcinoma, 5; rhabdomyosarcoma, 6; angiofibrosarcoma, 1; fibrosarcoma, 1; fibroma with radiation effect, 2.

DR. HORN: Most of you recognized an epidermoid carcinoma; some thought it was associated with a pseudosarcomatous metaplasia, and others with a real sarcoma, variously called fibrosarcoma and rhabdomyosarcoma. A few thought it was primarily a sarcoma, probably with secondary epithelial, hyperplastic reaction — pseudoepitheliomatous hyperplasia, perhaps. Perhaps some did not get neoplastic or neoplastic-appearing epithelium in their sections. The problem revolves around whether we are dealing with two tumors, a carcinoma and a sarcoma; with carcinoma with a spindle-cell component; or with a pseudosarcomatous change in the stroma.

The illustration shows epidermoid carcinoma. It is fairly well differentiated, but shows the usual changes associated with malignant tumor, including pleomorphism and alteration in nuclear size and structure. Notice the way in which the groups of cells tend to spindle out. A little further away from the recognizable epidermoid tumor, groups of cells in twos or threes, forming spindled structures, can be seen. Two or three epidermoid cells form one long, spindle-shaped structure, which I think is several cells, but it could conceivably be a long, spindle cell with multiple nuclei. One section contained what could easily be interpreted as a strap cell, or a neoplastic muscle cell with a couple of nuclei. It resembled two elongated, epidermoid tumor cells.

The cells in another section were spindled and showed neither multinucleation nor multiple cells in a single group. The picture is complicated by a vascular hemorrhagic-appearing stroma which likely has some alteration in fibroblasts as well, perhaps resulting from the radiation. I have attempted to demonstrate transitions from what clearly looks like an epidermoid carcinoma into a spindle-celled epidermoid carcinoma. I think these spindle cells are epithelial tumor cells, and that this is essentially a spindle-celled epidermoid carcinoma.

I do not think it is a pseudosarcomatous change in the stroma or a biphasic, or collision, tumor. These have been found in the oral cavity and related cavities. They commonly are seen following radiation therapy; they seldom metastasize, but tend to kill by local growth. Foote, in the 1960 ASCP seminar, discussed a similar case, and concluded that it was all carcinoma with a pseudosarcomatous appearance, plus a hemorrhagic and vascular stroma.

DR. SEVERANCE: This patient died three or four months after the initial operation. After operation, external radiation therapy was used. At the time of death the patient had extension of disease into the neck. The physician thought the extensions were metastatic nodes, but there was infection and local recurrence, and no autopsy.

DR. DeCOURSEY: About 20 years ago, Maximow and Bloom, in their histology text, wrote that the epithelium and the muscularis of the tongue originate from the same embryologic layer. They commented that in this area, if there was a tumor of one side, the epithelial, there would probably be a tumor-like lesion of the other side, and that the tumor would usually be on the muscular side with a pseudoepitheliomatous type of situation. What is the present status of this viewpoint?

DR. HORN: I don't know. I think this is all an epidermoid tumor. Recognition of the

spindle-cell epidermoid carcinoma goes back many years, and it seems to have been forgotten until the recognition of this type of picture following radiation therapy. It was the older writers who pointed out that these tumors rarely metastasized but killed by local extension.

DR. DeCOURSEY: Granular cell myoblastoma of the tongue causes epitheliomatous hyperplasia, does it not?

DR. HORN: Granular cell myoblastoma apparently stimulates epithelial overgrowth any place where it occurs near a mucosal surface.

DR. DeCOURSEY: Elsewhere as well as in the tongue?

DR. HORN: Yes. I have seen it in the larynx and trachea.

1. Foote, F. W., Jr.: Case 22, 26th Seminar of American Society of Clinical Pathologists, 1960.
2. Lane, N.: Pseudosarcoma (Polypoid Sarcoma-Like Masses) Associated with Squamous-Cell Carcinoma of the Mouth, Fauces, and Larynx, Cancer 10:19 (Jan.-Feb.) 1957.
3. Sherwin, R. P., et al.: Polypoid and Junctional Squamous Cell Carcinoma of the Tongue and Larynx with Spindle Cell Carcinoma ("Pseudosarcoma"), Cancer 16:51 (Jan.) 1963.

Case 10/Tumor Seminar

Diagnosis.—Mesenchymal hamartoma of liver.

Contributors.—Drs. N. H. Jacob, G. C. Mani, and J. K. Lowry, Santa Rosa Hospital, San Antonio.

History.—A 10-month-old white boy's abdomen enlarged progressively for three weeks. At birth he weighed 8 lb., and delivery and the baby's subsequent development had been normal. A tumor mass involving the right lobe of the liver was resected. The specimen weighed 2,850 gm., and the tumor itself was 18 cm. in maximum dimension. It was multilocular, with the spaces containing clear, amber fluid. The adjacent part of recognizable hepatic parenchyma was light tan-yellow, greasy, and glistening.

DR. MANI: I should clarify that specimen means the liver and the tumor, and about three-quarters of the liver was replaced by tumor.

DR. HORN: Not many people have seen a lesion like this before, and this is why I thought it would be a good case for the seminar. Much of the tissue making up the tumor has structures that I suppose we have to regard as bile ducts, but they are vague and irregular; they are lined with epithelium. There is edematous, myxomatous, poorly cellular connective tissue described by some as primitive, or embryonal, around these ducts. In some sections the tissue is

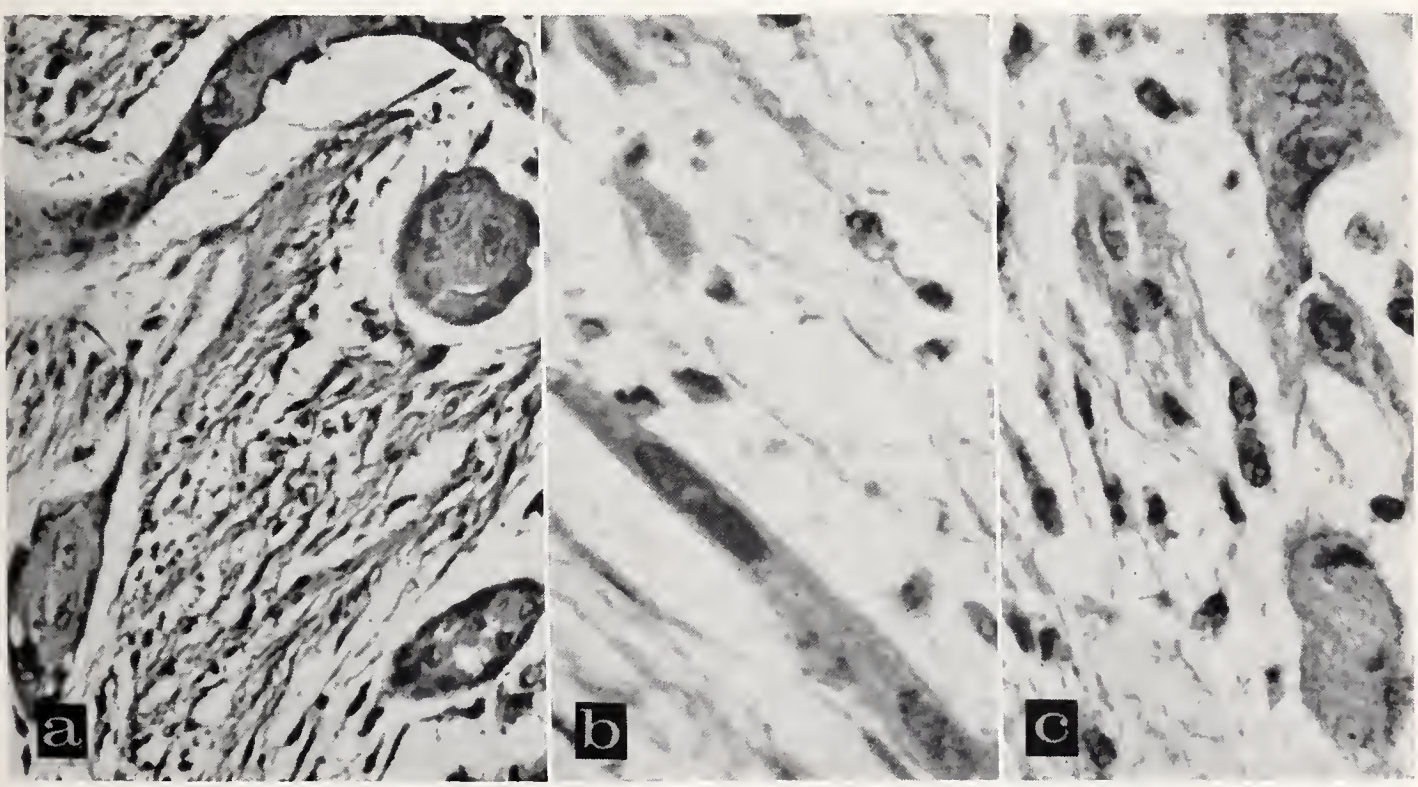


Fig. 10. Case 9. Spindle cell epidermoid carcinoma of floor of mouth. a. Note distinct epidermoid features. X 235. b. and c. X 366.6.

a little more cellular. Some of these structures look like liver cells, although they do not make very good cords. Making up a large part of this lesion, and being very characteristic of it, is tissue which gives the impression of spaces, but without any particular cellular lining—fluid containing, perhaps, collapsing after incision, or excision of the lesion, with delicate fibrous trabeculae apparently separating these spaces. These have been called lymphangiomatous, but I do not believe there has been any solid evidence to support this.

Submitted Diagnoses.—Mesenchymal hamartoma, 27; cystadenoma, 1; lymphangioma, 1; differentiated hepatoma, 1; fibrocystic disease, 1; cholangioma, 1.

DR. HORN: Most of the group called this a mesenchymal hamartoma, and as far as I am concerned this fits perfectly. I wonder if the people who called it other things had not heard of this lesion before, because it is uncommon. I had never heard of it until recently, when Dr. W. H. Long, Lubbock, sent me an example of it. This is referred to in Edmondson's fascicle on tumors of the liver and biliary tract. He collected 12 or 15 cases and called them mesenchymal hamartomas. Incidentally, Dr. Long's patient was also a 10-month-old white boy.

DR. MANI: Some cases have, in a sense, some amusing aspects, and unfortunately some tragic aspects. The surgeon who removed this tumor was not convinced of our diagnosis of hamartoma, and we sent specimens to the Armed Forces Institute of Pathology. They diagnosed hamartoma, referred us to Edmondson's fascicle, and asked us for some tissue because they, too, thought the case was quite unusual. Their note said that this patient should do well, but just the opposite occurred. The child died, probably as the result of extensive surgery and complicating pneumonia. No remaining tumor was found at autopsy.

DR. W. H. LONG, Lubbock: When I saw the slide from this case I thought there had been a mix-up, because the features were identical to those in the case I had sent to Dr. Horn. Incidentally, in our case, resection was carried out without difficulty and there was no evidence of spread beyond the liver. Unfortunately, about three hours after surgery, the child began to bleed. Apparently he had afibrinogenemia, and he died.

DR. HORN: Edmondson reported that in eight cases, the tumors were removed successfully, and in three cases, the patients died postoperatively. Postoperative death apparently is not unusual with these infants.

DR. MANI: It was quite a formidable task to remove this tumor, with its great bulk.

DR. HORN: This is described as 18 cm. in diameter, and almost 3,000 gm., and Dr. Long said his was 15 cm. They are big tumors.

1. Edmondson, H. A.: Atlas of Tumor Pathology, Sect. 7, Fasc. 25, Washington, D. C., Armed Forces Institute of Pathology, 1958.

Case 11/Tumor Seminar

Diagnosis.—Thymoma.

Contributors.—Drs. N. H. Jacob, G. C. Mani, and J. K. Lowry, Santa Rosa Hospital, San Antonio.

History.—A 73-year-old white man had prolonged massive bleeding from the tracheobronchial tree. Chest x-ray revealed a large lesion in the lower two-thirds of the left hemithorax, pushing the heart to the right. Ten years previously, the patient was told that he had a small tumor in the left lower lobe, but nothing was recommended and he had remained well. At thoracotomy an 1,800 gm., malignant-appearing, bosselated tumor, measuring 19 by 18 by 12 cm. and partially surrounded by a gray, thickened capsule, was found rising from the anterior mediastinum and pushing the lung posteriorly. The cut surface was gray to tan-purple, bulged slightly, and contained numerous small, cystic areas about 1 mm. in diameter.

DR. HORN: What made this tumor malignant appearing? Let's look at the diagnoses.

Submitted Diagnoses.—Bronchial carcinoma, 15; thymoma, 11; bronchial adenoma, 3; mesothelioma, 1; seminoma, 1.

DR. HORN: I wonder why so many people called this bronchial carcinoma since it is described distinctly as a mediastinal tumor. It conceivably could be a metastasis. I thought that this was an epithelial-appearing thymoma. Some of my associates called it a carcinoma of the thymus, but I doubt that it is malignant. Would the contributor care to comment on the gross ap-

pearance of the external surface of the specimen?

DR. MANI: I don't know why it was described as a malignant-appearing tumor, either. I suppose its large size gave reason for concern.

Fig. 11. Case 10. Mesenchymal hamartoma of liver. X 169.5.

DR. HORN: The cut surface is fleshy and grayish, with areas of hemorrhage. Many thymomas show the same fleshy, grayish, or grayish-yellow, waxy appearance. Striking lobulation is a characteristic of many thymomas, although it is not seen in the seminar case. Some areas of this tumor did show an extreme degree of degeneration and hyalinization, and the cavities were filled with assorted cellular debris and fibrin. In

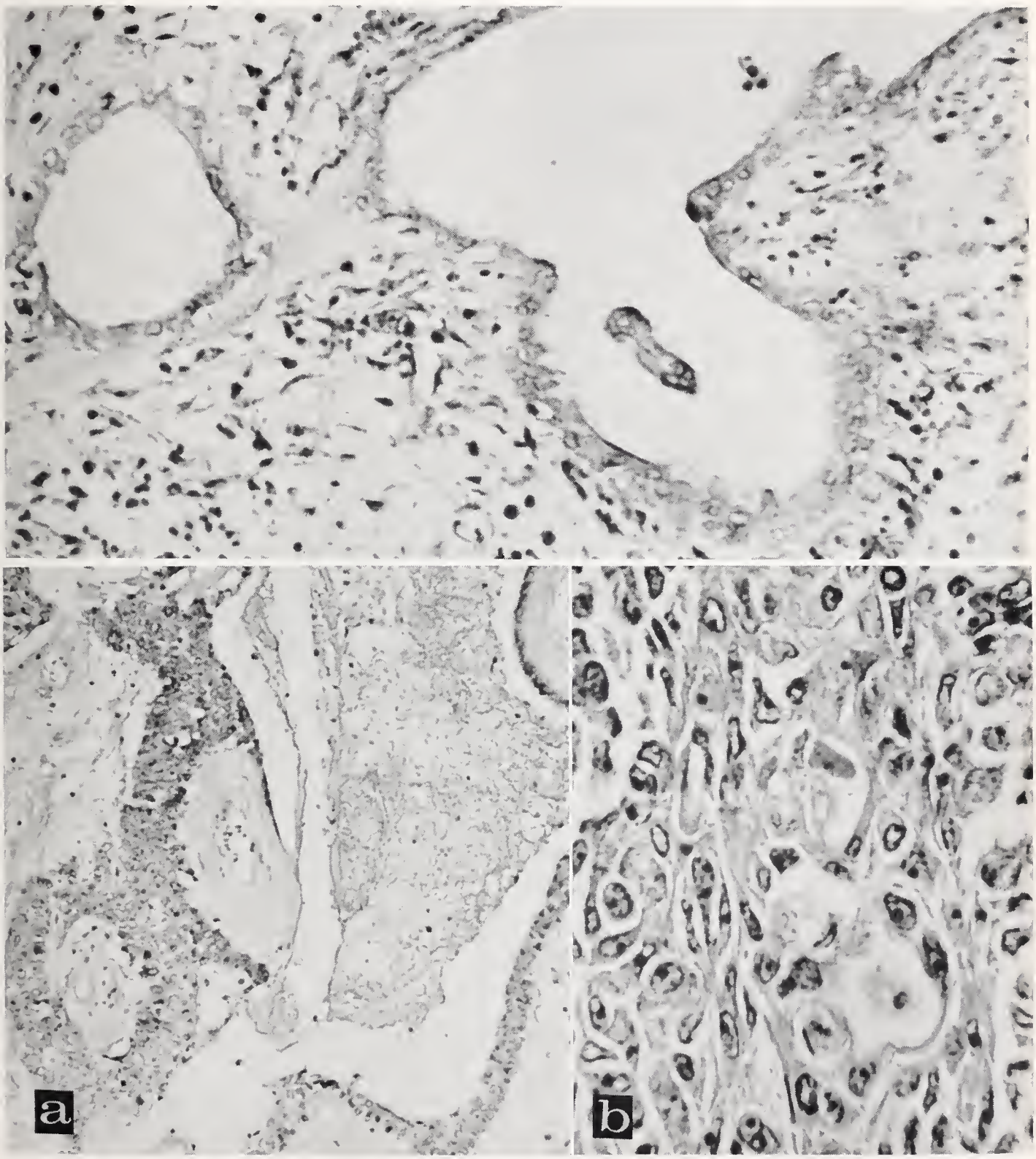


Fig. 12. Case 11. Thymoma. a. Degeneration, hyalinization and cyst formation. X 91.8. b. Cellular area. X 331.5.

more cellular, better preserved portions of the tumor are areas of considerable lymphoid infiltrate, and lobulation can be seen microscopically, although it is not readily apparent grossly. Higher magnification shows epithelial appearance, with many of the cells tending to spindle a bit. It is a pavement type of epithelium; the cells seem to fit together nicely, and have well-outlined, well-defined cytoplasm generally.

In places, there is simulation of epidermoid carcinoma, with bodies resembling epithelial pearls, or Hassall's corpuscles. These are the pavement-like, epidermoid cells, rounding up to simulate a Hassall's corpuscle, and central canals can be seen.

I recently examined the thymus from an infant who died of cystic fibrosis of the pancreas. The specimen was lymphocyte-depleted, or involuted, and the Hassall's corpuscles actually dilated to form real canals, or tubular structures. One of my recent residents studied involuted thymus glands, and deduced evidence that Hassell's corpuscles are really the primitive thymic canal and its branches surrounded by epithelioid cells, only secondarily being compressed and appearing as solid structures as the lymphocytes fill out the thymus. Then with the lymphocyte depletion of involution, they take on this appearance. Another slide from the case shows a canal, if you will, containing bits of keratin and surrounded by epidermoid cells.

I think this is a thymoma. It probably is benign, although a certain number of these are locally invasive and may recur locally; a very few may metastasize. With widely metastasizing epithelial thymic tumors, it may be difficult to assure oneself of thymic origin.

DR. MANI: The only therapy was surgical removal. The patient has gained 30 lb. and has no signs of tumor seven to eight months after surgery.

DR. RICHMOND: Why did he bleed from the tracheobronchial tree?

DR. MANI: I don't know.

1. Maldonado, J. E., et al.: The Thymus Gland and Its Relationship to the Hemopoietic and Immunologic Systems: Review, Mayo Clin. Proc. 39:60 (Jan.) 1964.
2. Shier, K. J.: Morphology of Epithelian Thymus; Observations on Lymphocyte-Depleted and Fetal Thymus, Lab. Invest. 12:316 (March) 1963.
3. Thomson, A. D., and Thackery, A. C.: Histology of Tumours of the Thymus, Brit. J. Cancer 11:348 (Sept.) 1957.

Case 12/Tumor Seminar

Diagnosis.—Intraductal carcinoma of breast (with limited infiltration).

Contributors.—Drs. N. H. Jacob, G. C. Mani, and J. K. Lowry, Santa Rosa Hospital, San Antonio.

History.—A 34-year-old white woman had a lump in her breast for six months, which changed with her menstrual periods. The specimen was 105 gm. of gray, resilient to firm breast tissue with multiple areas which were quite firm but not hard or gritty. The surface was covered with a slimy, clear mucoid material.

DR. HORN: First, to establish the diagnosis, one section contains areas of invasive ductal carcinoma, as well as an area of stromal invasion. This occupies a relatively small part of the sections that I had to study, and some of your sections may not show invasive growth. The bulk of the growth is intraductal, and it appears to be a papillary growth. The papillae tend to cross the lumen of the duct and hitch up with one another to give a gross, cribriform pattern. To go back a step, we usually think, when evaluating intraductal growths in the breast in terms of the cytologic appearances of the cells involved: Do they have the cytologic features that we have been taught to associate with malignant cells, or don't they? I find this an unreliable criterion on which to base a diagnosis of cancer on the one hand, or benign proliferation on the other. I go more by the pattern of the growth. There are a number of different patterns that intraductal cancers do take, but they all fit into one of several patterns. One is interlacing growth, papillary growth, with the papillae from various portions of the duct surface meeting somewhere in the middle and forming a more or less confluent papillary growth. This, to me, is intraductal carcinoma. One feature that I think is important is that there is little or no stroma in these papillae; they are almost entirely epithelial. Some areas are more purely papillary, and we do not see the anastomoses between the various papillae; but this is one of the patterns I would identify with malignancy.

Dr. Stout has called attention to snouts or nipples on the one hand, and bubbles, sometimes burst bubbles, on the other, in the proliferative lesions of the breast. The snouts or nipples—and they are difficult for me to see or to demonstrate—he identifies

with benign proliferative lesions, and the burst or unburst bubbles with evidences of malignancy. When you can see them, they may well be helpful.

A portion of the breast with papillary proliferation lining the duct initially looked benign; later I decided it was carcinoma, and now I don't really know. If I had to make up my mind on the basis of this appearance, I could not make a diagnosis of cancer, although I might be suspicious. However, since I believe there is cancer elsewhere, this is not important.

A good cribriform pattern shows a solid proliferation of cells, with lumina within the solid cell masses. This is a pattern of carcinoma.

Submitted Diagnoses. — Intraductal carcinoma, 26; papillomatosis, 2; fibroadenomatosis, 1; fibrocystic disease, 1; adenomatous hyperplasia, 1.

DR. HORN: I thought this was breast carcinoma, primarily intraductal and papillary, but with some invasive growth, and the majority of you have agreed, although a few have considered it benign.

DR. MANI: Sections from tissue removed at radical mastectomy showed axillary lymph node involvement with adenocarcinoma in about 2 of 20 lymph nodes. After nine months, the patient has no further evidence of tumor. She has had a nephrectomy and x-ray treatments.

DR. GALINDO: Dr. Horn, how would you handle frozen sections of tissue such as we have seen in these slides?

DR. HORN: I doubt that I could recognize it as carcinoma. If I were suspicious, I would tell them to wait; if I weren't, I would have to reverse my diagnosis when the paraffin sections became available. I hate to do that, but it sometimes is necessary.

DR. MANI: This was troublesome on frozen section, and we waited.

DR. HORN: Many intraductal carcinomas are impossible to diagnose positively on frozen section, and I would rather wait two days than run any risk of doing a radical mastectomy for a benign lesion.

DR. BRANAM: Someone once commented on the possibility of waiting one or two weeks following biopsy, without affecting the prognosis.

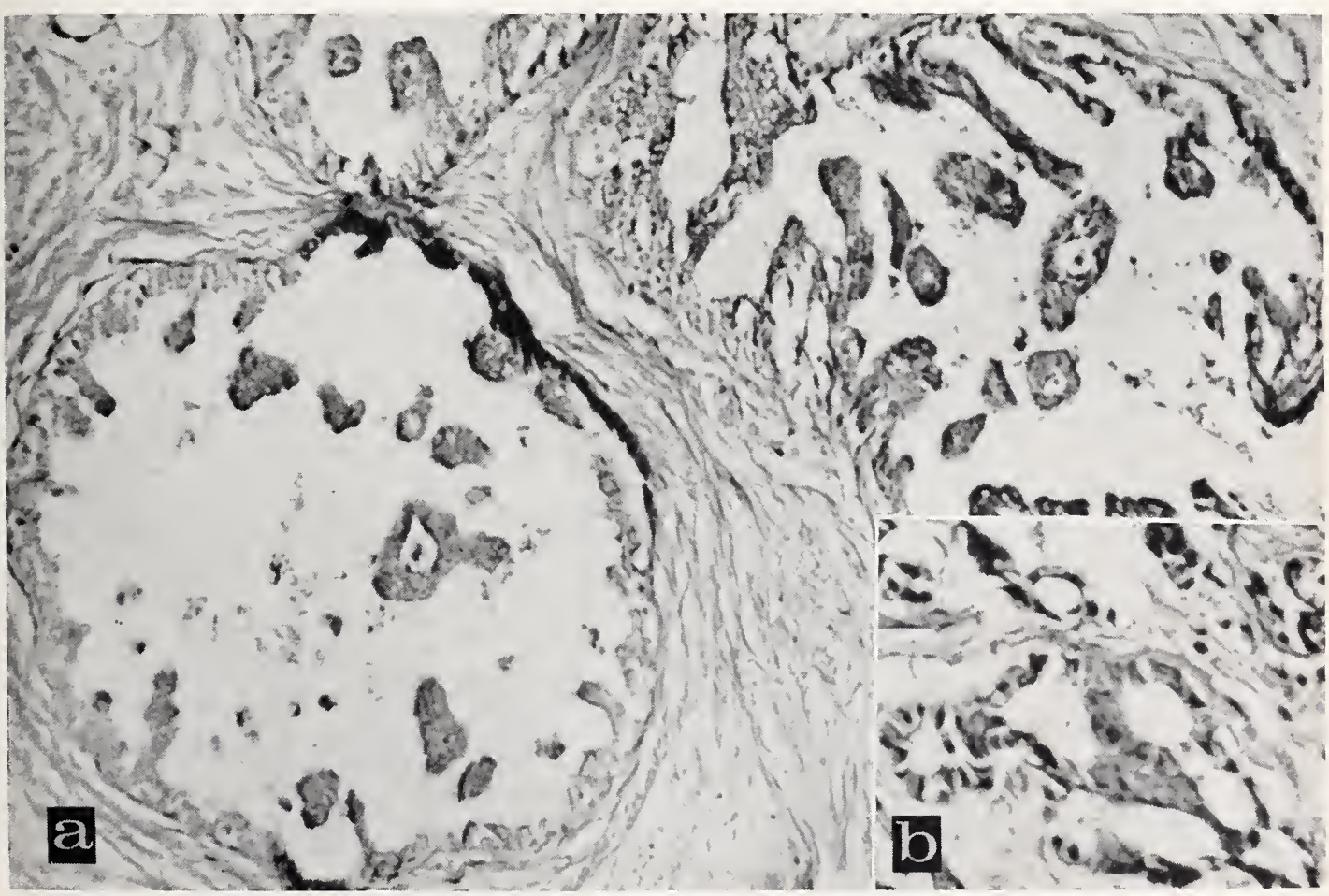


Fig. 13. Case 12. Intraductal carcinoma of breast, with limited infiltration. a. Intraductal character. X 118.5. b. Area of infiltration. X 154.5.

DR. HORN: I think Haagenson has evidence that this short wait does not jeopardize the patient's chance. His is not a large series, and I would doubt its statistical validity. On the other hand, Ackerman reported a series in which the wait was considerably longer, a matter of months, between simple or partial mastectomy and radical mastectomy, and in that group of patients Ackerman thought it did jeopardize the patient's chances.

DR. R. E. BRIERTY, Brooke General Hospital: Regardless of pleomorphism, if you see a solid pattern without any luminary holes in the duct at all, do you revert to the shape of the cells, rather than their size and their pattern?

DR. HORN: I suppose I do, but I am confusing myself and maybe you, too, because I think of this as pattern. There are benign lesions in which the ducts are solidly filled with cells, and there are certainly malignant ones, and the cells are distinctly different in the two.

DR. LUNDBERG: Do you consider intraductal hyperplasia a premalignant lesion?

DR. HORN: The pathologist seldom suggests simple mastectomy for a benign lesion except as a salve to his own conscience. There is convincing evidence that benign proliferative lesions occur more often in cancerous than in noncancerous breasts. But I cannot pick out the individual, or group of individuals, who have a better chance of developing cancer. When we see a benign lesion and say, "This is bad; we'd better do a simple mastectomy," we are doing that for us, and not for the patient.

DR. GALINDO: At Robert B. Green Hospital we have a paucity of carcinomas of the breast; many of our patients are indigent and they have quite a few babies. Have you found any relationship between the large number of children and breast feeding, and the low incidence of carcinoma, in your area?

DR. HORN: Not in our area, but several reports indicate that in the breast which functions normally, cancer is less likely to develop than in a breast which does not function normally, for whatever reason.

1. Stout, A. P.: Observations on Biopsy Diagnosis of Tumors, Cancer 10:912 (Sept.-Oct.) 1957.

Case 13/Tumor Seminar

Diagnosis.—Papillary diffuse mesothelioma of pleura.

Contributors.—Drs. N. H. Jacob, G. C. Mani, and J. K. Lowry, Santa Rosa Hospital, San Antonio.

History.—A 48-year-old white woman had three episodes of pleural effusion after October, 1962. Cytologic studies were negative and acid-fast bacilli did not grow. In October, 1963, hysterectomy and bilateral salpingo-oophorectomy, done because of slight fullness in the adnexa, showed endometriosis of the ovaries, and fibroids. Abdominal exploration showed no other abnormalities. Thoracotomy in March, 1964, revealed a heavy, thick, meaty-looking tissue approximately 4 cm. in diameter bridging the intracostal space, attached to the diaphragm and to the anterior thoracic pleura. Nothing was palpated in the lung; there was no clinical evidence of other tumor.

DR. HORN: We can see a focus of tumor set in a background of nondescript, fibrous tissue stroma, and the tumor itself is composed of rather solid cords of cells centrally, but peripherally having a papillary configuration. This is true of each of the many tumor cell deposits. Higher magnification shows nothing different; these are fairly uniform cells, and they do not look highly anaplastic.

Submitted Diagnoses. — Papillary mesothelioma, 16; metastatic adenocarcinoma, 5; papillary carcinoma, 1; lobular carcinoma, 1; dysgerminoma, 1; carcinoid, 1; endometriosis, 1.

DR. HORN: Considerably more than half of you called this papillary mesothelioma, and the next most prominent diagnosis is carcinoma. I think this is a papillary mesothelioma, and I admit that we must rule out metastatic adenocarcinoma. That is always the situation before we make a diagnosis of mesothelioma, other than those of the fibrous type, which do not simulate carcinoma at all.

A variant of the mesothelioma pattern is the tubular one, and some people lump tubular and papillary mesotheliomas together. They do occur together quite commonly. The papillary and tubular mesotheliomas are more common in the peritoneum than in the pleura.

I want to show you a slide of a papillary mesothelioma of the pleura. It is probably

benign, although the section is from autopsy and we cannot observe the clinical behavior of the lesion. It is heavily fibrous and has a good stroma, and in that respect is unlike the seminar case. Also, the cells are uniform and well differentiated. Stout and Ackerman have, on occasion, illustrated seminars and articles with mesotheliomas from cows. This one came from a lion.

In the seminar case, I had difficulty interpreting, from the gross description, whether this is a diffuse or localized mesothelioma; it is probably diffuse. Most diffuse mesotheliomas behave in malignant fashion by spread over the serosal surface rather than by metastasis. In the peritoneum, they tend to produce intestinal obstruction. Their course in the pleura is uncertain. I think it will be difficult or impossible to cure, but I would not anticipate metastasis.

DR. MANI: Dr. Lowry, do we know whether this was localized or diffuse?

DR. LOWRY: It was localized in a diffuse manner; there was definitely tumor left behind, but it was not a covering of the entire pleural cavity.

DR. HORN: We should consider the possibility of metastasis. This woman did have pelvic disorders, and one would think of papillary ovarian carcinoma. I decided to be trusting and believe those who said the major abnormalities in the pelvis were endometriosis and fibroids.

DR. MANI: The patient received cobalt irradiation therapy after the biopsy. After nine months, she shows no signs of tumor anywhere in the body, and has had no more effusion. Her most recent chest x-ray showed thickening of the pleura, which we presume is at least partly the result of irradiation.

DR. HORN: Have cytotoxins been instilled into the pleural cavity?

DR. MANI: No.

DR. HORN: I believe there has been a recent report of good symptomatic results with local instillation of some cytotoxins in serous cavities.

DR. LOWBEER: We had a case which illustrates the difficulties that occur even if we have an autopsy. This person had diffuse, extreme thickening of one pleura, which looked exactly like a pleural mesothelioma of this type. There was some extension into the lungs from the outside in the form of lymphatic streaks, and at the hilus there was a similar extension, so that the bronchus seemed to be involved from the outside. Microscopically this was a tubular tumor with extensive mucin production, and in addition there was an enormous number of psammoma bodies. Although psammoma bodies would go with a pleural mesothelioma,

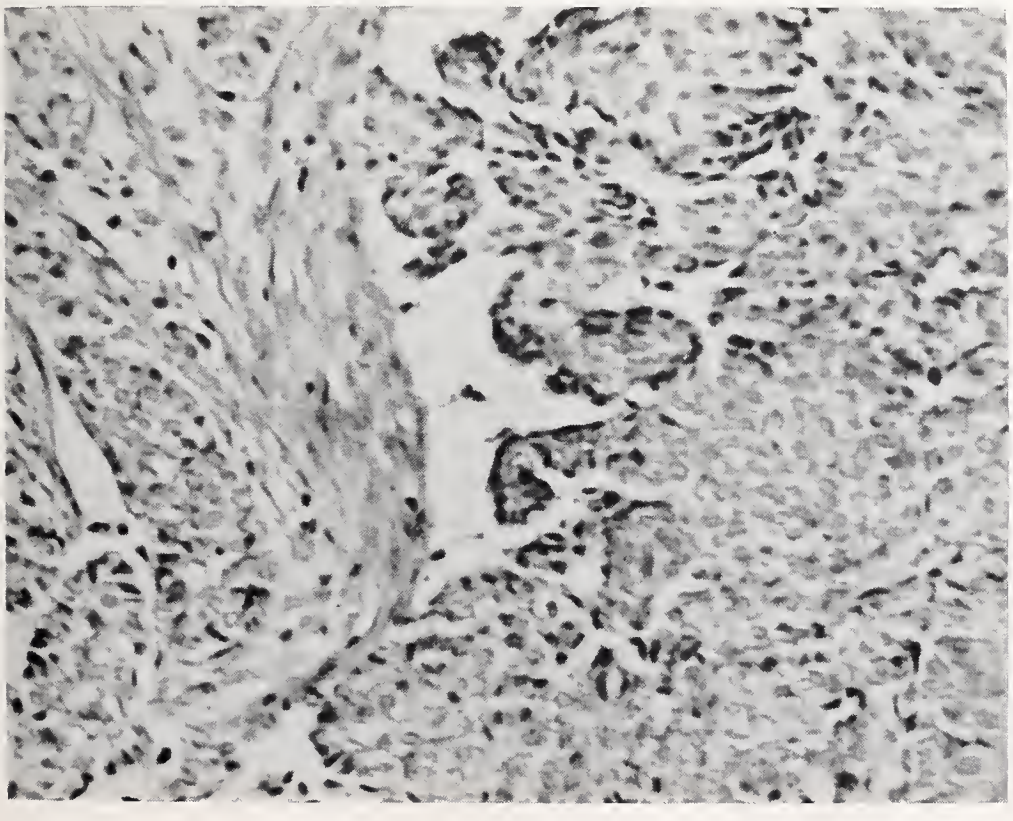


Fig. 14. Case 13. Papillary diffuse mesothelioma of pleura. X 162.

mucin production to this extent would not. There was a connection with the bronchus at the hilus, and particularly with the salivary gland. We could not bring ourselves to consider this a pleural mesothelioma, although grossly it looked like one. Papillary carcinomas originating from mucinous glands of the bronchus produce mucin, but they may also have psammoma bodies, as do other papillary tumors. I would like to call it a papillary mesothelioma, but since there was such a relation, primary or secondary to the peribronchial glands, and so much mucin production, I did not dare.

DR. HORN: I agree; to make a firm diagnosis of any papillary or tubular mesothelioma without complete autopsy examination leaves the possibility that autopsy may reveal our mistakes. I have seen papillary bronchial carcinomas with numerous psammoma bodies, but I do not recall that they also secreted excessive mucin. Mesotheliomas can produce mucin, but it is said that the demonstration of hyaluronic acid would rule out carcinoma, and definitely establish mesothelioma.

1. Godwin, M. C.: Diffuse Mesotheliomas; With Comment on their Relationship to Localized Fibrous Mesotheliomas, Cancer 10:298 (March-April) 1957.
2. Winslow, D. J., and Taylor, H. B.: Malignant Peritoneal Mesotheliomas; Clinicopathological Analysis of 12 Fatal Cases, Cancer 13:127 (Jan.-Feb.) 1960.

Case 14/Tumor Seminar

Diagnosis. — Mesonephric carcinoma of vagina.

Contributors.—Drs. N. H. Jacob, G. C. Mani, and J. K. Lowry, Santa Rosa Hospital, San Antonio.

History.—A 65-year-old white woman had a pink, mucoid vaginal discharge for two weeks. Total abdominal hysterectomy and subtotal vaginectomy with resection of subjacent rectum was performed. On the superior third of the posterior vaginal wall was a well-defined, faintly papillary, soft, light tan-gray fungating lesion measuring 3 by 2.5 cm. Thirty years before, her right ovary and part of the left had been removed because of nonmalignant tumor.

DR. HORN: The tumor has several different patterns. First is a tubular one in which the tumor cell cytoplasm is clear or pale, and vaguely eosinophilic material is in the lumina of many of the tubes. Higher magnification shows the tubular structures with eosinophilic concretions, and solid portions of the tumor. There is a tendency toward papillary structure. All these things indicate mesonephric carcinoma.

Submitted Diagnoses. — Mesonephric carcinoma, 11; metastatic adenocarcinoma, 11; Gartner's duct adenocarcinoma, 1; alveolar soft part sarcoma, 1; lymphangioma, 1; adenosis of vagina, 2.

DR. HORN: Gartner's duct carcinoma means the same thing, and some have recognized carcinoma but presumed it metastasized from somewhere else. Perhaps they are thinking of the ovarian operation some 30 years before, but I prefer to ignore it. Adenosis or adenoma of the vagina is presumably a comparable lesion, although benign, whereas I think this one is malignant.

Primary carcinomas of the vagina are rare, and most of them are epidermoid carcinomas. Probably all of the rare adenocarcinomas are of mesonephric origin. Mesonephric tumors have been described in the vagina, uterus, parametrium, and ovary, and, according to Teilum, comparable tumors have occurred in the epididymis and testis.

In a similar case, a 14-year-old girl had an ulcerated lesion high in the cervix; its pattern was essentially identical with the seminar tumor, having papillary configuration and the clear cells. Another tumor from the cervix of a seven-year-old girl, had clear cells; it was a polypoid lesion, and they can mimic the clinical appearance of botryoid sarcoma.

Novak called three of his eight vaginal mesonephric tumors benign, and one of them was polypoid. Two of the five malignant tumors were polypoid. One of his patients died with metastatic disease, but he offers no follow-up on the others. In Teilum's series, the patient with vaginal disease died, so apparently these are genuinely malignant tumors, unless we are dealing with simple adenomas or "adenosis of the vagina."

DR. MANI: This patient died five days after surgery. The clinical pattern was that of a pulmonary embolus, but no autopsy was obtained.

DR. F. P. BORNSTEIN, El Paso: We had a similar case; about ten years ago the patient had a vaginal cyst removed which was thought to be a rest cyst. A large tumor developed in this area, which had the pattern we have seen, and radical surgery was performed. Invasion was not in the lymph

nodes, but was the rather far-reaching parametrial type.

DR. HORN: Do you think she probably had a Gartner's duct cyst six or eight years ago?

DR. BORNSTEIN: Yes.

DR. HORN: There was a bizarre tumor of the vagina presented at the 1952 ASCP Seminar (Case 2). I believe it is similar.

1. Neubecker, R. D., and Breen, J. L.: Embryonal Carcinoma of Ovary, Cancer 15:546 (May-June) 1962.
2. Novak, E., et al.: Probable Mesonephric Origin of Certain Female Genital Tumors, Amer. J. Obst. Gynec. 68:1222 (Nov.) 1954.
3. Teilum, G.: Histogenesis and Classification of Mesonephric Tumors of the Female and Male Genital System and Relationship to Benign So-Called Adenomatoid Tumors (Mesotheliomas), Acta Path. Microbiol. Scand. 34:431, 1954.

Case 15/Tumor Seminar

Diagnosis.—Glomus tumor of nasal cavity and ethmoid sinus.

Contributors.—Col. H. B. Hoeffler, M.C., and Capt. R. E. Jones, Jr., M.C., Brooke General Hospital, Fort Sam Houston.

History.—A 53-year-old woman had repeated bouts of epistaxis for three years, and recent severe bleeding. Biopsy was made of a large tumor filling the right posterior portion of the nose. X-rays showed that the right ethmoid and right maxillary sinuses were opacified with questionable destruction of part of the maxillary sinus wall. At operation the tumor was blue-gray and vascular, broke through the lateral wall of the nose, and filled half the right nasal cavity. It was encapsulated and easily dissected.

DR. HORN: When I selected this case for a seminar the choice seemed perfectly logical, but later I began wondering why I picked it. This tumor had such a nice pattern that it just had to be something, and I am still not really certain what it is.

A few shreds of the respiratory mucous membrane are left. I thought I had gotten more of it in the picture. Beneath this is a cellular tumor, or a tumor which is remarkable for both its cellularity and vascularity. In one section the vessels are moderately compressed, although they are certainly numerous and prominent. They form a web or a network within the interstices of which the cells are grouped.

This woman's chief problem was epistaxis, and the reason for it is quite prominent. These nests or groups of cells are generally separated from the endothelium by a little bit of edematous, or hyaline fibrous tissue. At higher magnification, you can see that the cells are generally small, uniform, and orderly. There is not a lot of pleomorphism; there is not much hyperchromatism; there is little mitotic activity or cytoplasm to be seen. The nucleus is perfectly visible, and in the few cells where cytoplasm is seen, it is not sharply outlined.

Submitted Diagnoses. — Nonchromaffin paraganglioma, 12; glomus tumor, 8; hem-

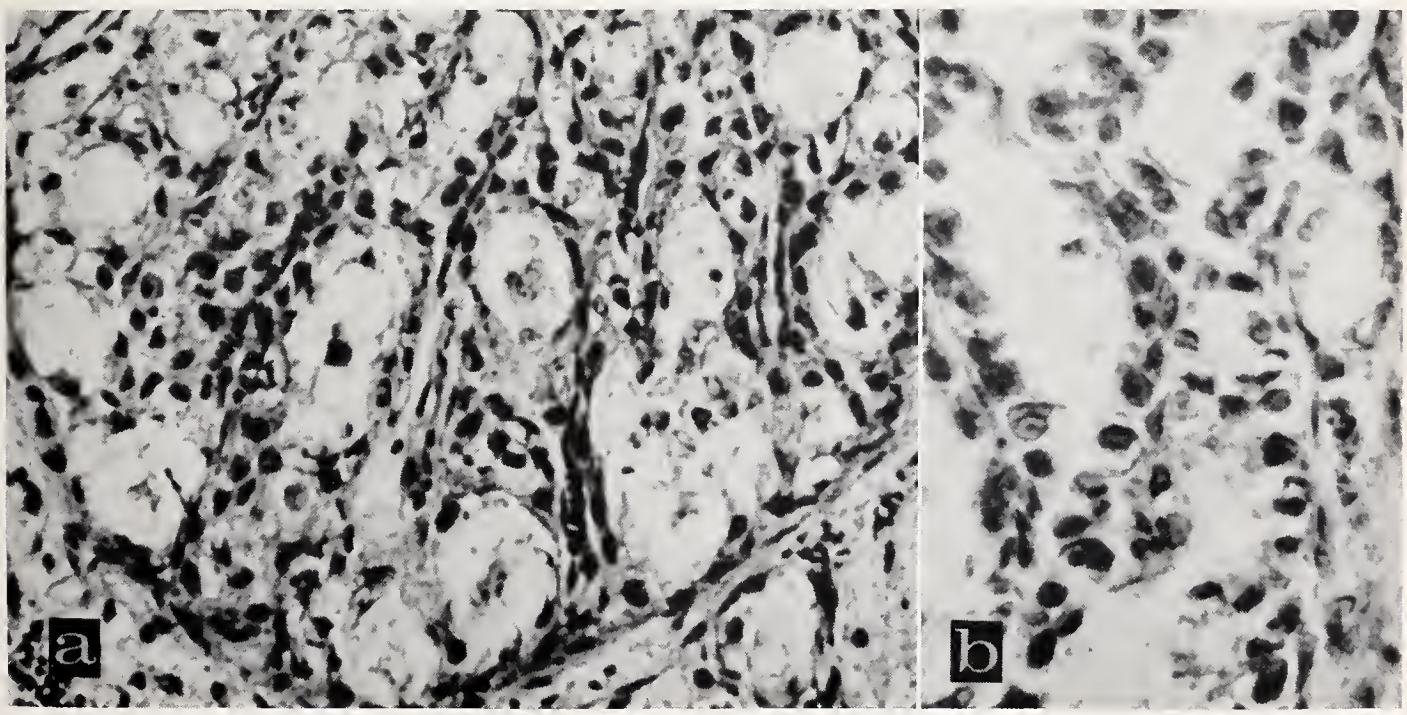


Fig. 15. Case 14. Mesonephric carcinoma of vagina. a. X 156. b. X 260.

angiopericytoma, 5; chemodectoma, 4; ameloblastoma, 1; undifferentiated carcinoma, 1; transitional cell carcinoma (Schneiderian type), 1.

DR. HORN: Twelve people have called it a nonchromaffin paraganglioma and four a chemodectoma, which to me means essentially the same thing. Eight have called it a glomus tumor, and another five, hemangiopericytoma. The diagnoses made by two eminent consultants shook me a bit, because they were not much in line with my thoughts. I cannot accept this as a carcinoma, which is the diagnosis of one consultant. The cells are too small and too innocent-looking, and too regular to be those of an undifferentiated or transitional cell carcinoma of the nasal or related cavity. Also, I cannot accept this as an esthesioneuroblastoma or neuroepithelioma. It has the wrong pattern, and is arising in a wrong place. It does not extend from the olfactory mucous membrane, but arises in the ethmoid sinus and breaks through into the nasal cavity. A glomus tumor is the only logical diagnosis that I can make. I would not be averse to calling it a hemangiopericytoma, which does not show the whole organoid development of a glomus tumor. A glomus tumor, to me, is a highly organized subdivision of the hemangiopericytoma. There seems to be a considerable confusion between the paragangliomas and the chemodectomas, and the glomus tumor. "Glomus jugulare" is labeled "glomus," and "nonchromaffin paraganglioma of the glomus jugulare" is translated into "glomus tumor." "Glomus tumor" is used as a synonym for "chemodectoma," "carotid body tumor," or what have you; whereas I believe these things are completely distinct: the glomus tumor or hemangiopericytoma being a vascular tumor with proliferation of a specific cell, the pericyte if you will, as opposed to the nonchromaffin paraganglioma, which is a neoplastic proliferation of cells of sympathetic or parasympathetic nerve origin and make up the paraganglia.

I believe this seminar case is a glomus tumor in a region where, as far as I know, none have been reported. One variety of glomus tumor is relatively poor in vessels. One glomus tumor of the stomach, reported by Kay and his collaborators, is fairly solid, and although there are vessels they have been, for the most part, squeezed shut. Where the vessels are open, the cells seem to be grouped at a distance from the endothelium or the cell lining. Another such tumor has a pattern which is perhaps more conventional; the vessels are much more prominent, having the character of sinusoids. One feature that is not seen in the seminar case is a sharp cell border.

In contradistinction, a photomicrograph of a glomus tumor of the skin or subcutaneous tissue shows pericytes which are not immediately beneath the endothelium, but are characteristically separated from it by connective tissue, which is either hyalinized or edamatous, or both. I can see some resemblance to the tumor in the seminar case, which I believe is a real glomus tumor. They have been reported in such a variety of places, including bones, uterine musculature, and joint capsule, that I do not find it difficult to recognize one almost anywhere else.

CAPTAIN JONES: In fresh tissue and in paraffin sections we were unable to stain any granules of any type in the cells, which supports that diagnosis. The biopsy specimen was identified as glomus tumor, but after we saw the whole tumor we were not sure what it was. We have a follow-up of one year, and thought there was a recurrence three months ago. Exploratory surgery revealed a nasal polyp, but no residual tumor.

DR. HORN: After you made a diagnosis of glomus tumor from the biopsy did you change your mind, or did a consultant talk you out of it?

CAPTAIN JONES: We had several consultants of your high caliber, and they talked us out of it. I notice only one of the diagnoses they gave, however, was among those made by this group, which is interesting.

DR. MANI: Colonel Hoeffler, you are going to have to defend your consultants.

COLONEL HOEFFLER: We did think this was a good glomus tumor, and I think Dr. Severance concurred; tissue from the second operation, however, seemed as though it had lost its vascular character, and some areas looked a little more invasive. With diligent searching we found several mitoses, and I called it a low-grade, undifferentiated carcinoma. We sent this to several pathologists and one called it low-grade, undifferentiated

carcinoma, and another thought it was an esthesioneuroblastoma. There is no unanimity of opinion. I could not disagree vigorously with Dr. Horn's opinion. His discussion supports his logical diagnosis, the majority of people here concur. The clinical course is in keeping with a benign lesion.

DR. SEVERANCE: I had trouble with this diagnosis; but I leaned toward benign nonchromaffin paraganglioma. When I looked at the slide again recently I even wondered whether it could be a pituitary chromophobe adenoma coming down from the nose, but then I said, "They wouldn't do that to me. There is nothing said about the vision or anything," so I went back to nonchromaffin paraganglioma.

1. Kay, S., et al.: Glomus Tumors of the Stomach, Cancer 4:726 (July) 1951.
2. Mendeloff, J.: The Olfactory Neuroepithelial Tumors; Review of Literature and Report of Six Additional Cases, Cancer 10:944 (Sept.-Oct.) 1957.

Case 16/Tumor Seminar

Diagnosis.—Malignant lymphoma (Hodgkin's disease) of testes (bilateral).

Contributors. — Drs. H. J. Schattenberg and E. T. Standley, San Antonio.

History.—A 72-year-old man had uniform, painless bilateral enlargement of the testes, beginning about one year before examination. Both testicles were removed and were ovoid, averaging 6.5 by 10.5 cm.

Submitted Diagnoses.—Lymphoma (reticulum cell, 12; Hodgkin's, 1), 13; seminoma, 6; embryonal carcinoma, 5; interstitial cell tumor, 4; androblastoma, 1; angiosarcoma, 1; undifferentiated carcinoma, 1.

DR. HORN: I thought that this was malignant lymphoma. I called it Hodgkin's disease, but I would not quibble with anybody who preferred to call it reticulum cell sarcoma. The recognition of Reed-Sternberg cells is subjective, and my own subjectivity changes from day to day. I hope it doesn't change from hour to hour.

There are essentially two appearances of the tumor. In one, the structure tends to be fairly loose, the cells are widely dispersed in a loose, not very heavy, fibrous stroma. Other portions of the tumor are densely cellular, and the cells seem distinctly larger. Many of the nuclei are bean-shaped, or kidney-shaped. Nucleoli are frequently prominent. There is a lot of mitotic activity.

Usually in the denser portions of the tumor one can detect remnants of seminiferous tubules, and some cells within them resemble the cells outside, and might tempt one to think in terms of a seminoma or a

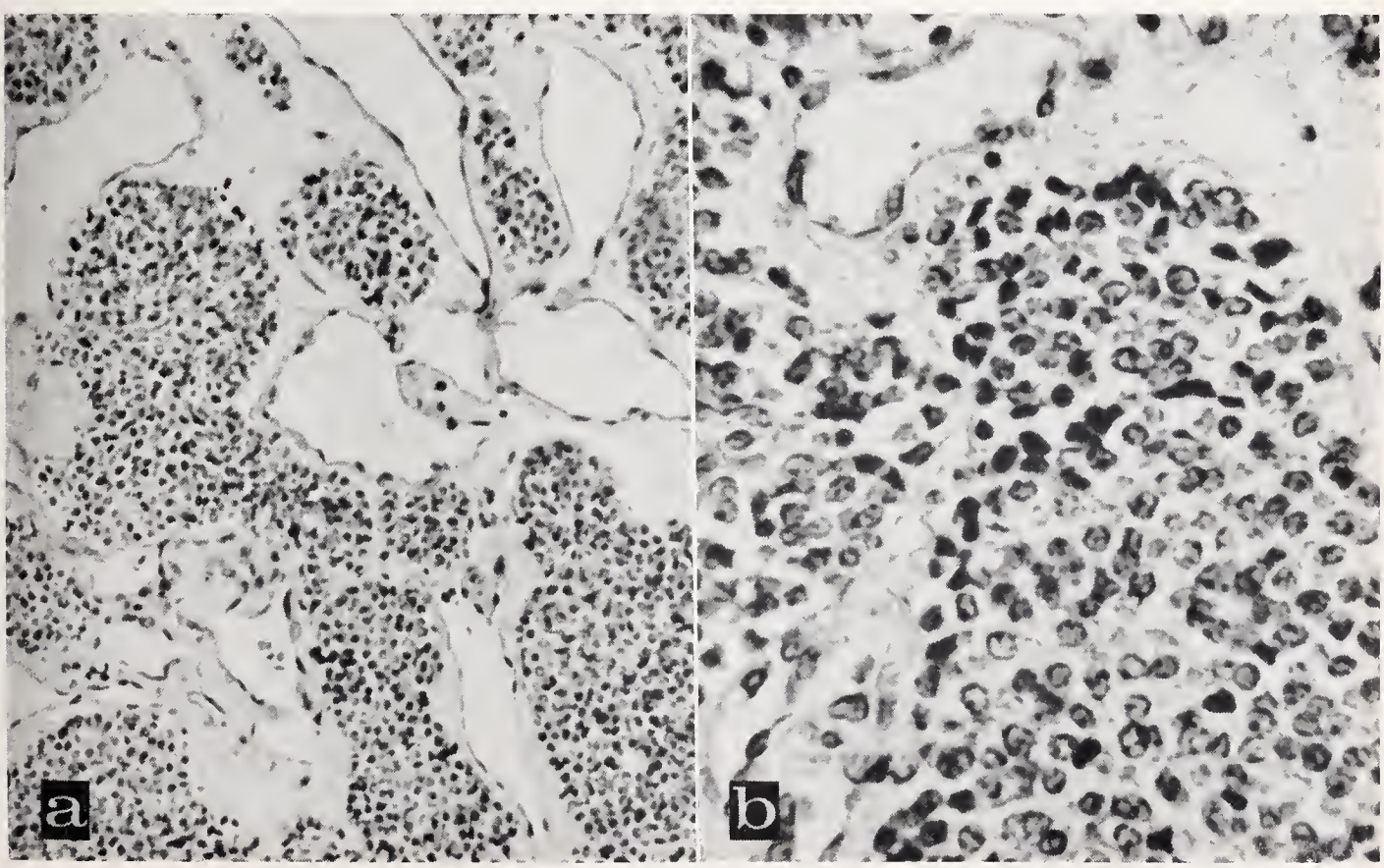


Fig. 16. Case 15. Glomus tumor of nasal cavity and ethmoid sinus. a. X 119.6. b. X 337.8.

germ-cell tumor. I do not believe, however, that all of these are tumor cells. Some may be, but on the whole I think the appearances are not those of a seminoma or other variety of germ-cell tumor. The cells themselves are not quite correct, nor do we have the organization of the usual seminoma, the cell grouping, nor the lymphocytic infiltrate which does not always occur in all of the seminomas. Its absence does not rule out that diagnosis, but I think this is a more diffuse lesion than the usual seminoma, which is one of my reasons for considering it to be a lymphoma.

Photomicrographs at higher power give the impression that many of the cells are large, and that the nucleoli are often prominent, many of them multinucleated. I suppose I parlayed this impression into agreeing that I see Reed-Sternberg cells and making a diagnosis of Hodgkin's disease, but you may not agree. I found it difficult to pick out one convincing Reed-Sternberg cell, and then get a photomicrograph of it, so if anyone wants to hedge the diagnosis of Hodgkin's disease and stick to reticulum cell sarcoma, I have no quarrel.

It is difficult to find out a great deal about testicular lymphomas. We have many cases in our files of involvement of the testes by malignant lymphoma, but most are instances of disseminated malignant lymphoma, and the testicles are just one site of involvement. In one case we thought we were dealing with primary testicular lymphoma; there was replacement of the entire organ by yellowish, fleshy tumor tissue, and there were nodules along the spermatic cord. Inguinal node involvement developed shortly after the orchiectomy. The patient died within ten months, and at the time of his death he had widespread abdominal and pelvic involvement, some in lymph nodes and some involving soft tissues or viscera. With this relatively short interval, it seems reasonable to believe that this man had malignant lymphoma elsewhere than in the testes at the time of the orchiectomy, especially since there were nodules along the vas.

In another case of testicular lymphoma, three of us initially diagnosed granulomatous inflammatory process. Others considered it a lymphoma. One consultant actually made a diagnosis of Sertoli cell tumor. I don't know whether he thought it was benign or malignant. Later, the disease appeared in cervical lymph nodes, and probable Reed-Sternberg cells were seen. This man died within two years. We did not have an autopsy, and our knowledge of the extent of his disease is somewhat sketchy. Possibly he had disseminated lymphoma, or at least lymphoma extending beyond the testis, at the time of the initial diagnosis. Malignant lymphomas of the testis frequently are part of generalized lymphoma, or at least part of lymphomatous disease with involvement of other organs than the testis, although they may present as primary lesions of the testis. Not uncommonly they are bilateral, and in bilateral testicular tumors, lymphoma rates fairly high; even so, lymphoma presenting first in the testis is rare. On the contrary side, a colleague had a testicular enlargement which was diagnosed as lymphoma. He brought the slides to me, and I thought he had a granulomatous inflammation. This time I turned out to be right.

Mistakes can be made both ways; but I think our patient has Hodgkin's disease or reticulum cell sarcoma, and I would be interested in hearing the follow-up.

DR. STANDLEY: After surgery, the patient had enlargement of the inguinal lymph glands. He was referred to the M. D. Anderson Hospital, but declined to have a biopsy of the lymph glands. He died three and one-half months later; there was no autopsy.

DR. HORN: This suggests that his lesion was not necessarily primary in the testes, even though it presented there first.

DR. LOWBEER: We had an extraordinary experience with a tumor of this kind, which illustrates some of the points Dr. Horn has made. A middle-aged man complained about urinary difficulties. An obstruction was found at the right ureteral orifice due to a large mass. Biopsy showed an undifferentiated neoplasm which was sent to many consultants. I though it was essentially a granuloma, and others thought it was a renal cell carcinoma. He was treated with irradiation, and the tumor disappeared. At one time, his right testicle had been injured, and had been tender ever since, but nobody paid the slightest attention to it. Four years later, a large tumor mass developed suddenly in that same testicle. The testicle was removed and showed a reticulum cell sarcoma. It was identical with the retroperitoneal mass biopsied four years earlier. The assumption is that he had the tumor in

the right testicle all the time, only it was quite small, and a metastasis to the retroperitoneal lymph node was the first clinical manifestation.

DR. GUILLEBEAU: We had a similar case to this at Lackland Air Force Base Hospital. We made the diagnosis of reticulum cell sarcoma. Exploratory surgery of the abdomen showed no lymph nodes; the spleen and liver were normal. Since there was no other evidence of tumor, we did not use radiotherapy. As far is I know he still has no further evidence of disease.

DR. HORN: Yours is as well documented a case as you can have of a real primary.

DR. MANI: We had a child born with bilateral testicular lymphosarcoma who died at about one year of age of disseminated lymphosarcoma proved at autopsy. I thought it was a curious variant.

DR. DeCOURSEY: What has become of the idea that to be sure that it is a Reed-Sternberg or Hodgkin's cell, the nucleolus be stained red?

DR. HORN: I do not have any such positive, definite criteria. To me it either looks like a Reed-Sternberg cell or it doesn't.

1. Hotchkiss, R. S., and Laury, R. B.: Concomitant Bilateral Malignant Testicular Tumors, J. Urol. 63:1086 (June) 1950.
2. Melicow, M. M.: Classification of Tumors of Testis: A Clinical and Pathological Study Based on 105 Primary and 13 Secondary Cases in Adults, and 3 Primary and 4 Secondary Cases in Children, J. Urol. 73:547 (March) 1955.

Case 17/Tumor Seminar

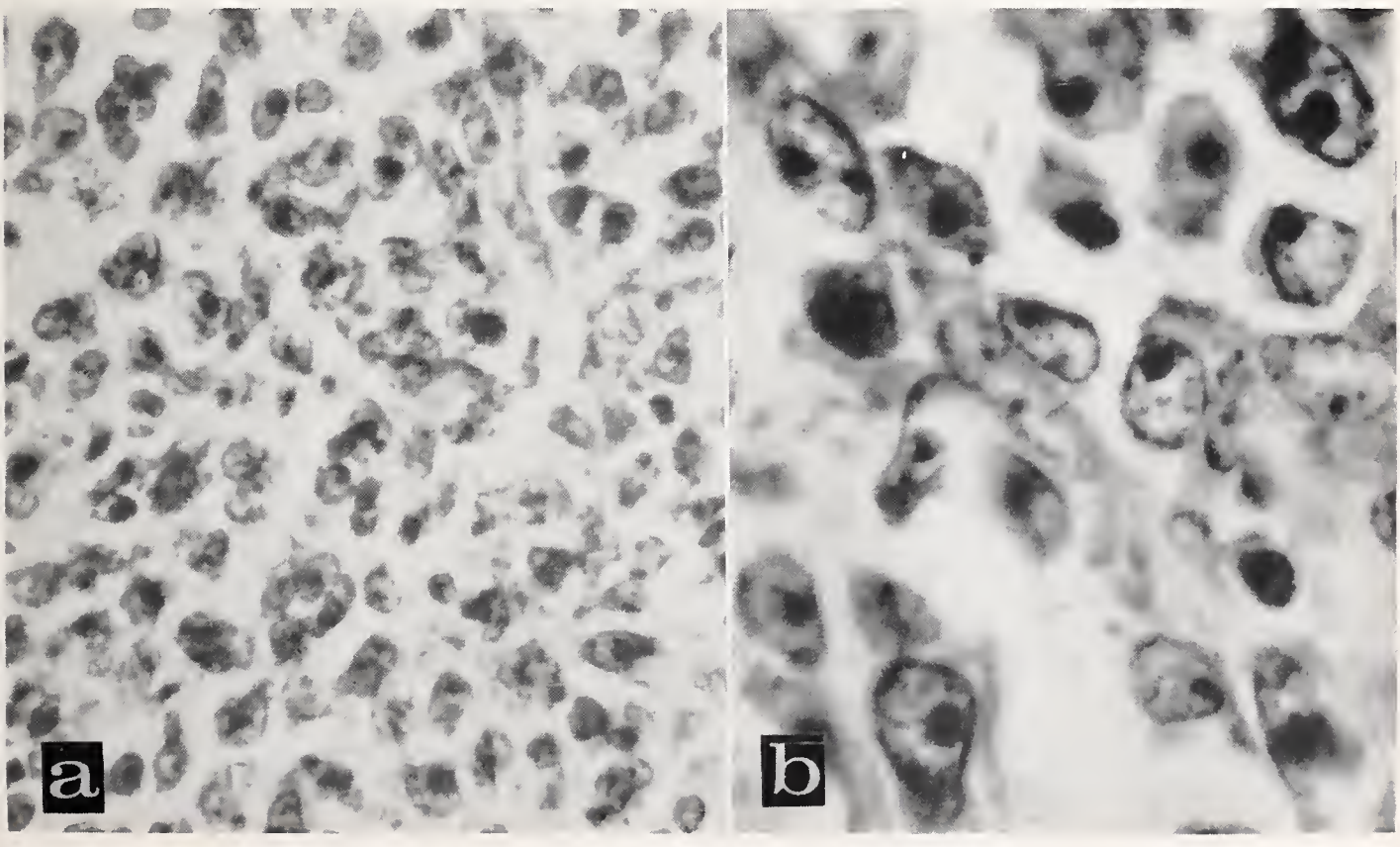
Diagnosis. — Metastatic malignant melanoma.

Contributor.—Dr. Horn.

History.—A 72-year-old white man with no history of injury or weight loss noted a nontender mass in the right axilla for one week. History included treated pernicious anemia, epilepsy controlled by Dilantin, cerebral hemorrhage nine years before, transurethral resection of the prostate two years earlier, hemorrhoidectomy, and appendectomy. The soft, freely movable tumor in the right axilla was 4 to 5 cm. in diameter. Studies were noncontributory. X-rays did not show any bone involvement by the tumor. Following biopsy, radical excision and axillary dissection were done. There was no metastasis to the lymph nodes.

DR. HORN: Several photomicrographs illustrate the varied appearances of this tumor. First is a spindle-celled tumor, not

Fig. 17. Case 16. Malignant lymphoma (Hodgkin's disease) of testes. a. X 400. b. X 885.6.



very good spindles, to be sure; many of them are more polygonal—a jumbled arrangement. You may see some arrangement in interlacing cords; they are not really good bundles, but resemble a tight, pinwheel whirl, and the structure is a bit more loose in some areas. In one section is a peculiar pattern which I have seen before, and I hope sometime it gets sufficiently etched on my memory so that I will realize what it means. There are packed cells around the outside of what appears to be a blood vessel, and another essentially similar grouping with a tendency toward degenerative change at a greater distance from these vessels. In another section are small, dark cells, bigger than lymphocytes, and I think they are tumor cells. This probably should be a clue to the nature of the lesion. Finally, one section strongly suggests that our lesion is in a lymph node, which I did not realize initially. It shows a lot of lymphoid tissue peripherally with a capsule that suggests lymph node—the sinusoids are not good, but I think that is what they are.

Submitted Diagnoses. — Leiomyosarcoma, 12; malignant schwannoma, 4; adult rhabdomyosarcoma, 4; hemangiopericytoma, 4; malignant hemangiopericytoma, 2; hemangioendothelioma, 1; fibrosarcoma, 3; neurofibrosarcoma, 1; synovial sarcoma, 1.

DR. HORN: All of the various types of spindle-cell tumor are represented. I do not remember what I called this when I first saw it, but it was probably fibrosarcoma. Since my diagnosis was incorrect, I feel free to introduce this case to humiliate all of us. Actually, a biopsy was done at this procedure. It was a fairly good-sized specimen, but nevertheless it was a biopsy, and was followed by a more radical procedure. This man returned in five months, and at this time said that his right thumb had gradually turned black on the tip approximately 15 years before, and approximately five years before, the area of pigmentation diminished except for a small black spot under the nail. Approximately five months ago, the spot had begun to grow, pushing the nail away from the skin and becoming painful. About the same time, the patient noticed a mass in his axilla. This was excised, and is the tumor that you have seen. A section from the thumb lesion shows tumor cells within interstices in the epidermis. I do not think these are really within the epidermis; it is a tangential cut. But comparison of this section with one of malignant melanoma with abundant pigment settles the whole problem.

This is a mistake that I hoped I would never make again. My associate, when shown these slides, made the correct diagnosis. I would like to discuss one other case because so many of us were trapped on this lesion.

An 18-year-old boy had a tumor of the

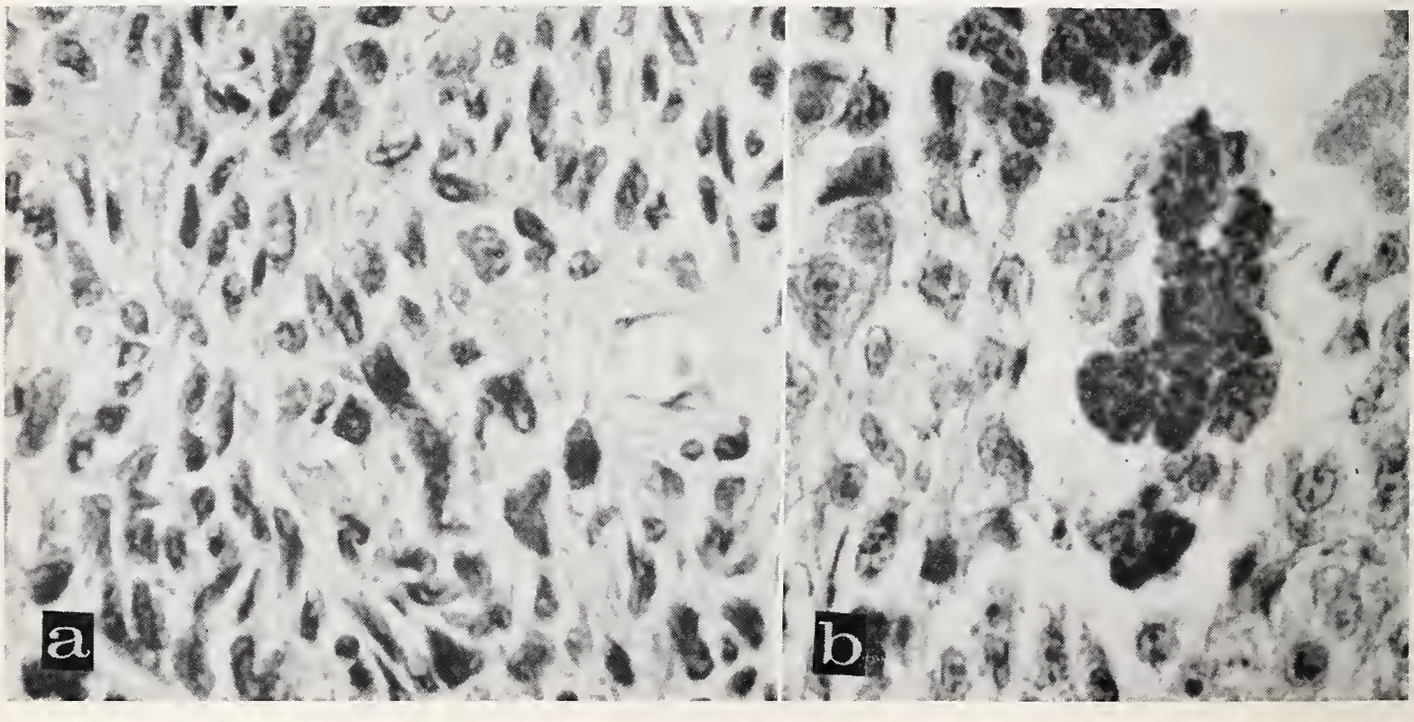


Fig. 18. Case 17. a. Metastatic malignant melanoma. X 334.8.

b. Primary lesion discovered later. Melanin is abundant. X 401.7.

axilla, and it showed many different appearances, much as in our first case, but with even more variation. There was a loose structure with spindle cells. Another pattern seen ought to mean something: cells were set in stroma in a peculiar fashion, and many were multinucleated. There was loose-textured appearance in some areas, with spaces; Tissue like this suggested cartilage, primitive cartilage, or precartilage to some of the consultants. We also saw the whorling around blood vessels that appeared in the seminar case. One section reminded one of the consultants of the neuroid appearance which he sometimes sees in neurogenic tumors.

In Michigan, the Michigan Cancer Registry has a board of consultants, and any pathologist can send specimens there for consultation. The submitter is anonymous but the consultants are known; perhaps they shouldn't be after this fiasco. Their diagnoses included lymphangioendothelioma, unclassified mesodermal tumor, malignant mixed mesodermal tumor, suggestions of rhabdomyosarcoma, leiomyosarcoma, possible cartilaginous tumor, possible vascular tumor, hemangiopericytoma, angioendothelioma, malignant hemangiopericytoma, malignant tumor, neurogenic, and so on. The patient had another resection, and some of the tissue had an appearance readily compatible with malignant melanoma, and there was abundant pigment. At this time, a history was obtained (I do not know the time interval) that three years before, a pigmented tumor had been resected from the skin of the interscapular area. This is a horrible example, and I hope you may learn as much from it as I have.

DR. BRANAM: How frequently do you encounter cases that seem to have a good histology for malignant melanoma, but no primary can be found?

DR. HORN: Case 2 was a malignant melanoma and the primary was not found. Some of these primaries can be in the skin in innocent-looking lesions. They can be, as Dr. Andujar pointed out, in a variety of internal sites as well. The anus is a dandy place to miss them.

COLONEL HOEFFLER: We had a case with a different outcome. A member of our staff had a dark brown lesion under his fingernail, and it looked like a melanoma. The case was presented to the tumor board, and after a great deal of discussion it was decided that a small excisional biopsy would be made. The "lesion" was a small deposit of silver; the man was a dentist. Some cases turn out more happily than Dr. Horn's.

DR. C. J. THUSS, JR., Brooke General Hospital: Dr. Horn, how often do you see amelanotic melanomas, or do you believe in them? If so, do you think if you look far enough and wide enough you will find pigment? Do you use Dopa stain?

DR. HORN: It is true that the more sections you look at, the more time you take, and the more special procedures you use to demonstrate melanin, the more often you will find it. Certainly, good observers have not always found pigment. Perhaps, if we exhausted the methods of looking, we might find pigment in all cases; I simply don't know.

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Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.

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NEWS

Texas Physicians May Participate In Individual Responsibility Program

The right of the individual physician to participate voluntarily in an individual responsibility program to prevent third party interference in the doctor-patient relationship was endorsed as ethical by the House of Delegates of the Texas Medical Association at its 99th Annual Session in Austin.

"Quality medical care can only be assured by maintaining the fundamental principles of the patient-physician relationship free from the influence of third parties," the House resolution stated.

A section of the Principles of Medical Ethics of the American Medical Association was cited: "A physician should not dispose of his services under terms or conditions which tend to interfere with or impair the free and complete exercise of his medical judgment and skill or tend to cause a deterioration of the quality of medical care."

Under the individual responsibility program, the physician, in filling out insurance forms, may fill out a statement covering only the technical information on his medical services to his patient. He would be responsible to his patient only.

On financial matters, the patient would be responsible for payment for services rendered. The insurance company would be responsible directly to the patient.

The physician could voluntarily refuse assignments, thereby giving third parties (those other than the doctor or the patient) no economic or legal grounds on which to establish fee schedules or relative value scales or to influence medical decisions.

The individual responsibility standard form may be used as a means of placing responsibility for settling claims "where it belongs," with the insurance company and the patient.

"The standard," the delegates agreed, "was not meant to obstruct settlement of any claim but was designed to have the doctor furnish to the patient only medical information necessary for the settlement of the claim."

Dr. Howard R. Dudgeon, Jr., Waco surgeon, was named President-elect of the Association at the House of Delegate's final meeting Sunday, April 17.

Earlier, Dr. James D. Murphy of Fort Worth was installed as 1966-67 President at the Annual Luncheon at which Gov. John Connally was guest speaker. Dr. Murphy succeeded Dr. David Wade, Austin psychiatrist.

Also elected was Dr. John K. Glen of Houston to serve as Vice President for 1966-67.

Re-elected were Dr. Charles Max Cole of Dallas, Speaker of the House of Delegates, and Dr. Wendell H. Hamrick of Houston, Vice Speaker.

Dr. Charles P. Hardwicke of Austin and Dr. John M. Smith, Jr., of San Antonio both were named to additional terms as trustees.

Dr. Dudgeon, TMA's new President-elect, has served as a member of the House of Delegates since 1951 and was the 1965-66 Vice President of the Association. Previously he was a member of the Council on Scientific Advancement, chairman of the Committee on Cancer, and secretary and chairman of the Section on Surgery. In addition he was president of the Twelfth District Medical Society in 1953 and president of the McLennan County Medical Society in 1957.

A fellow of both the American College of Surgeons and the Texas Surgical Society, he formerly was chief of staff and chief of surgical service of Hillcrest Baptist and Providence Hospitals in Waco.

Author of 25 published medical papers, Dr. Dudgeon was president of the Third District, Texas Chapter, American College of Surgeons in 1955, and president of the Central Texas Journal Club, 1945-46.

A 1934 graduate of the University of Texas Medical Branch in Galveston, he is the son of Dr. Howard R. and Sue Montgomery Dudgeon, pioneer leaders in Texas medicine. His father served as President of the TMA, and his mother as President of the Woman's Auxiliary.

Dr. Dudgeon was recently elected to serve on the Waco City Council.

In the House of Delegates' election, the following Councilors were also named: Dr. A. H. Daniell of Brownfield, Second District; Dr. James G. Morris of Lubbock, Third District; Dr. Jay J. Johns of Taylor, Seventh District; Dr. Stanley E. Thompson of Richmond, Eighth District; Dr. James H. Sammons of Highlands, Ninth District; and Dr. Paul R. Meyer of Port Arthur, Tenth District.

Delegates to the American Medical Association are Dr. Sammons, Dr. Harvey Renger of Hallettsville, Dr. G. W. Cleveland of Aus-

tin, and Dr. John L. Otto of Galveston. Alternate delegates are Dr. N. L. Barker of Paris, Dr. Haden E. McKay, Jr., of Humble, Dr. Ridings E. Lee of Dallas, and Dr. E. P. Hall, Jr., of Fort Worth.

A total of 3,503 members, guests, exhibitors, and Auxiliary members attended the 99th Annual Session, slightly more than the 3,469 who attended last year's session. This year's attendance included 1,714 TMA members, 868 guests, 386 exhibitors, and 535 Auxiliary members.

Next year's centennial Annual Session will be held in Dallas.

Hospital-Based Physicians

In other action pertaining to ethics, the House of Delegates adopted a resolution to assist hospital-based physicians in their efforts to separate their professional fees from hospital charges. It declared that a physician should not engage in practices which dispose of his professional attainments or services to any hospital, corporation or lay body by whatever name called or under conditions which permit the sale of the services of that physician by such agency for a fee.

The House also disapproved of any arrangement under which any member of the Texas Medical Association merges his (or her) professional fee with hospital costs into a single charge to the patient. Local and county medical societies were urged to assist in implementing these policies through all appropriate legal, ethical, and communications channels available.

On the grounds that mandatory participation in a benevolent program is offensive, the House declined to approve resolutions calling for assessment of all members of the Texas Medical Association to provide more money for the Physicians Benevolent Fund. Instead, delegates approved a substitute resolution which notes that the Fund is worthy of bequests, memorial donations, and personal contributions, and calls for membership support.

Patient Care, Public Health

The House commended Dr. John F. Thomas for bringing to its attention the efforts to utilize "do-it-yourself" cytological kits in Texas, and endorsed his resolution disapproving the practice of public advertising, by whatever media, of "do-it-yourself mail-in kits" for the cytological detection of cancer.

Outgoing President Dr. Wade (left) turns over his gavel to the incoming TMA head, Dr. Murphy, during a luncheon installation ceremony.

The delegates disapproved a recommendation that a training program for non-psychiatrist physicians be sponsored by the TMA Section on Nervous and Mental Diseases in cooperation with other allied organizations and that a liaison committee be formed to formulate a teaching plan. Instead, delegates recommended that the TMA Committee on Mental Health continue to sponsor education of non-psychiatrist physicians.

Delegates recommended that various local medical advisory committees be urged to recommend a physician, preferably with psychiatric training, for the post of director of local community mental health centers.

Delegates approved a change in the Code for Physicians and Attorneys of Texas. A new section providing guides in dealing with release of medical records, in accordance with present laws, was approved.

Delegates recognized as extremely important the modernization of abortion laws in Texas and recommended that, if necessary, a special committee be appointed to study the problem.

The House adopted the recommendation of the Committee on Public Health to endorse and cooperate in an education program dealing with illicit traffic in amphetamines and barbiturates, passed by the last Texas Legislature.

Delegates also reaffirmed their previous action that privileged communications laws are undesirable.

Referred to the Committee on Military and Veterans Affairs was a resolution calling for VA Hospitals to subscribe to the same utilization standards as civilian hospitals.

Resolutions dealing with criticism of utilization review committees and withdrawal from the Joint Commission on Accreditation of Hospitals were disapproved, although problems pertaining to regulations regarding such committees were recognized. Delegates noted that if there are inequities in the Joint Commission requirements, specific suggestions to change them might be made to appropriate councils and committees.

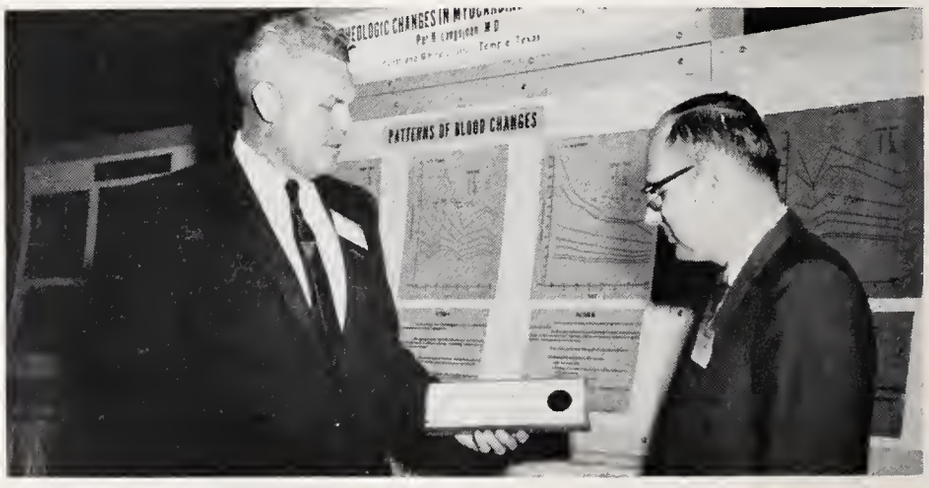
The House commended persons

Dr. Richard R. Royer, Detroit otolaryngologist, who conducted a refresher course, speaks at a scientific session.

Demonstrating plastics in medicine before the Eidophor closed-circuit color television cameras is John Autian, Ph.D., director of the Drug-Plastic Research and Toxicology Laboratories, Balcones Research Center, University of Texas.

Dr. David Wade (center), is shown during a light moment, visiting with a group of technical exhibitors at the reception given in their honor.

Gov. John Connally arrives at auditorium to speak at the Annual Luncheon.

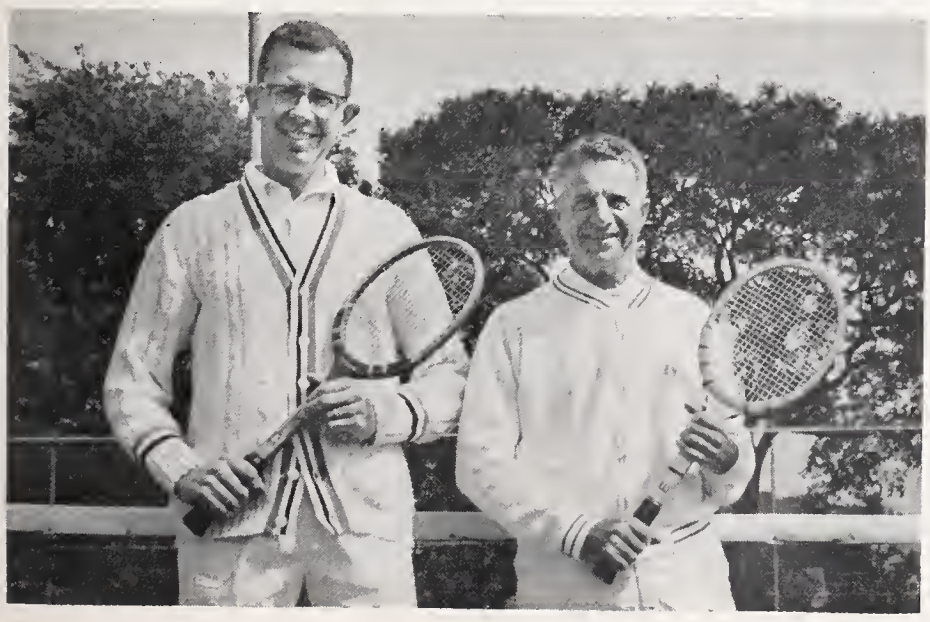


Dr. Per H. Langsjoen (left) of Scott and White Clinic, Temple, shows his Aesculapius Award plaque to his assistant, Dr. Santiago A. Sanchez. He received the plaque for his exhibit, "Rheologic Changes in Myocardial Infarction."

Roy Franklin, Jr. (right), author of the 1966 Guy Witt Memorial Paper, receives his prize from Dr. Charles L. Bloss of Dallas. Mr. Franklin is a senior medical student at the University of Texas Medical Branch at Galveston.

Dr. James D. Murphy, 1966-67 President, discussed the changing medical horizon before the House of Delegates.

Dr. Charles Max Cole of Dallas, Speaker of the House of Delegates, presides at a business session of the Delegates.



William Squires (right) of San Antonio, field manager of Parke Davis and Company, presents a reproduction of a History of Medicine painting to Dr. G. V. Brindley, Sr., of Temple, for display in TMA Headquarters in Austin. The picture was one of four framed reproductions presented by Parke Davis to the TMA.

Dr. Robert Mertz (left) of Corsicana won the singles championship of the TMA Tennis Tournament from challenger Dr. Bernard T. Fein of San Antonio.

involved in the execution of the billboard campaign against disease, approved by the House last year, and noted that it was "one of the most valuable public relations measures that has been available to the Texas Medical Association in recent years."

In addition to topics already planned or underway for the campaign (cancer, measles, tetanus, PKU, polio, mental health, heart disease, tuberculosis, eye diseases, and prevention of brain damage), it was suggested that topics include venereal disease, diabetes, and other medically related problems.

The House expressed thanks to the sponsoring societies, voluntary health agencies, and pharmaceutical companies for their contributions, and recommended that the President consider appropriate letters of appreciation and that county societies thank local advertising agencies which furnish billboards.

Government Programs

The delegates concurred with previous discussion by Dr. David Wade, the outgoing President, that the Association must continue to be active in maintaining communications with all phases of government related in any way to medicine. They also agreed that it is necessary to explore new methods by which the role of medicine can be strengthened and through which current concepts of the patient-doctor relationship can be maintained.

Delegates approved a resolution supporting the Executive Board of the Texas Medical Association in its action on Jan. 23, 1966, in exploring the possible need for a statewide advisory committee for the Heart Disease, Cancer, and Stroke program. They recommended that the Association actively determine the most effective method or methods of applying for a planning grant and see that a written planning grant proposal is submitted to the appropriate authority in the Department of Health, Education and Welfare. Such an effort will insure coordination and cooperation between major medical institutions and private practicing physicians in Texas and will also insure that there will be no change in the current concept of the private practice of medicine.

Delegates commended health insurance companies cooperating in the Texas 65 program for insuring elderly persons and expressed regret that the discontinuance of many insurance programs was necessary. They urged companies to continue present coverage to persons who desire it.

Dr. Howard R. Dudgeon, Jr., Waco surgeon, takes office as 1966-1967 President-elect of Texas Medical Association.

Reactivation of Committee

The delegates called for reactivation of the Committee on Liaison with Blue Shield, with membership to be composed equally of physicians on the Board of Blue Shield and physician-members of the Texas Medical Association not associated with Blue Shield. Delegates noted that many problems which have arisen might have been partially solved had the committee been active.

Delegates approved a resolution calling for children examined under the Project Head Start program and found to be possible heart patients to be referred to private physicians chosen by the children's parents, if they have a private physician, rather than to be referred to a physician selected by Head Start personnel.

Outgoing President Dr. Wade forecast that many smaller specialized paramedical groups will come to the Texas Medical Association for ethical advice and will seek its assistance in communicating with government. This was referred to the Council on Public Relations and Public Service, which will consider study of the problem and laying out guidelines for future alliances with such groups.

Other Programs

The House approved the formulation of a central statewide Committee on Voluntary Areawide Hospital and Health Care Facility Planning, to be composed of six health groups and laymen, and encouraged the committee to proceed as quickly as possible in working out the mechanics and functions of this project.

A resolution calling for the Texas Medical Association to help devise a program to train medical secretaries and to provide refresher courses where needed was turned down on the basis that this could be better determined at the local level, if and when there is local need. It was noted that the Texas Medical Assistants Association is a source of continuing education.

Delegates adopted a resolution allowing Texas Delegates to the American Medical Association full latitude to determine what course of action might be most appropriate regarding a resolution pertaining to inequities in the assessment of dues of military members of the AMA.

At the same time, the House adopted a resolution recommending that the Texas Delegation be authorized to support a proposal for an increase of AMA dues up to $25.

Constitution and By-Laws Changes

A recommendation that the name of the journal of the Texas Medical Association be changed from Texas State **Journal of Medicine** to Texas **Medicine** was referred to the Board of Councilors to interpret whether such a change should be a constitutional amendment. The Councilors determined that the Board of Trustees had the right to change the name of the journal.

The House adopted a resolution

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Perry C. Talkington, M.D.

Medical Director
Charles L. Bloss, M.D.

Clinical Director
Howard M. Burkett, M.D.

Associate Psychiatrists
James K. Peden, M.D.
Jerry M. Lewis, M.D.
Ward G. Dixon, M.D.
Claude L. Jackson, M.D.
E. Clay Griffith, M.D.
John Henry Reitmann, M.D.
Dode Mae Hanke, M.D.
Maurice S. Green, M.D.
Stanley L. Seaton, M.D.
Thomas H. Allison, M.D.

Clinical Psychology
David Lipsher, Ph.D.
Charles Jack Black, Ph.D.
Dale R. Turner, Ph.D.
John T. Gossett, Ph.D.

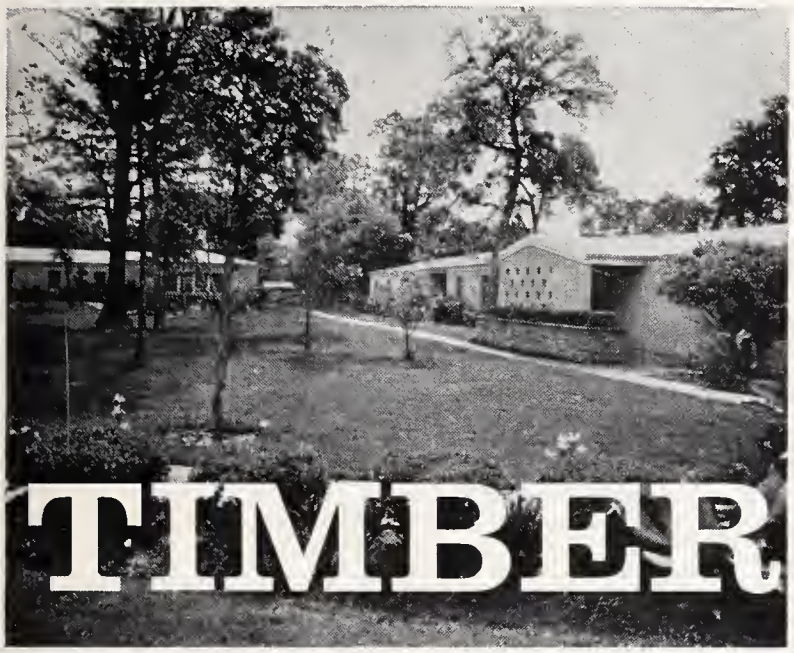
Social Work
Bill M. Turnage, M.S.S.W.
Robert M. O'Connor, M.S.W.
Barbara Lewis, M.S.S.W.
Carroll David, M.S.S.W.
Gerald Spalding, M.S.W.
Sally Stansfield, M.S.W.

Business Manager
Ralph M. Barnette, Jr., B.B.A.

Occupational Therapy
Geraldine Skinner, B.S., O.T.R.

Recreational Therapy
Lois Timmins, Ed.D., Director
Jacqueiyn Vaughan, B.S.
Claudette Gardner, Psychodrama
Mary Weldon, B.S., Music Therapy

Director of Nurses
Frances Lumpkin, R.N., B.S.



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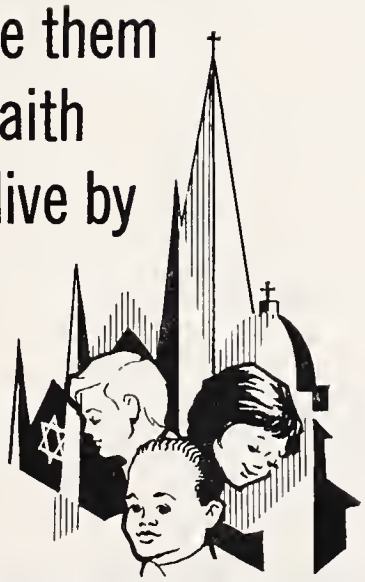

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submitted by the Board of Councilors asking that the Council on Constitution and By-Laws propose and submit to the House of Delegates necessary amendments regarding changing the status of a regular member to a provisional member of the local medical society when the member has been placed on probation by the Texas State Board of Medical Examiners.

A decision regarding possible formation of a scientific section on urology was deferred until the Texas Urological Society has completed a study and the topic is reconsidered by the Council on Annual Session.

Referred back to the Council on Constitution and By-Laws were proposed changes dealing with the nature of district societies and changes dealing with military membership and membership of certain other categories of physicians not required to register under the Medical Practice Act of Texas.

Approved was a recommendation which called for the immediate Past President of the Association to be included for one year as an ex officio member of the House of Delegates. This requires a constitutional change and must wait one year before the final vote can be taken.

Adopted was a by-laws change which adds a sentence to Chapter VIII, Section 2. The new sentence will read: "Should any standing committee fail to meet during the entire year between the end of one annual session and the end of the following annual session of the Association the incoming President shall have the prerogative of replacing or discharging any or all members of the committee, and/or of recommending to the House of Delegates that the committee itself be abolished."

The delegates adopted a change dealing with leave of absence. It allows county medical societies to grant a leave of absence to physician members who leave the active practice of medicine for a proposed period of one year or more to engage in training, missionary service outside the geographical limits of the United States of America, temporary service in the US Armed Forces, temporary civilian service outside the geographical limits of the United States, and sick leave.

Medical Assistant Week in Texas on May 15-21 has been proclaimed by Gov. John Connally. Witnessing the signing of the proclamation in the Governor's Office in Austin are Miss Josephine Estrada of San Antonio, president of the Texas Medical Assistants Association, and Dr. David Wade of Austin, Past President of the Texas Medical Association. Highlight of the week was the annual convention of the Texas organization on May 19-22 at the Sheraton-Lincoln Hotel in Houston.

Dr. LeMaistre Named UT Vice Chancellor

Dr. Charles A. LeMaistre, professor of internal medicine and associate dean of the Southwestern Medical School in Dallas, will become vice chancellor for health affairs for the University of Texas, with headquarters in Austin, according to an announcement by Chairman W. W. Heath of the UT Board of Regents.

Dr. LeMaistre will be responsible for the effective and economical coordination of the medical schools and other health science programs in education and research, Mr. Heath said. An earlier vice chancellorship for medical affairs which was chiefly advisory was suspended several years ago.

A native of Alabama and a graduate of the University of Alabama and Cornell University, Dr. LeMaistre has had a distinguished career both in research and in medical administration. He has served in major academic posts at both Cornell and Emory University. Appointed to the Southwestern Medical faculty as full professor in 1959, he became associate dean in 1965. During the past year he has conducted a system-wide survey of the medical schools of the University of Texas.

His public appointments have included consultantship for the Veterans Administration and serv-

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References: 1. Shuman, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuck, E., et al.: Angialagy, 12:102, 1961. 3. Rawls, W., et al.: Med. Times, 87:818, 1959.

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Among the speakers at the Memorial Service are (from left) Dr. R. Vincent Murray of Austin, who gave the benediction; Mrs. Charles D. Bussey of Dallas, who gave the memorial address for members of the Auxiliary; and Dr. R. Maurice Hood of Austin, who paid tribute to deceased physicians.

ice as senior assistant surgeon in the US Public Health Service. A member of many scientific and medical societies, Dr. LeMaistre has published widely in professional journals.

Dr. Langsjoen Is Winner Of New Exhibit Award

Winners of awards for outstanding scientific exhibits were announced during TMA's Annual Session in Austin.

The Aesculapius Award for the most outstanding scientific exhibit was presented to Dr. Per H. Langsjoen of Scott and White Clinic, Temple, for his presentation: "Rheologic Changes in Myocardial Infarction."

The award, an innovation this year, included a certificate and a cash prize of $200 presented by Mead Johnson Laboratories to encourage the development of scientific exhibits.

Other winners included first place award in the Individual Exhibits category, given to Dr. Harold C. Urschel, Jr., and Dr. Donald L. Paulson of Dallas. Second place winner was Dr. Robert J. Rowe of the University of Texas Southwestern Medical School and Baylor University Medical Center, Dallas. Dr. Robert B. Crouch and Dr. Peter Sullivan of Houston took honorable mention.

First place winners in Group Exhibits were Dr. Marjorie Hendrickson and Dr. Stewart A. Wilber, University of Texas M.D. Anderson Hospital and Tumor Institute, Houston. Second place went to a group of physicians from Scott and White Clinic, Temple: Dr. N. C. Hightower, Dr. D. K. Wentz, Dr. R. D. Haines, Dr. A. C. Broders, Dr. H. Laurens, and Dr. J. A. Bargen. Honorable mention was given to Dr. R. A. Murray, Dr. H. H. Brindley, Dr. D. E. Pisar, and Dr. H. R. Collins, also of Scott and White Clinic.

In the Institutional Exhibits category, first place was a presentation of Baylor University College of Medicine, Houston, prepared by Dr. Denton A. Cooley, Dr. Robert D. Bloodwell, Dr. Grady L. Hallman, Dr. Sarjit Gill, and Dr. Arthur C. Beall, Jr. The Virginia State Penitentiary (Richmond) exhibit, prepared by Dr. Harry Brick, Mr. W. H. Dout, Jr., and Mr. W. C. Perdue, took second place. Honorable mention went to the Austin State Hospital for an exhibit executed by Mr. John Middleton, Dr. Clarence R. Miller, and Miss Velma Stigler.

The American Cancer Society, Texas Division, Austin, won first place in Educational-Promotional Exhibits. Second place went to the Texas State Department of Health, Division of Occupational Health and Radiation Control, with Mr. David K. Lacker and Mr. Julian Castillo participating. Honorable mention was given to the Texas Hospital Association Health Careers Program, Texas Hospital Association, Austin.

Memorial Service Honors 166 Dead

A Memorial Service held in the Commodore Perry Hotel in Austin on the last morning of the TMA's Annual Session paid tribute to 166 deceased members of the Texas Medical Association and the Woman's Auxiliary.

The memorial address for deceased physicians was given by Dr. R. Maurice Hood of Austin, and for deceased Auxiliary members, by Mrs. Charles D. Bussey of Dallas.

Mrs. Hallmark Named President of Auxiliary

Mrs. James Allen Hallmark of Fort Worth was installed as 1966-67 President of the Woman's Auxiliary to the Texas Medical Association at the group's final luncheon in Austin, meeting in conjunction with the TMA's 99th Annual Session.

Taking office as President-elect was Mrs. Frank M. Posey, Jr., of San Antonio.

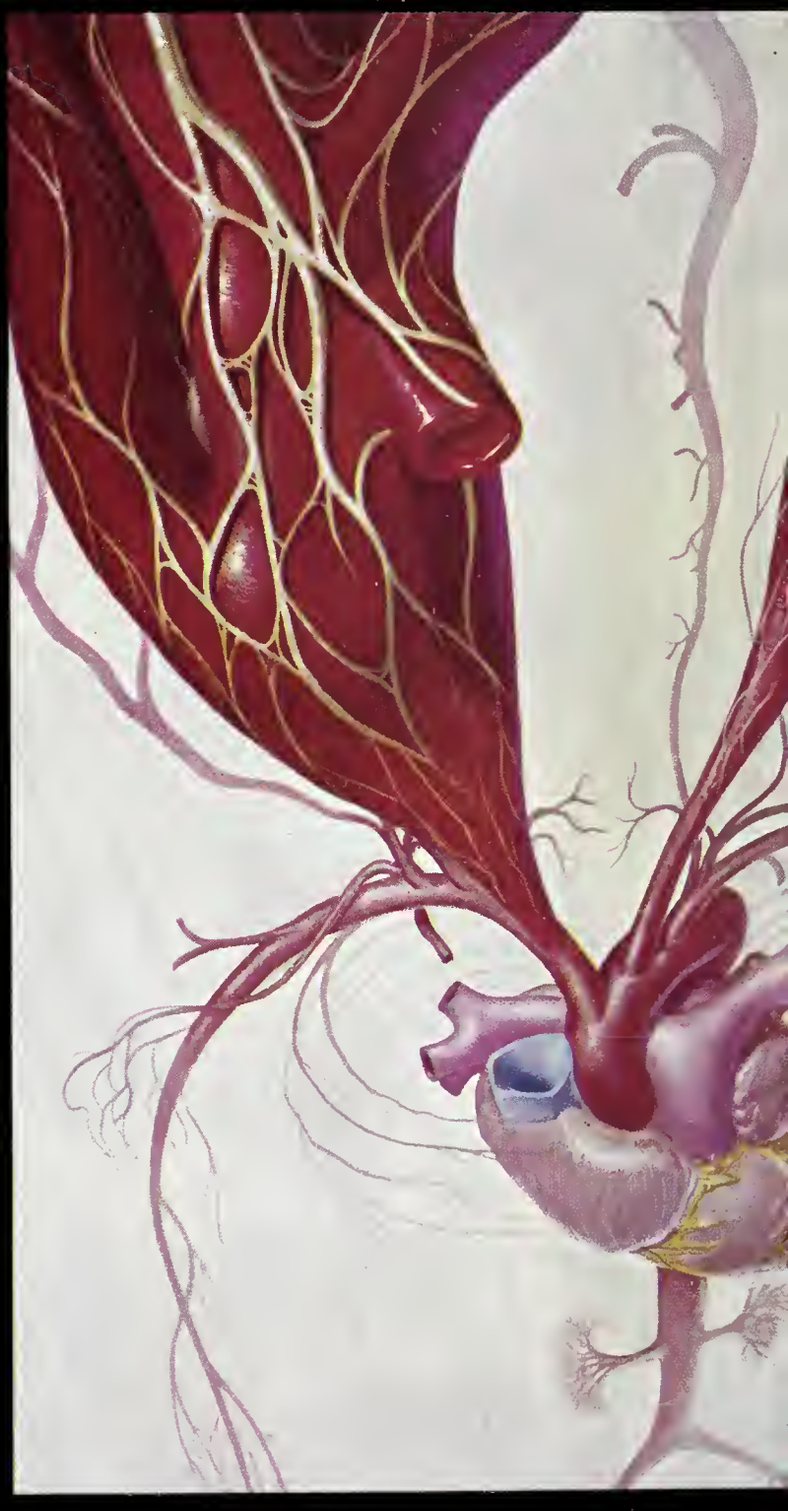
Other officers are Mrs. James C. Price of Gonzales, first vice president; Mrs. S. Braswell Locker of Brownwood, western regional vice president; Mrs. York Lancaster of Port Lavaca, southern regional vice president; Mrs.

Taking office as President-elect of the Woman's Auxiliary is Mrs. Frank M. Posey, Jr., of San Antonio. She was installed at the Auxiliary's Annual Session in Austin last month.

A Key Site of Action of the Protoveratrine A in Salutensin

"The main function of the carotid sinus is regulation of the blood pressure...."[1]

The veratrum component of Salutensin acts here (and in the myocardium), initiating "...a reflex fall in blood pressure through a generalized vasodilation and fall in heart rate."[2]



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In addition, to facilitate and maintain blood pressure reduction, Salutensin incorporates reserpine and a highly effective thiazide.

In general, side effects have been reported infrequently but may include those listed in the therapeutic summary.

Simple dosage—low-cost therapy: Many patients on Salutensin respond to 1 tablet *b.i.d.* Long-term economy is assured, since dosage can frequently be lowered after initial control is established.

Available: Prescription-size bottles of 60 tablets.

References: 1. Editorial: JAMA *191*:592 (Feb. 15) 1965. 2. Meilman, E., in Moyer, J.H.: Hypertension, Philadelphia, W.B. Saunders Company, 1959, p. 395.

BRISTOL THERAPEUTIC SUMMARY
For complete information consult Official Package Circular.

Indications: Essential hypertension.

Warnings: Small-bowel lesions (obstruction, hemorrhage, perforation) have occurred during therapy with enteric-coated formulations containing potassium, with or without thiazides. Such potassium formulations should be used with Salutensin only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs.

Contraindications: Salutensin is contraindicated in severe depression.

Precautions: Azotemia, hypochloremia, hyponatremia, hypochloremic alkalosis and hypokalemia (especially with hepatic cirrhosis and corticosteroid therapy) may occur, particularly with pre-existing vomiting and diarrhea. Potassium loss, which may cause digitalis intoxication, responds to potassium-rich foods, potassium chloride or, if necessary, stopping therapy. Serum ammonia elevation may precipitate coma in precomatose hepatic cirrhotics. Discontinue therapy two weeks before surgery or if myocardial irritability, progressive azotemia or severe depression occur. Exercise caution with patients with peptic ulcers or renal insufficiency (if severe, Salutensin is contraindicated).

Side Effects: Hydroflumethiazide: Purpura plus or minus thrombocytopenia, hyperuricemia, leukopenia, hyperglycemia, glycosuria, malaise, weakness, dizziness, fatigue, paresthesias, muscle cramps, skin rash, epigastric distress, vomiting, diarrhea and constipation. *Reserpine:* Depression, peptic ulceration, diarrhea, Parkinsonism, nasal stuffiness, dryness of the mouth and, with overdosage, agitation, insomnia and nightmares. *Protoveratrine A:* Nausea, vomiting, cardiac arrhythmia, prostration, excessive hypotension and bradycardia. (Treat bradycardia with atropine and hypotension with vasopressors.)

Usual Dose: 1 tablet *b.i.d.*

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James H. Kreimeyer of Rusk, eastern regional vice president; Mrs. W. Ray Hanna of Sulphur Springs, northern regional vice president.

Also Mrs. Robert E. Leslie of El Campo, recording secretary; Mrs. Frank B. Gooch of Fort Worth, corresponding secretary; Mrs. Richard B. Johns of Abilene, treasurer; Mrs. John B. Bourland of Dallas, publicity secretary; and Mrs. R. C. L. Robertson of Houston, parliamentarian.

During the Auxiliary's 48th Annual Session, presided over by Mrs. Russell L. Deter of El Paso, the 535 members attending the meetings agreed to donate more than $1,300 to the American Medical Association Education and Research Foundation (AMA-ERF), and $1,000 to the Texas Medical Association's Physicians Benevolent Fund.

Delegates also heard a report on the Youth Safety Conference, sponsored in March by the Woman's Auxiliary and the TMA, with the Texas Department of Public Safety, the Allstate Foundation, Dallas Citizen Traffic Commission, Dallas Junior Chamber of Commerce, and the Texas Safety Association as cooperating organizations.

Meeting in Dallas, the teenage delegates, limited in number to 500 chosen by high school principals from all over the state, gathered to hear a series of talks, demonstrations, and films on automotive safety and to participate in a mock trial.

Mrs. Hallmark, the Auxiliary's new President, has long been active in Auxiliary affairs as well as in Fort Worth civic activities. As woman's chairman of the Water Board Drive, she wrote a resume which was sent by the Chamber of Commerce to local voters. That resume later was awarded first prize by the Sinclair Oil Company in a national advertising contest.

She served as woman's chairman of the Texas Academy of General Practice 1963 convention, in addition to her many activities in the Woman's Auxiliary to the Tarrant County Medical Society, ranging from committee assignments to president, and in the state Auxiliary.

She is active in the Fort Worth Woman's Club and the Community Theater. A gourmet cook, greenthumb gardener, and flower arranger, Mrs. Hallmark is a voracious reader, covering a wide range of subjects from novels to history and politics. Travel has long been a delight to her, and she is considered an astute business woman, having even contracted and supervised the building of her husband's office and of the family home.

Mrs. James A. Hallmark of Fort Worth, new President of the Texas Medical Association's Woman's Auxiliary, holds a bouquet of dollar bills donated by the Tarrant County Woman's Auxiliary. Instead of roses, Mrs. Hallmark was presented with the dollars at her installation to further her favorite project, the TMA Physicians Benevolent Fund.

Mrs. Hallmark attended Texas Christian University and the University of Colorado, receiving both bachelor and master degrees from the University of Texas.

Dr. and Mrs. Hallmark have two sons, James Allen, Jr., and Shelby Stuckert. They are members of the Methodist church.

Endowed Professorship Goes to Medical Branch

An endowed professorship, memorializing the late James W. Rockwell of Houston, has been established at the University of Texas Medical Branch at Galveston.

University Chancellor Harry Ransom said the endowment, given by Rockwell Fund, Inc., will create at the Galveston school the James Wade Rockwell Professorship in Library Science and Medical History.

Mr. Rockwell, a member of the University Board of Regents for several years, was a long-time supporter of University of Texas programs.

Outdated Product Standards Due Official Withdrawal

A number of existing Simplified Practice Recommendations and Commercial Standards may be withdrawn by the Office of Product Standards (formerly the Office of Commodity Standards) of the National Bureau of Standards.

These standards are considered obsolete, technically inadequate, no longer acceptable to nor used by the industries concerned, or otherwise not in the public interest. Many of the standards are 30 years old, and some have not been revised since they originally were published.

Those which could affect the medical profession are standards on hospital plumbing fixtures, dental hypodermic needles, dental brush wheels, packaging of dental plaster, investment, and artificial stone.

Also dental lathe grinding wheels, dental cone-socket mirrors, dental excavating burs, hospital rubber sheeting, surgeons' rubber gloves and surgeons' latex gloves.

"The doctor will be in as soon as one of them draws a short straw."

OAA Benefits Told By Insurance Group

In the latest figures for the Old Age Assistance health benefits program of the Kerr-Mills Act, 379,262 hospital admissions have been recorded between Jan. 1, 1962, when the program began, to April 1, 1966.

Handled by Blue Cross-Blue Shield of Texas, 944,844 payments, amounting to $92,236,078, have been made to doctors and hospitals during that period, with more than 97 cents of each dollar dedicated to this purpose being paid in benefits.

The OAA program will be concluded on June 30.

According to Blue Cross-Blue Shield figures, there have been 346 hospital admissions which have resulted in stays of more than 200 days. One patient, it was noted, stayed 1,369 days, more than three years and nine months.

Despite these long-stay cases, however, the average stay for hospitals has been only slightly more than nine days, considerably less than the experience in other states.

"We believe that this fact is attributable primarily to the efforts of the medical profession and the hospitals to make certain that the benefits under this program are wisely and appropriately used," stated Mr. Fred R. Higginbotham, of Blue Cross-Blue Shield of Texas.

The Texas program, according to Mr. Higginbotham, has been widely acclaimed as an outstanding success throughout the nation.

Rather than establish another state program, it was determined that benefits for the needy aged in Texas would be provided on an insured basis whereby these persons, with complete preservation of human dignity, could obtain necessary health benefits on the same basis as any other person with Blue Cross-Blue Shield coverage, Mr. Higginbotham said.

"I've never achieved much fame, but many people are resting on my laurels."

Plan to Facilitate Drug Information

A cooperative plan between private industry and government has been designed to make scientific journal information on drugs more readily available to the scientific community.

Under the arrangement between the Pharmaceutical Manufacturers Association and the National Library of Medicine, PMA will hire and finance four interns to study NLM's medical literature indexing and retrieval techniques.

Each trainee will spend part time at NLM and part at PMA learning the principles of the jobs. The two organizations will exchange advice and information concerning published drug reports. Later this information will be shared with others in the drug field through a PMA Service Center.

The agreement will become effective by July 1 and will run for two years. The program, however, will extend beyond that period to provide a continuing scientific literature service with special application to pharmaceutical research.

Dr. Delgado Receives Acclaim for Teaching

Dr. Jaime N. Delgado of Austin, associate professor pharmaceutical chemistry at the University of Texas, has been given an award for excellence in teaching by the University's Students' Association.

A co-author of the *Texas Medicine* column, "Advances in Drug Research," Dr. Delgado was one of 15 UT faculty members chosen to receive the award.

Another winner, Dr. Natalie Barraga, assistant professor of special education, was a speaker at the Conference on Physicians and Schools, held during the Texas Medical Association's Annual Session. She discussed emotional stresses in educational settings.

Grant Awarded UT For Plastics Study

A grant of $103,000 has been awarded to the University of Texas Drug-Plastic Research and Toxicology Laboratories, College of Pharmacy, by the National Institutes of Health.

Dr. John Autian, laboratory director, and Dr. Wallace L. Guess, assistant director, will be in charge of the research.

The funds will be used for continued research in plastics to be used as collection, storage, and administration devices for blood and blood products. One of the main objectives of the study is to determine whether one or more constituents from the plastic material will enter the blood product and thus alter the product.

The development of techniques for predicting the behavior of plastic material under a number of environmental conditions will also be studied.

Pamphlets

"The Cancer Story," a booklet prepared by the National Cancer Institute and issued by the US Public Health Service, explains to the general reader the nature of cancer, how it is diagnosed and treated, and how science is learning more about it.

The 52-page publication is expected to receive wide use in junior and senior high schools as a teaching aid. A simplified version of "Science and Cancer" for those who have not studied biology, it introduces essential technical terms with a glossary to explain the terms.

Single copies may be requested free from the US Public Health Service, Washington, D.C., 20201. Multiple copies may be purchased from the Superintendent of Documents, US Government Printing Office, Washington, D.C. 20402. The price is 25 cents each, with a discount on quantities over one hundred.

A new illustrated pamphlet, "Peptic Ulcer," is now available from the US Public Health Service, Department of Health, Education, and Welfare.

The 13-page pamphlet presents general background information on

For cold hands and feet, nothing beats hot stoves—but they *are* awkward to carry around. Now Gerilid, in good-tasting take-along chewable tablets can provide rapid vasodilation of peripheral circulation, bringing real warmth to the extremities and decreasing sensitivity to sudden temperature change. Patients *like* Gerilid and *know* they are getting relief.

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peptic ulcers, in easily understood language. It outlines causes, symptoms, techniques for diagnosis, and current methods of treatment.

Single copies of "Peptic Ulcer" —USPHS Publication No. 280, Health Information Series No. 71 —are available from the US Public Health Service, Washington, D.C. 20201. It may also be purchased from the US Government Printing Office, Washington, D.C. 20402, for 15 cents per copy.

"It's Not Too Late to Stop Smoking Cigarettes!," written by Alton Blakeslee, a science writer, has been published by the nonprofit, educational Public Affairs Committee.

Two research studies, conducted over a period of six to eight years, provide the evidence for Mr. Blakeslee's pamphlet. One study, which involved microscopic examination of cells from the lungs of smokers, non-smokers, and ex-smokers, concluded that "safety calls for 'giving up the habit now'."

The second study, conducted by the American Cancer Society, is a continuing study of more than a million living persons.

"This big study," writes Mr. Blakeslee, "shows dramatically that people who keep on smoking die sooner than those who do not smoke."

The pamphlet is available for 25 cents from the Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.

"Mental Health of Children—The Child Program of the National Institute of Mental Health," a new publication of the US Public Health Service, Department of Health, Education, and Welfare, discusses activities designed to provide techniques, knowledge, and services that can help reverse the rising tide of emotional disturbance in children and enhance the well-being and productivity of all youth.

In describing programs to develop better mental health for the nation's youths, the report draws from findings of 153 investigations supported by the National Institute of Mental Health.

Single copies of the pamphlet, USPHS Publication No. 1396, are available without charge from the Public Information Section, National Institute of Mental Health, Bethesda, Md. 20014. Multiple copies can be purchased from the Superintendent of Documents, US Government Printing Office, Washington, D.C. 20402, at 40 cents per copy.

"Epidemiological Study of Cancer and Other Chronic Diseases," a monograph issued by the National Cancer Institute of the National Institutes of Health, US Public Health Service, reports a group of studies in the epidemiology of cancer and cardiorespiratory diseases.

It includes findings from several large-scale prospective studies — those in which data about habits and other personal characteristics of healthy people are recorded and later related to their illnesses or cause of death.

Edited by William M. Haenszel, chief of the Institute's Biometry Branch, the monograph includes four interim reports of three prospective studies on the relation of smoking to health. Three papers deal with the design of a study to determine the rates of cardiorespiratory diseases and cancer among middle-aged United States, British, and Norwegian men, and to try to find reasons for the apparently large differences among the groups.

The monograph is available from the Superintendent of Documents, Government Printing Office, Washington, D.C. 20402. The single-copy price is $3.75.

AMA-ERF Funds Go to Texas Schools

The American Medical Association Education and Research Foundation distributed $26,462 to Texas medical schools during April.

More than $1 million was distributed nationwide, representing contributions from physicians and woman's auxiliaries in 1965 to the AMA-ERF Funds for Medical Schools program. The money goes toward faculty salaries, improvement of laboratories and libraries, student assistance programs, and general operating expenses.

Recipients of the funds included the University of Texas Medical Branch at Galveston, $8,455; Baylor University College of Medicine, Houston, $8,748; the University of Texas Southwestern Medical School, Dallas, $8,113; and the University of Texas South Texas Medical School, San Antonio, $1,146.

Obstetrics, Gynecology Post Endowed at Dallas

A new professorship of obstetrics and gynecology has been established at the University of Texas Southwestern Medical School in Dallas by the University's Board of Regents.

The Gillette Professorship has been endowed by a gift of approximately $100,000 from Mr. and Mrs. W. Everett DuPuy of Houston and the Fred G. or Maggie R. Gillette Foundation of Houston. Mrs. DuPuy is the daughter of Mr. and Mrs. Gillette, and members of both families are alumni of the University of Texas.

33 Physicians Awarded Honorary Memberships

Thirty-three Texas physicians have been awarded honorary membership in the Texas Medical Association by the House of Delegates.

Among the doctors who were nominated for the honor by their county medical societies was Dr. A. Lane Mitchell of Houston, who was named posthumously.

Other doctors honored include Dr. Fred H. Cariker, Childress; Dr. William P. Klingensmith, Sr., Amarillo; Dr. Charles F. Bailey, Ballinger; Dr. C. C. McClure, Jacksboro; Dr. Elisha D. Embree, New Braunfels; Dr. William R. Powell, Laredo; Dr. C. J. I. Ekman, Livingston.

Also Dr. Otis O. Benson, Jr., Dr. Howard M. Bush, Dr. Edward Alexander Cayo, Dr. Thomas Hilliard Sharp, all of San Antonio; Dr. James A. Greene, Dr. David Greer, Sr., Dr. Herbert T. Hayes, all of Houston; Dr. Peyton C. Clements, Dr. Linwood H. Denman, Dr. L. T. Tinkle, all of Lufkin.

Also Dr. Martin L. Edwards, Sr., Hawkins; Dr. William P. Ball, Dr. Marshall T. Knox, both of Cleburne; Dr. Thomas E. Crump, Cameron; Dr. Hugh V. Helbing, Dr. Joseph Hamilton Steger, both of Fort Worth; Dr. Hershall Laforge, Uvalde.

Also Dr. James William Bass, Dr. Ernest W. Breihan, Dr. Harold T. Nesbit, Dr. Curtice Rosser, Dr. Ralph C. Smith, all of Dallas; Dr. Lane B. Cooke, Dr. Davis Scott Hammond, Dr. Owen R. O'Neill, all of Paris.

Amigos de Honduras

A stela from the ruins in Copan, an ancient Mayan city dating back to 800 A.D. No work of this artistic quality is produced in Honduras today.

Dr. Ralph D. Eichhorn and a group of youths from two Houston Baptist churches last summer put friendship theories to actual practice. As the *Amigos de Honduras,* the youngsters spent three, six, or nine weeks giving medical assistance to the Honduran people. Coordinated by Guy Bevil, Minister to Youth of the Houston River Oaks Baptist Church, the program provided a broad spectrum of immunization to the people of Honduras.

Youth volunteers, aged 15 to 21, from the River Oaks and Willow Meadows Baptist Churches, accompanied by counselors and 12 doctors from AMDOC, administered immunizations and other medications. Prior to leaving for Honduras, the 268 youngsters practiced giving shots on oranges and each other.

Bevil and the Honduran health authorities devised a program in which the group, *Amigos de Honduras*, would administer diphtheria-tetanus, diphtheria-pertussis-tetanus, smallpox, polio, BCG, and typhoid vaccines, and worm medication.

Cooperation was received from the Honduran health authorities and army. Everywhere the *Amigos* went, the army allowed their trucks and jeeps to pass without inspection, and even bulldozed a way to villages without roads. Honduras has approximately 100 miles of paved roads—the most "incredibly horrible" roads, reported Dr. Eichhorn. A 100-mile drive took six hours by jeep.

The army and health authorities also contacted and prepared the villages for the *Amigos* visit. Village *alcaldes* cooperated and lined up the families for inoculations. When the course of immunization was completed in one village, leaders of nearby towns would bring their people in for inoculations.

The medical problems of Honduras are numerous and insuperable. Lack of sanitation facilities and failure to pen in domestic animals result in universal water contamination.

Even though many devise shoes from rubber tires, most Hondurans go barefoot. Hookworm is epidemic, and mass enteric infections result from water contamination, improper food handling, lack of food inspection, cross contamination from insect vectors, and the use of night soil in agriculture. Bodies of young and old alike are bloated from worm contamination. Any improvement program in Honduras would have to begin with the fecal, domestic animal and water contamination problems.

Although fresh fruit is available, most of the people eat rice, beans, and *tortillas*, supplemented by an occasional egg, or small piece of pork or chicken. The absence of meat in the general diet leads to severe malnutrition and beriberi.

A combination of enteric infection, worms, and malnutrition results in severe anemia and generalized weakness. Parasites, enteric infection, malnutrition, and tuberculosis remain the major health problems in Honduras. The government is striving to eradicate tuberculosis by increasing the use of x-ray, BCG immunization, and treatment with chemoprophylaxis. Dr. Eichhorn found it remarkable that the major health killers of our nation, cancer and heart disease, were rarely seen among Hondurans.

A predominately illiterate people, many Hondurans place education as their first necessity instead of sanitation, public health, and preventive medicine. Dr. Eichhorn believes that in this military dictatorship, the health problems could be successfully attacked by government programs.

Another problem of the country is the cut, slash, and burn or "milpa" system of agriculture, a system 2,000 years old. A forest is felled, burned, and corn is planted, often on vertical slopes. The area is used up and the natives move to an adjoining plot of ground to repeat the process. Soon the forest disappears and the wasted land turns victim to erosion and desolation.

There is no telephone communication in Honduras, not even in most state capitals. The only means of communication between the towns is telegraph; these lines are frequently down, or the telegraph office is closed due to a fiesta. When a telegram arrives, it is garbled and often three to four days late.

Probably the most important problem of the *Amigos* was lack of communication. One group wired, "We are out of vaccine, alcohol, needles, and syringes. What do we do?" At the time, Dr. Eichhorn did not have an available jeep or supplies, so he simply wired back, "Smile!"

The *Amigos* faced many health problems. In preparing for their own medical needs, they received medication to ward off enteric infections, known to be rampant, and malaria, which has been controlled.

Since water was contaminated, it had to be kept in canteens and sterilized by boiling or iodination. Iodine tablets, even though they made the water distasteful, made it safe for brushing teeth, cleaning glasses and silverware, and cleaning the rinds of fresh fruit before slicing.

Knowing little about the environment of the country, the *Amigos* were prepared for flies and mosquitoes, but not for fleas. Everyone in the group was flea-bitten, and there was nothing to relieve the itch.

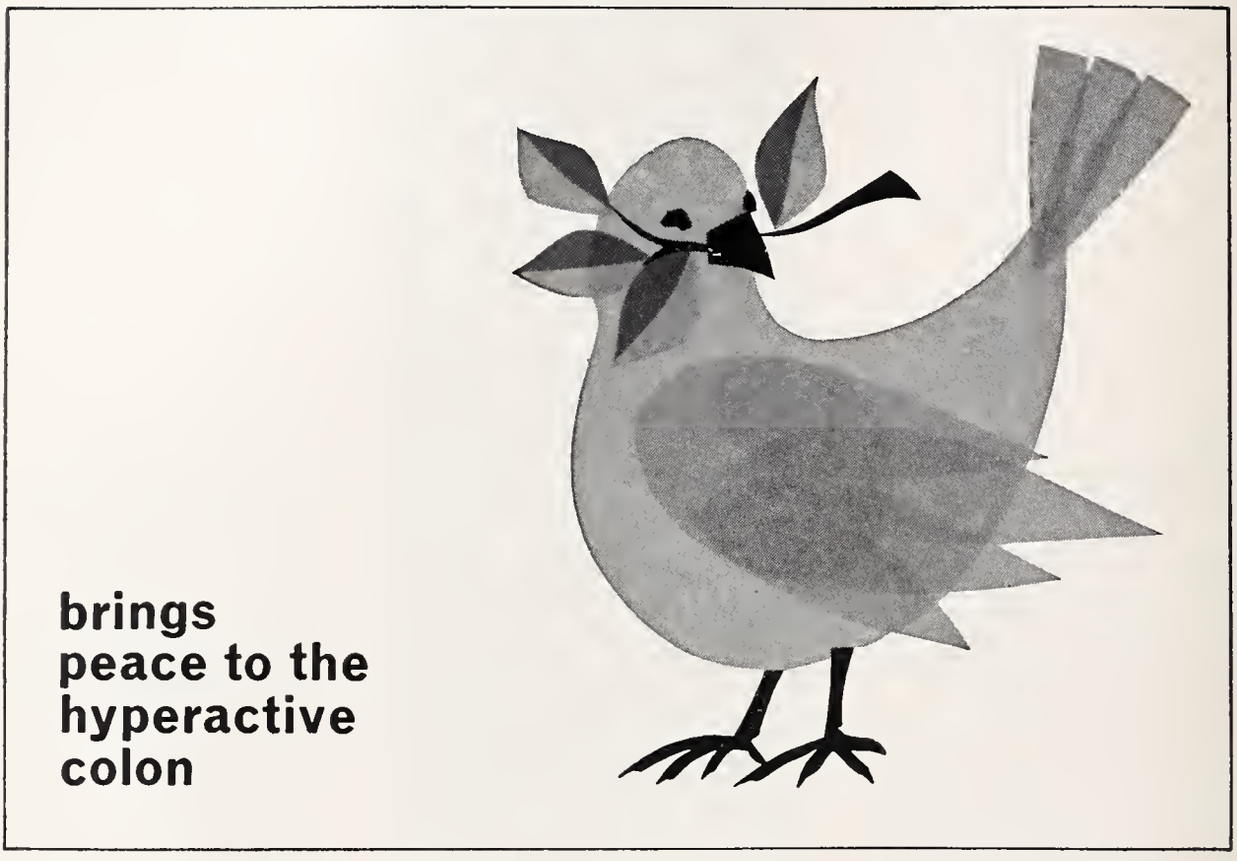
The *Amigos* lived in homes of the villagers, who were responsible for feeding and contributing to the cost of food. Even the poorest Honduran contributed. Coming to the immunization center with a slip of paper stating they had contributed 25 *centavos* to feed the *Amigos de Honduras* gave a real sense of pride. The Hondurans also provided secretarial help and worked with the group, making it a community project.

Another difficulty was the unattractive food. Although the Hondurans attempted to give the best they had, the American stomach demanded more than *frijoles*, rice, and *tortillas*. Occasionally there was a jar of jelly or peanut butter.

Once there was a critical shortage of drug supplies. When the truck arrived the group found, to their dismay, it carried only IBM cards and an old Billy Graham movie. However, the drug supply was aided by a Canadian doctor who landed his private plane on the small cow pastures that served as air fields, and picked up or delivered the necessary material.

Despite the problems and the "culture shock" encountered by the group, the *Amigos de Honduras* survived the primitive Honduran villages; living in small square adobe huts with dirt floors, fleas, and pigs wandering in and out. By working with the Hondurans, and giving medication, the young *Amigos* fulfilled their mission, and perhaps returned a little more mature. A final count revealed 1,156,000 inoculations were administered to 571,000 persons.

A small girl being treated for malnutrition and worms in a Honduran hospital.



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1. Riese, J. A.: Amer. J. Gastroent. 28:541 (Nov.) 1957

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Charitable Immunity—Here Today . . . Gone Tommorrow?

On Jan. 5, 1966, the Supreme Court of Texas served notice to charitable organizations operating in this state and taking advantage of the Doctrine of Charitable Immunity that such organizations may in the future be held liable for the negligent acts of their employees.

Since 1943, when the Supreme Court delivered its opinion in *Southern Methodist University vs. Clayton*, the Doctrine of Charitable Immunity as applied by the Texas Courts holds that a charitable organization is not liable for the negligent acts of its employees and agents, provided that said corporation has not been negligent in hiring or keeping the employee or agent whose negligence caused the injury.

While the Supreme Court upheld the doctrine in its opinion in the case of *Mary A. Wilkins vs. The Southcrest Baptist Church*, it may have, at the same time, sounded its death knell. The facts in this case were as follows:

Mary A. Wilkins slipped and fell on the floor of the Southcrest Baptist Church of Lubbock. She brought suit for approximately $60,000 for injuries, alleging that the Church was negligent in permitting its floors to be in a slick and dangerous condition, or in failing to remove some slippery substance from the floor. The Church filed a Motion for Summary Judgment to which was attached a copy of the corporate charter. The charter showed that the Church was incorporated for the support of public worship. It had no capital stock, and it was provided that no dividends should ever be paid. It was stipulated that the church had a liability insurance policy but the amount of the coverage and provisions of the policy were not shown. At trial, the District Court granted the defendant's Motion for Summary Judgment, relying on the Doctrine of Charitable Immunity as set forth in the *Clayton* case.

In the majority opinion, Justice Norvell, speaking for the Court, indicated that perhaps this field of charitable immunity was one in which the Legislature was the proper arm of government to change the court-adopted rule which frees charitable organizations from liability. There was no question, however, that the real reason and basis for the decision was that of past reliance by charitable organizations on the doctrine as set forth in the *Clayton* case. This was made clear by Justice Norvell:

"It would seem that Texas charities, including churches, missions, hospitals, the Salvation Army, and other similar organizations should be entitled to rely upon the decision of this Court in *Southern Methodist University vs. Clayton*, and other cases following such decision. Such institutions may have, with justification, made or refrained from making, plans in light of the holdings herein mentioned. That group involved the governing board of the Baptist Church herein involved. We therefore hold that the church was not liable for the petitioner's injuries."

In a concurring opinion, Justice Greenhill agreed with the majority but on the limited grounds that the Doctrine of Charitable Immunity should be overruled prospectively. While not stating unequivocably that he opposed the Doctrine of Charitable Immunity, Justice Greenhill left little doubt that he favors the abolition of such a rule, as stated in the following opinion:

"I am impressed with the arguments made that the Doctrine of Charitable Immunity may now be unsound in the light of current conditions, particularly as to some 'charities' which now enjoy immunity. Some 'charities' are now large business institutions which make substantial charges for their services. It is difficult, for example, to consider a person who pays $20 to $50 per day for a hospital room to be the object of charity and entitled to no protection from the negligent acts of the employees of the hospital."

Justice Walker, in his concurring opinion, stated his position:

"I concur in the judgment of affirmance in this case, but would announce now that the Doctrine of Charitable Immunity will not be recognized in cases hereafter arising."

In a strong dissenting opinion, Chief Justice Calvert, joined by Justice Smith, wrote that he favored the abolition of the Doctrine of Charitable Immunity outright and immediately.

It thus became clear that not one member of the Supreme Court was willing to argue for the Doctrine of Charitable Immunity on its merits. Prudence requires one to recognize that indications point to the abolition of the Doctrine of Charitable Immunity the next time the question comes before the Supreme Court.

Further, the decision in the *Wilkins vs. Southcrest Baptist Church* sounds in clear and unmistakable tones the warning that organizations previously protected from liability by the Doctrine of Charitable Immunity should now protect themselves with liability insurance.

The *Wilkins* case may mean that henceforth the Court will no longer follow doctrines long established in this state unless those doctrines are justified and equitable in the Texas of the 1960's.

—Philip R. Overton, LL.B.,
Austin

"I must admit, doctor, you're willing to listen to the patient's opinion."

UT Receives USPHS Grant For Air Pollution Research

A University of Texas scientist has been awarded a grant from the US Public Health Service to conduct research which could lead to a breakthrough in the growing problem of air pollution.

The grant to Dr. James R. Brock, associate professor of chemical engineering, is for a three-year program in theoretical research aimed at determining how tiny particles behave and interact in the air pollution phenomenon.

The $56,770 grant will enable Dr. Brock to help determine how Knudsen aerosols, such as smoke, are dispersed and move about in the atmosphere. A theoretical solution to such a question could lead to a practical application for eliminating pollution of the air.

Dr. Brock said graduate students at the University will assist him in the research.

Home Health Grants Awarded to 27 States

The US Public Health Service has announced that $5,104,794 has been awarded to 27 states from the Federal State Formula Grant Fund to encourage augmentation of Home Health Services in communities throughout the country. Texas is not among the recipient states.

The total $9 million grant program apportions funds among the states and territories of the US, based on a formula of the numbers of aged and the income level. The minimum award is $75,000.

Grants are distributed following approval of the state's plan to help Home Health Agencies already in existence to qualify under Medicare standards, and to develop new agencies in communities where this resource is lacking.

"Guess what the doctors have done to the heart."

Name Is Changed To *Texas Medicine*

Beginning with this issue, you will be reading *Texas Medicine* each month instead of the *Texas State Journal of Medicine.*

The journal's name was changed officially by the Board of Trustees of the Texas Medical Association at the Annual Session. The new name became effective with the May issue.

The change was made to simplify the longer name and to make it more compatible with the TMA official designation.

When Texas physicians first organized their professional group in January, 1853, the organization was called the Texas Medical Association. However, in 1869 the name was changed to the Texas State Medical Association and in 1901, to the State Medical Association of Texas. Not until 1951 was the original name, Texas Medical Association, readopted.

Now the TMA's official journal follows suit with its simpler, more accurate designation, *Texas Medicine.*

50 States Now Participating In AF Medical Rejectee Plan

With the approval of an Ohio contract, health referral and counseling programs for Armed Forces medical rejectees are now being conducted in all 50 states, Surgeon General William H. Stewart of the US Public Health Service has announced.

A total of $15 million has been awarded to the 50 states, with Ohio being the last state to enter the program.

Under terms of the contract, state agencies screen and evaluate Armed Forces Examination Station medical records of men rejected from military service for medical reasons. They counsel these young men, more than 90,000 to date, concerning their health needs and refer them to health and rehabilitation resources in their own communities for appropriate care.

Personals

Dr. Wayne E. Truax of Galveston left on April 15 for Vietnam with five other physicians, as part of Project Viet-Nam which recruits US doctors for short-term, unpaid service.

Dr. Truax and the other five doctors will work in provincial hospitals in various sections of the country, where they will minister to sick and wounded Vietnamese civilians. To date, 100 physicians have participated in the project which was initiated last summer.

Currently an associate professor in the Department of Internal Medicine at the University of Texas Medical Branch in Galveston, Dr. Truax is also affiliated with the John Sealy Hospital.

The first Gold Scalpel award of the International Cardiology Foundation was presented in New York recently to **Dr. Michael E. DeBakey** of Houston.

The foundation is the fund-raising organization of the International Society of Cardiology.

Dr. DeBakey, director of the Cardiovascular Research Center of Baylor University College of Medicine in Houston, received the award for his pioneering use of synthetic substitutes for blood vessels.

Dr. John M. Dietschy, assistant professor at the University of Texas Southwestern Medical School in Dallas, has been appointed a Markle Scholar in Academic Medicine by the John and Mary R. Markle Foundation of New York.

The Foundation's accompanying grant is the sum of $30,000, paid at the rate of $6,000 a year for five years to the medical school, to be used to supplement salary, aid research, or otherwise assist in the development of the Scholar as a teacher or investigator. The appointment of the 25 Scholars chosen this year, including Dr. Dietschy, begins July 1, 1966.

Dr. Denton A. Cooley of Baylor University College of Medicine in Houston served as class chairman for the reunion of the 1941 arts and sciences graduates of the University of Texas at Roundup activities in Austin in April. The 1941 graduates of the University celebrated their 25th reunion, and members of the 1916 class marked their 50th graduation anniversary.

Dr. Lee E. Farr of Houston served as chairman for a Conference on the Pediatric Significance of Peacetime Radioactive Fallout, sponsored by the American Academy of Pediatrics in San Diego, Calif., March 14-16.

Dr. Farr, chairman of the Academy's Committee on Environmental Hazards, is professor of nuclear and environmental medicine at the University of Texas Graduate School of Biomedical Sciences at Houston.

Dr. Mylie E. Durham, Jr., of Houston has been named general chairman of the Local Committee on Arrangements for the American Medical Association's Clinical Meeting to be held in Houston, Nov. 26-29, 1967.

Dr. Durham's nomination by the Harris County Medical Society was confirmed in April by the AMA's Board of Trustees.

Dr. Roy E. Joyner of Texas City has been named to the board of directors of the Industrial Medical Association. He will serve a three-year term on the board, according to an announcement made at the Association's Golden Anniversary meeting held in April in Detroit.

A former president of the Texas Industrial Medical Association, Dr. Joyner is plant medical director for the Chemicals Division of Union Carbide Corporation in Texas City.

Dr. Walter J. Burdette of Salt Lake City, head of the department of surgery at the University of Utah, has been appointed professor of surgery and associate director for research at the University of Texas M.D. Anderson Hospital and Tumor Institute in Houston.

Dr. William K. Wright of Houston recently was named southern regional vice president of the American Academy of Facial Plastic and Reconstructive Surgery, Inc. The election was held during the group's scientific meeting in April in San Juan, Puerto, Rico.

Dr. Wright is clinical assistant professor in otolaryngology at Baylor University College of Medicine and the University of Texas Postgraduate School. He is an attending otolaryngologist at Methodist Hospital, Hermann Hospital, St. Joseph's Hospital, St. Luke's Hospital, and Texas Children's Hospital in Houston.

Anton H. Berkman, Ph.D., professor of biological sciences at Texas Western College, will retire May 28 after a teaching career of more than 40 years.

Dr. Berkman was chairman of the Department of Biological Sciences from 1927 until he became dean of the College's Graduate School in 1958. He was known for inspiring many young persons to enter the field of medicine.

Methodists Name Head Of Social Health Group

Dr. Frank M. Bockus of Houston has been named director of the Department of Social Health, Division of Alcohol Problems and General Welfare, Methodist Board of Christian Social Concerns.

He will be responsible for developing Methodist study and action programs related to social health. Some of the current programs he will participate in are the "American Study on Healing and Salvation" and Methodism's Focus on Mental Health.

An ordained Methodist minister, Dr. Bockus is associate professor of pastoral theology at the Institute of Religion, Texas Medical Center, in Houston. He will assume his new position on June 1.

Talks on School Health To Precede AMA Meet

The eighth annual Preconvention Session on School Health will be held on Sunday, June 26, at 7:30 p.m., at the Palmer House in Chicago, preceding the American Medical Association's annual convention.

Sponsored by the AMA's Department of Health Education and the American School Health Association, the session will offer speakers from the fields of medicine, science, and education, discussing various aspects of school health programs.

Invited to attend the meeting are physicians interested in school health programs, as well as educators, nurses, dentists, and other related professionals.

Additional information may be obtained from the Department of Health Education, American Medical Association, 535 N. Dearborn St., Chicago, Ill. 60610.

Officers Announced By Related Societies

Related and specialty organizations of the Texas Medical Association elected new officers at meetings held in conjunction with the TMA's 99th Annual Session.

Among the officers who have been announced are the following:

Texas Air-Medics Association.—Dr. James M. Kent, Dallas, president; Dr. David McCullough, San Antonio, president-elect and vice president; Dr. Claire F. Miller, Waco, secretary-treasurer.

Texas Neuropsychiatric Association and Texas District Branch, American Psychiatric Association.—Dr. Alfred H. Hill, San Antonio, president; Dr. James K. Peden, Dallas, president-elect; Dr. William Pryor Shelton, Dallas, vice president; Dr. Irvin M. Cohen, Houston, secretary-treasurer.

Texas Orthopaedic Association.—Dr. Gerald S. Ahern, Corpus Christi, president; Dr. Frank F. Parrish, Houston, president-elect; Dr. Kermit W. Fox, Austin, vice president; Dr. L. Ray Lawson, Dallas, secretary-treasurer.

Texas Otolaryngological Association.—Dr. Ben T. Withers, Houston, president; Dr. Thomas W. Folbre, San Antonio, president-elect; Dr. Lloyd A. Storrs, Lubbock, vice president; Dr. Jesse D. Gomillion, Midland, treasurer; Dr. Henry E. Maddox, III, Houston, program chairman.

Texas Society of Anesthesiologists.—Dr. Ivey E. Lamberth, Tyler, president; Dr. Fred P. Thomas, Houston, president-elect; Dr. Adolph H. Giesecke, Jr., Dallas, vice president; Dr. Gerald G. Mullikin, Longview, secretary-treasurer; Dr. Leo S. M. Duflot, Galveston, Dr. Thomas, and Dr. Mullikin, delegates to the American Society of Anesthesiologists; Dr. Milner S. Thorne, Austin, Dr. Robert B. Krause, San Antonio, and Dr. Valington F. Borum, Fort Worth, alternate delegates.

Texas Chapter, Flying Physicians Association.—Dr. James A. Martin, Dallas, president; Dr. Walter A. Brooks, Quanah, vice president; Dr. Richard L. Gilkey, Clarendon, secretary-treasurer.

Texas Society of Plastic Surgeons.—Dr. Dean C. Kipp, Dallas, president; Dr. John P. Gannon, Houston, vice president; Dr. James

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Texas Association of Public Health Physicians.—Dr. John B. Bryson, Belton, president; Dr. Francine Jensen, Houston, president-elect; Dr. Charles M. Parker, Wichita Falls, vice president; Dr. Santino J. Lerro, LaMarque, secretary-treasurer; Dr. B. M. Primer, Austin, Dr. William R. Metzger, Corpus Christi, and Dr. Neal M. Atkins, Waco, board of trustees.

Texas Ophthalmological Association.—Dr. Henry L. Hilgartner, Austin, president; Dr. Harl D. Mansur, Jr., Wichita Falls, president-elect and vice president; Dr. James L. Mims, Jr., San Antonio, secretary; Dr. Joe L. Bussey, Fort Worth, treasurer; Dr. Harold E. Hunt, Paris, Dr. Rex C. House, Corpus Christi, Dr. Jerry Johnson, Beaumont, one-year councilors; Dr. Thomas L. Royce, Houston, Dr. Edwin G. Grafton, Dallas, two-year councilors; Dr. John C. Koch, Lufkin, Dr. Ennis E. Moss, Lubbock, three-year councilors.

Texas Physical Medicine and Rehabilitation Society.—Dr. Jewel S. Daughety, Dallas, president; Dr. Charles R. Peterson, Houston, vice president; Dr. Vincent J. Kitowski, Houston, secretary.

Texas Academy of General Practice.—Dr. Guy T. Denton, Jr., Dallas, president; Dr. Silas W. Grant, Hillsboro, president-elect; Dr. Sam A. Nixon, Floresville, vice president.

Texas Proctologic Society.—Dr. H. Gray Carter, Dallas, president; Dr. James M. Hampton, Houston, secretary-treasurer.

Texas Industrial Medical Association.—Dr. Robert R. Dugan, Houston, president; Dr. Lee Lloyd Pickett, Houston, president-elect; Dr. James K. Stewart, Beaumont, vice president; Dr. Charles O. Bruce, Port Neches, secretary-treasurer.

Texas Chapter, American College of Chest Physicians.—Dr. John L. Kee, Dallas, president; Dr. John P. McGovern, Houston, first vice president; Dr. George O. Hutcheson, Fort Worth, second vice president; Dr. Paul L. Richburg, Dallas, secretary-treasurer.

Texas Society of Athletic Team Physicians.—Dr. A. Ross Davis, Houston, president; Dr. Dan Spivey, Lufkin, president-elect; Dr. E. Frank Wallace, vice president; Dr. W. Cone Johnson, Temple, secretary-treasurer.

Texas Medical Students Get Grants for Foreign Study

Three junior medical students from Texas have been awarded foreign fellowships which will enable them to obtain supervised medical experience in relatively underdeveloped areas of the world for a ten-week period this summer.

The Association of American Medical Colleges has announced that Smith Kline & French Laboratories of Philadelphia has given grants to Richard S. Bransford, a junior at Johns Hopkins University School of Medicine in Baltimore; Linda-Jane Irwin, a junior in the University of Texas School of Medicine, Galveston; and Robert P. Watkins, Jr., a junior at Baylor University College of Medicine, Houston.

The three are among 35 junior and senior US medical students to receive grants this year, the seventh year that such awards have been offered.

Bransford, the son of Mr. and Mrs. Richard J. Bransford of Houston, will assist this summer at the Kijabe Mission Hospital, Kijabe, Kenya, in East Africa. Miss Irwin, who is from Honolulu, Hawaii, will work at the Vaiola Hospital, Nuku'alofa, in the Kingdom of Tonga, a British protected state east of the Fiji Islands. Mr. Watkins, a Californian, will assist in the Adebaba Yesus Clinic in Gondar, Ethiopia.

45,000 Expected To Attend AMA Meeting in Chicago

An attendance of more than 45,000 is predicted for the 115th Annual Convention of the American Medical Association, meeting at McCormick Place in Chicago, June 26-30.

This figure includes an estimated 15,000 physicians, 20,000 guests, 2,500 medical students, registered nurses, and technicians, 5,000 industrial exhibitors, and 2,500 exhibitor guests.

Exhibits, which will open on Sunday, June 26, will include 350 scientific displays and six special exhibits sponsored by the AMA Council on Postgraduate Programs.

Most of the scientific sessions will begin on Monday, June 27, and will continue through Thursday, June 30.

May Health Forum To Study Commission's Final Report

The 1966 National Health Forum, meeting at the Americana Hotel in New York City on May 9-11, is scheduled to produce the conference ground for a first public consideration of the final report of the four-year study of the National Commission on Community Health Services.

Participation in the Forum, by invitation only, will be limited to 600 discussants, who will seek ways to initiate and encourage appreciation and understanding of the Commission's recommendations.

1966 Biomedical Symposium To Cover Diagnosis, Therapy

From diagnosis to therapy will be the theme of the 1966 San Diego Biomedical Engineering Symposium, June 6-9 at the Kona Kai Club on Shelter Island, on the north shore of San Diego Bay.

Four biomedical topics will be explored in depth, one each day: Microanalytic Diagnostic Techniques (in vivo), Macroanalytic Diagnostic Techniques (in vitro), Therapeutic Effects of Extreme Environments, and Prosthetics, Artificial vs. Transplanted.

Two authorities in aerospace and undersea medicine will discuss "Manned Vehicular and Extra-Vehicular Activities in Outer Space and Inner Space," and a topical talk on "Biomedical Engineering in Vietnam" will illustrate the great strides recently achieved in combat surgery and medicine.

Registration information may be obtained from Mr. J. M. Hlavin, registration chairman, P. O. Box 2228, La Jolla, Calif. 92038.

Smoking, Health Congress To Convene in New York

The effects of smoking on health and the responsibilities of public officials and the medical profession to the public will serve as focal points for the First International Congress on Smoking and Health to be held at the New York Hilton Hotel in New York City June 5-7.

Dr. Alton Ochsner of New Orleans, president of the Congress,

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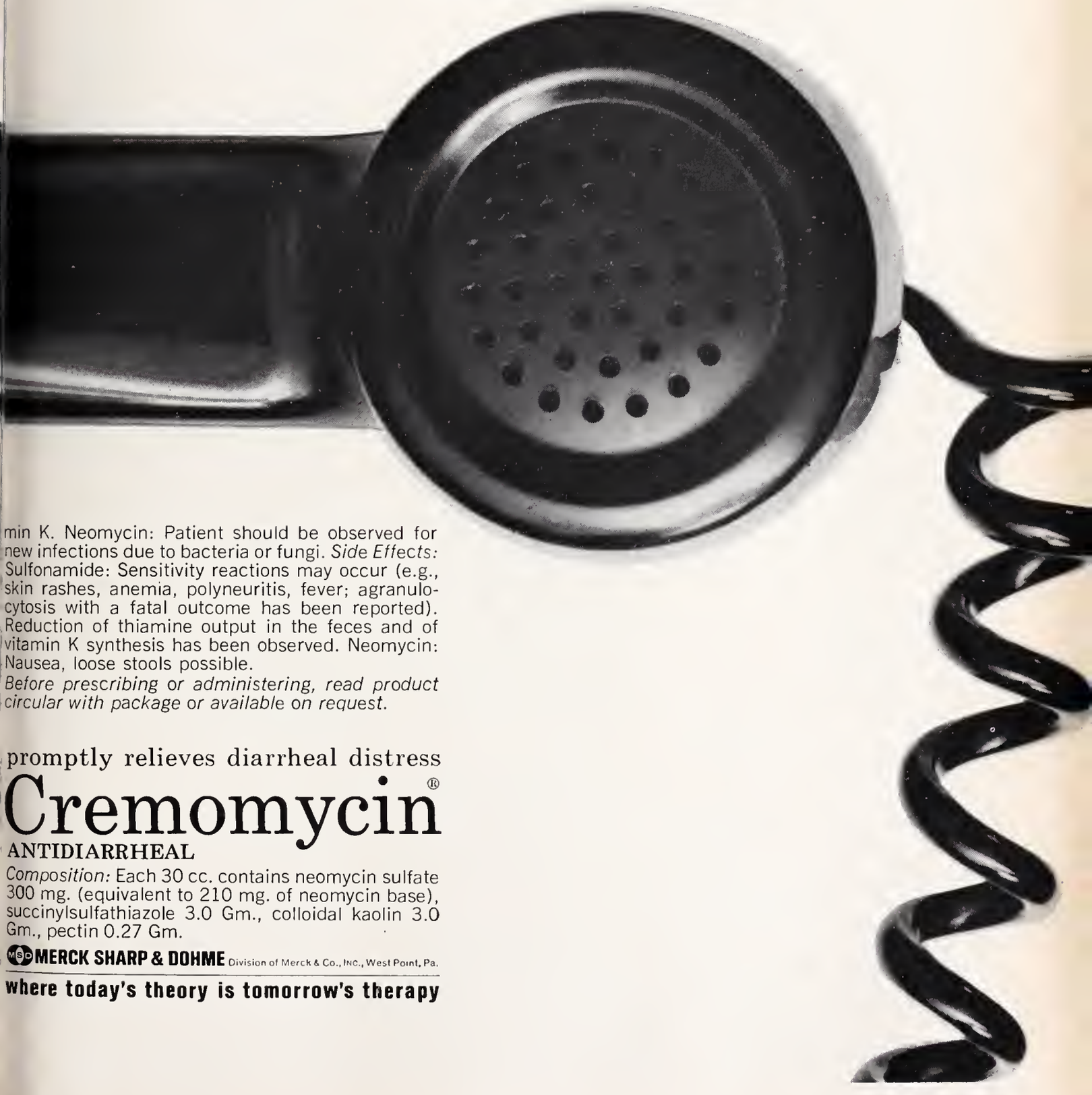

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announced that the three-day session will study ways of disseminating basic information regarding the effects of smoking on health to the public and particularly to young people.

During the meetings, internationally known authorities will present papers discussing their findings on the problem of smoking's effect on health.

The Congress, board of directors and its 75-member advisory board include representation from 25 countries. Civic leaders, government and Congressional leaders, and well known physicians are serving as leaders of the Congress.

Registration forms and information are available from the Congress office: Overseas Press Club, 54 West 40th St., New York, N. Y. 10018.

Proctologic Society Slates June Session in Cleveland

The American Proctologic Society will hold its 65th annual meeting June 20-22 in the Sheraton-Cleveland Hotel in Cleveland, Ohio.

Among the Texans participating in the sessions will be Dr. J. Arnold Bargen of Temple, who will take part in a discussion of ulcerative colitis; and Dr. John McGivney of Galveston, who will serve as chairman of a discussion course on proctologic problems.

Williston To Speak At Library Meeting

Approximately 700 members of the Medical Library Association are expected to attend the 65th annual meeting on June 6-10 at the Sheraton-Boston in Boston, Mass.

Among the speakers at the session will be C. Lincoln Williston, executive secretary of the Texas Medical Association, who will talk on "The Future Medical Society and the Library" at a gathering on June 6 of the Medical Society Libraries Group.

Also attending the meeting from Texas will be Miss Pauline Duffield, librarian of the TMA Memorial Library and chairman of the Marcia C. Noyes Awards Committee, which will present an award for the outstanding librarian of the year.

Chairman of the convention committee is Mr. Ralph T. Esterquest of the Francis A. Countway Library of Medicine at Harvard University, Boston.

Information on convention exhibits may be obtained from Mrs. Helen Brown Schmidt, executive secretary, 919 N. Michigan Ave., Chicago, Ill. 60611.

Drug Metabolism Set For Workshop Study

A Workshop on Drug Metabolism will be held May 23-27 at the Department of Pharmacology of the New York University Medical Center.

Sponsored by the Drug Research Board of the National Academy of Sciences, National Research Council and the Pharmaceutical Manufacturers Association Foundation, the workshop is designed for pharmacologists, biochemists, toxicologists, teratologists, and clinical pharmacologists who lack a previous knowledge of drug metabolism.

The workshop will consist of lectures, laboratory demonstrations, and experiments on the applications of drug metabolism to pharmacological and toxicological studies, according to Dr. Bert N. La Du, Jr., chairman of the Department of Pharmacology at NYU and director of the workshop.

In addition to workshop participants, now being selected by an admissions committee, a limited number of scientists in administrative positions will be accepted as observers. The faculty, which will include many of the nation's top experts in the field, will be announced later.

TB Association Elects Officers

Dr. Daniel E. Jenkins of Houston has been named president of the Texas Tuberculosis Association for 1966-67.

Other new officers include Dr. John H. Selby of Lubbock, president-elect; Mr. Henry Guerra of San Antonio, first vice president; Dr. E. N. Wilson, Jr., of Pearsall, second vice president; Mrs. Roy H. Bland, Jr., of Taylor, secretary; Mr. Joe K. Wells of Austin, treasurer; and Dr. Robert B. Morrison of Austin, assistant treasurer.

US-Mexico Public Health Meeting Set in Saltillo

The United States-Mexico Border Public Health Association will meet in Saltillo, Coahuila, Mexico, for its 24th annual meeting, June 6-10.

Sessions will be held in the Instituto Tecnologico Coahuila, according to Dr. Jorge Jimenez-Gandica of El Paso, secretary.

A special meeting on Thursday, June 9, will deal with venereal disease. Section meetings, beginning Tuesday, June 7, will cover the areas of preventive medicine, special public health service, environmental health, and general public health service.

Postgraduate Courses

Biomedical Engineering, Birmingham, Ala.—A 12-month postdoctoral program and a graduate program in biomedical engineering will be offered by the University of Alabama at the Center for Biomedical Engineering.

The postdoctoral program will begin in June for medical scientists with an M.D., D.M.D., D.D.S., or Ph.D. degree. The program will include a concentrated study of engineering and the mathematics of biomedical engineering.

The graduate program will be offered in the fall for selected graduate students, primarily with backgrounds in engineering, and will lead to a Ph.D. in biomedical engineering. This program will emphasize the academic disciplines of medicine, dentistry, engineering, and the life sciences, and their application to the solution of problems in medicine and dentistry.

Applications for admission to the graduate program will be accepted up to Aug. 1. Additional information may be obtained from the Center for Bio-medical Engineering, University of Alabama, 1919 Eighth Avenue South, Birmingham, Ala. 35233.

Solid-Waste Management, Cincinnati, Ohio, May 16-20.—A one-week course on elements of solid-waste management for technical and administrative personnel responsible for local, state, federal, and industry solid-waste programs

will be held at the US Public Health Service's Robert A. Taft Sanitary Engineering Center.

Course topics will include the national solid-waste problem, typical solid-waste programs, storage practices, collection methods, on-site disposal, transfer stations, mineral and fuel production wastes, waste salvage, commonly used disposal methods, sanitary landfill and incinerator design and operation, and municipal refuse composting.

Applications for the course should be addressed to the Director, Training Program, Robert A. Taft Engineering Center, 4676 Columbia Parkway, Cincinnati, Ohio 45226. No tuition or registration fee is required.

Annual Otolaryngologie Assembly, Chicago, Oct. 1-7—The 1966 assembly will be held in the new Illinois Eye and Ear Infirmary at the Medical Center. The Department of Otolaryngology of the College of Medicine of the University of Illinois will offer a condensed postgraduate basic and clinical program for practicing otolaryngologists under the direction of Dr. Emanuel M. Skolnik.

Interested physicians should write to the Department of Otolaryngology, P. O. Box 6998, Chicago, Ill. 60680.

Thirteenth Annual Anesthesiology Review Course, Lackland Air Force Base, June 6-10.—The review course will be sponsored by the Anesthesiology Service of the Wilford Hall USAF Hospital.

Requests for application forms and further information may be obtained by writing Lt. Col. Anthony J. DiGiovanni, Chief, Anesthesiology Service, Wilford Hall USAF Hospital, Aerospace Medical Division, Air Force Systems Command, Lackland Air Force Base, Texas.

New Books in TMA Library

Bettmen, H. W.: *Symposium on Vaseular Disorders of the Eye*, National Institutes of Health, Visual Sciences Study Section, St. Louis, C. V. Mosby Co., 1966.

Brazier, M., ed., UCLA Forum in Medical Science: *Brain Funetion No. 1: Cortieal Exeitability and Steady Potentials, Relations of Basie Researeh to Space Biology; Brain Funetion No. 2: RNA and Brain Function, Memory and Learning*, Los Angeles, University of California Press, 1963-1964.

Chandler, P. A., and Grant, W. M.: *Leetures on Glaucoma*, Philadelphia, Lea & Febiger, 1965.

Conn, H. F.: *Current Therapy 1966*, Philadelphia, W. B. Saunders Co., 1966.

Cornblath, M., and Schwarts, R.: *Disorders of Carbohydrate Metabolism in Infaney*, Vol. 3 in Series; Major Problems in Clinical Pediatrics, Philadelphia, W. B. Saunders Co., 1966.

Delacato, C. H.: *Diagnosis and Treatment of Speech and Reading Problems*, Springfield, Charles C Thomas, Publisher, 1963.

Kandel, D. B., and Williams, R. H.: *Psyehiatrie Rehabilitation, Some Problems of Researeh*, New York, Atherton Press, 1964.

Klemer, R. H.: *Counseling in Marital and Sexual Problems*, Baltimore, Williams and Wilkins, 1965.

Luisada, A. A., and Slodki, S. J.: *The Differential Diagnosis of Cardiovaseular Disease*, New York, Grune & Stratton, 1965.

Modell, W., ed.: *Drugs in Current Use 1966*, New York, Springer Publishing Co., Inc., 1966.

Paul, L. W., and Juhl, J. H.: *The Essentials of Roentgen Interpretation*, ed. 2, New York, Hoeber Medical Division, 1965.

Puestow, C. B.: *Surgery of the Biliary Tract, Panereas and Spleen*, ed. 3, Chicago, Yearbook, 1964.

Rather, L. J.: *Mind and Body in Eighteenth Century Medieine*, Los Angeles, University of California Press, 1965.

Rosen, E., and Gregory, I.: *Abnormal Psyehology*, Philadelphia, W. B. Saunders Co., 1965.

Selzer, A.: *The Heart: Its Funetion in Health and Disease*, Los Angeles, University of California Press, 1966.

Shirkey, H. C.: *Pediatrie Therapy*, ed. 2, St. Louis, C. V. Mosby Co., 1966.

Trease, G. E.: *Pharmaey in History*, London, Bailliere, Tindall and Cox, 1964.

Welch, C. E., ed.: *Advanees in Surgery*, Vol. 1, Chicago, Year Book Medical Publishers, 1965.

Williams, M.: *Mental Testing in Clinieal Practice*, New York, Pergamon Press, Inc., 1965.

Wilson, J. W., and Plunkett, O. A.: *The Fungous Diseases of Man*, Los Angeles, University of California Press, 1965.

Wolberg, L. R.: *Short-Term Psychotherapy*, New York, Grune & Stratton, 1965.

Book Notes

Gonadotropins: Physicochemical and Immunological Properties. Ciba Foundation Study Group No. 22, edited by G.E.W. Wolstenholme and Julie Knight. 125 pp., illus., Boston, Little, Brown and Co., 1965. $3.50.

THIS STUDY group was held in honor of Dr. Christian Hamburger, who has devoted the past 35 years, his whole scientific life, to this problem and who gave the historical introduction on gonadotropic hormones.

The papers presented are as much reviews to stimulate others to discuss their results as presentations of original material.

Foundations of Anesthesiology. Albert Faulconer, Jr., and Thomas E. Keys. 2 vols., 1337 pp., illus., Springfield, Charles C Thomas, 1965. $38.50.

FAULCONER AND KEYS have given us a book which can be considered a refinement and sequel to Keys' classical "History of Surgical Anesthesia." The book is a collection of some of the original works that have been done in anesthesia and is a fine work, well received and easily read. It is a foundation as well as a background for anesthesiology. It tells, in fact, how anesthesiology came into being.

The book is in two volumes with eight typical sections including respiratory physiology, inhalation, conduction, intravenous anesthesia, accessory agencies, and techniques. Each section has an introduction and a biography of each of the original contributors, followed by his classical work, complete and unabridged and in chronological order for that particular section. Vesalius, Wren, Davy, Long, Guedel, Leake, Lundy, and Waters, to mention a few, are included in the 170 contributors. There are a good number of diagrams, graphs, and photographs but no portraits. An index of personal names and subjects is included for reference.

These volumes should be available for medical students as supplementary reading and as required reading for anesthesiology residents. The practicing anesthesiologist will find them a pleasant way to fill out a picture of his specialty.

—ROBERT B. KRAUSE, M.D., San Antonio

Leukemia. W. Dameshek and F. Gunz. 2nd ed. 420 pp. New York, Grune & Stratton, 1964. $25.00.

THIS BOOK presents extensive information on various forms of leukemia. It deals not only with the clinical picture but presents many other interesting aspects. The first chapter describing some of the original clinical concepts concerning leukemia is interesting reading and worth the time of any medical history student. The chapter on etiology is an up-to-date, precise presentation of the accumulated knowledge concerning possible etiologic factors in the development of leukemia. This chapter alone is a worthwhile review for the practicing internist or hematologist. The sections devoted to pathological changes, clinical course, prognosis, and treatment of leukemia are excellent reviews. The concepts presented in the section on myeloproliferative and lymphoproliferative disorders, particularly the latter section, are extremely interesting and thought-provoking.

In summary, the authors present extensive up-to-date information concerning a group of disorders that too many clinicians dismiss as hopeless. This book is an excellent reference for the practicing internist or hematologist.

—PHIL DAVIS, M.D., Houston

Physiology of the Eye, Clinical Application. Francis Heed Adler, 4th ed. 889 pp. St. Louis, C. V. Mosby Co., 1965. $18.75.

TO ANY ophthalmologist who remembers the first edition published in the early 30's, the new edition is impressive. When one recalls the original edition, he realizes how much progress has been made during the last 30 years on this subject. Much of the material in this book is old and basic and remains unchanged, however, much of it is new and represents research done in the early 1950's which has clinical application.

Dr. Adler has drawn on much of the world literature and the references are well selected and well chosen. He has reduced to a minimum the basic material, and what is left has stayed close to its clinical application. For those who practice a subspecialty or who are interested in a subspecialty such as motility and glaucoma this book contains material of the first order.

—HAROLD M. BLOCK, M.D., Dallas

Ophthalmology, Principles and Concepts. Frank W. Newell, 491 pp. St. Louis, C. V. Mosby Co., 1965. $12.25.

THIS BOOK is written in the clear wording frequently seen in British ophthalmic literature. It covers the basic principles and concepts of modern ophthalmology, without excessive discussion of fine details of subject matter. In spite of the relative compactness of the volume the subject is covered in an excellent and refreshing manner with the wise use of a minimum of words. The illustrations are new and up-to-date. The text certainly is not a rehash.

Excellent reference lists are found at the end of each chapter for those who wish to explore a topic further. While the text is a bit limited for the specialist in ophthalmology, it would still make an excellent quick reference book to have on his desk. The book is an excellent review, as well as a reference, for those outside the field of ophthalmology. I would particularly recommend it to those in general practice who want a good basic reference on ophthalmology which is up-to-date and free from unnecessary words.

—J. L. MIMS, JR., M.D., San Antonio

Trauma to the Liver. G. F. Madding and Paul A. Kennedy, Vol. 3 in the series, *Major Problems in Clinical Surgery.* 134 pp. Philadelphia, W. B. Saunders Co., 1965. $6.00.

AS IN THEIR previous work on this subject, the authors provide a thorough, lucid, and completely modern presentation of the subject. Their personal experience as well as an excellent presentation of the significant available literature makes the book one of the most outstanding and best expressed texts in this field. The presentation of the anatomy of the liver, its variations, and their relevance to the surgery (elective as well as emergency) is helpful. The chapter on diagnosis and management should be perused by anyone responsible for the care of the injured. This chapter includes material so recent that all surgeons would benefit from its study. In the technical sections all known principles of care are carefully reviewed and clearly presented, and individual observations are documented. Complications, their diagnosis, and therapy are well presented.

In summary, this is an excellent presentation of material not well-collected elsewhere and is a worthwhile library addition as well as a source of new diagnostic and possibly therapeutic technique.

—EDWARD L. BUSCH, M.D., Fort Worth

Diseases of Medical Progress. Robert H. Moser, ed., 2nd. ed., 543 pp., Springfield, Charles C Thomas, 1964. $19.75.

THE SECOND edition of *Diseases of Medical Progress* is completely revised and expanded, with a new chapter on Psycho-semantics. Dr. Moser attempts to express the potentialities of a physician-patient relationship. This edition is a ready reference for the physician who wants to use a drug he has not employed previously. Attempts have been made to keep the book simple, readable, and interesting to all physicians.

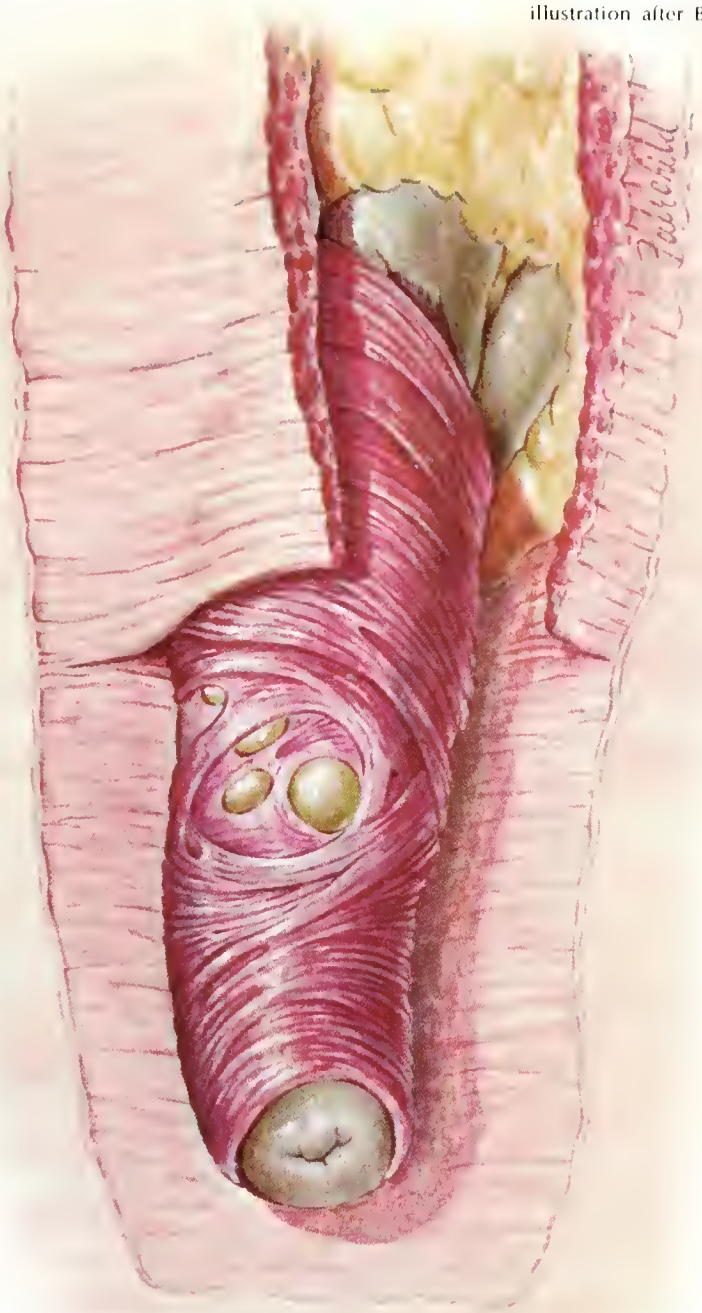
Medical Laboratory Technology. Matthew J. Lynch, 735 pp. Philadelphia, W. B. Saunders Co. 1963. $12.00.

THE AUTHORS of *Medical Laboratory Technology* have combined timely facts and theories with practice. Their method of presentation is easily read without sacrifice of the scientific material.

The chapter on blood coagulation is especially good. This subject can be confusing; however, as presented in this text, it is clear.

Eleven chapters are devoted to histologic technique. Students of medical technology will find these are an excellent review for the registry examination since all material is covered and well ex-

illustration after Boyden



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*Antacid and dietary measures are of primary importance in ulcer treatment and should not be neglected.

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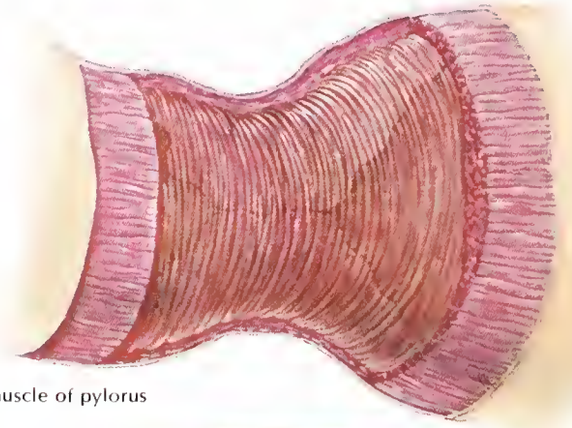
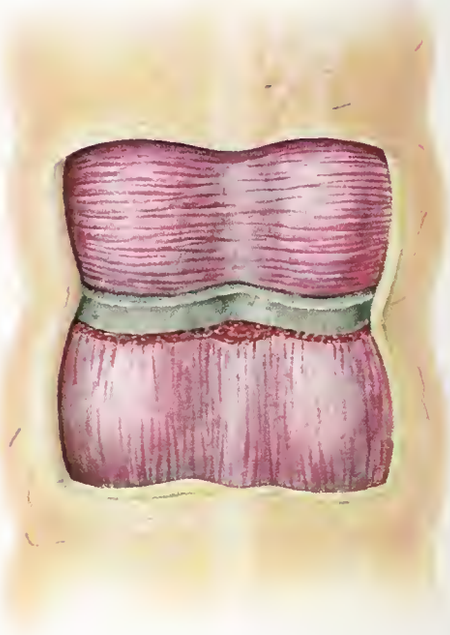
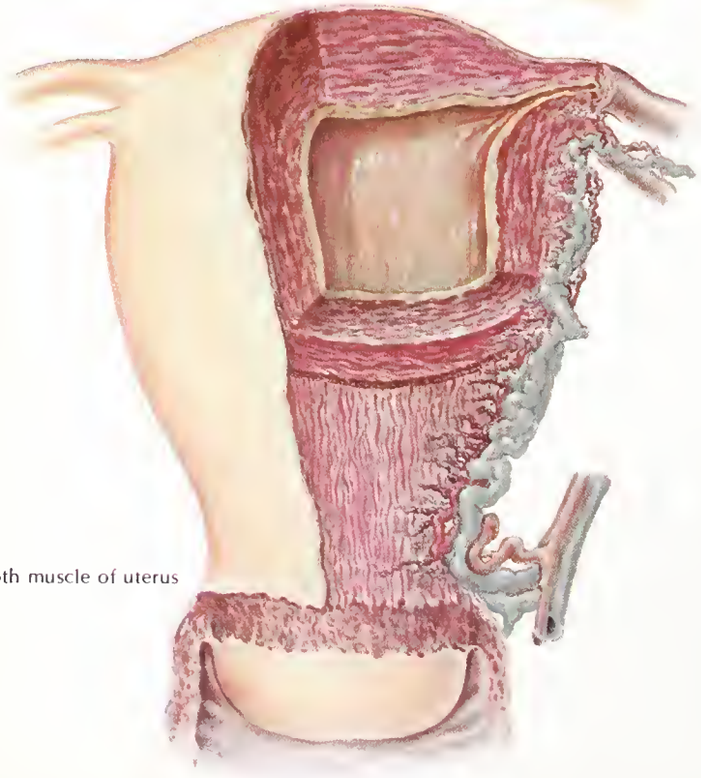


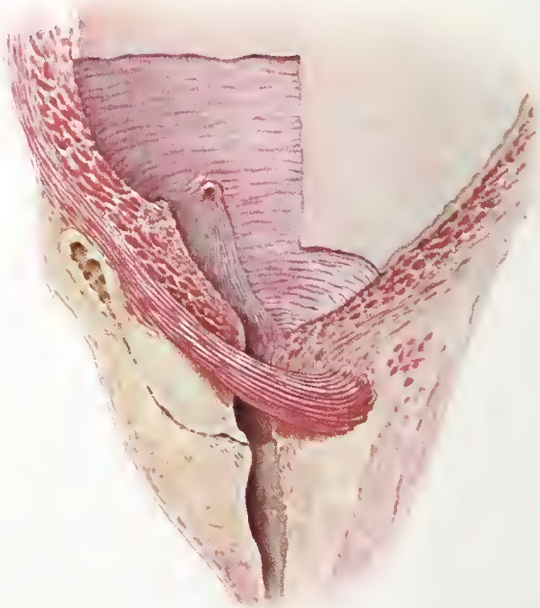
smooth muscle of pylorus



smooth muscle of colon



smooth muscle of uterus



smooth muscle of urinary bladder

plained. Moreover, the staining techniques are easy to follow.

I strongly recommend this book as a reference in every laboratory and possibly as a text for students of medical technology.

—MARTHA MADSEN, M.D., Midland

Hashish: Its Chemistry and Pharmacology. Ciba Foundation Study Group No. 21, edited by G.E.W. Wolstenholme and Julie Knight. 92 pp., Boston, Little, Brown and Co., 1965. $2.95.

PARTICIPATING IN this study are many of the world's leading authorities in pharmacology, psychiatry, and biological sciences. The study presents a good pharmacological description of hashish, with a long section on chemical analysis of the drug, done in an attempt to isolate the active ingredients.

The habituating properties of hashish are discussed, as well as factors leading to the habitual use of the drug. There is greater incidence of use of this drug in Eastern countries, especially tropical parts, possibly due to easier availability and cheapness of the drug.

An interesting discussion of the problem by the experts who met in this symposium is included. This book will be of benefit to anyone who wants a thorough knowledge of hashish. It is fairly easy to read and is complete.

—STUART S. NEMIR, JR., M.D., Austin

Pediatric Therapy. 2nd ed., Harry C. Shirkey. 1223 pp., illus., St. Louis, C. V. Mosby Co., 1966. $18.50.

THIS REFERENCE volume is written by 84 authoritative clinicians in their specific fields. The book is well organized and easy to read. Up-to-date information is given in a concise manner and is adequately illustrated. Each section is followed by generally good reference material.

This second edition has several new sections to add to its completeness. Of practical value are the new sections on pediatric orthopedics and antibiotic therapy.

This book should make an excellent reference volume for both pediatrician and generalist.

—K. DYO, M.D., Austin

Synopsis of Cardiology. William I. Gefter, Bernard H. Pastor, and Ralph M. Myerson. 877 pp., illus., St. Louis, C. V. Mosby Co., 1965. $9.85.

ONE OF MOSBY'S Synopsis Series, this work is well done and as thorough as a treatise of this size can be. In format and content it is very similar to Herrmann's Synopsis of the Heart and Arteries, also by Mosby, with the addition of advances which have occurred in cardiology in the past few years.

This text will have particular attraction to medical students or other students of cardiology who desire a book to read through as a review or to use as a quick and concise reference when seeking information on a particular syndrome.

—THOMAS M. RUNGE, M.D., Austin

Surgery of the Foot. 2nd ed., Henry L. DuVries, 586 pp., illus., St. Louis, C. V. Mosby Co., 1965. $17.50. In collaboration with members of the faculty of the University of California School of Medicine, San Francisco.

THE FIRST EDITION of this book is replete with instructions regarding the surgical care of foot disabilities. The second edition contains the same information but has been amplified so that it is difficult to understand to what audience this book is directed. The original volume was written at the request of Dr. DuVries' "students and colleagues" at a time when he was chairman of the Department of Surgery of the Illinois College of Chiropody and Foot Surgery. The present edition has the same information including sections varying from a "1-2-3" type of instruction for removing a small exostosis to "1-2-3" instructions for such operations as a Brockmann procedure. The content varies from elemental surgical principles to descriptions of the most advanced type of foot reconstruction. Singularly there is no emphasis on possible complications.

The format is excellent with clear illustrations and diagrams. One diagram which appears twice, showing a pull-out wire in the anterior tibial tendon, is incorrect.

It is surprising to find such a limited discussion of gout in a book on surgery of the foot.

The section on the anatomy of the foot and electromyography of foot muscles during ambulation, running, etc., is informative. Numerous references to the English literature make this a source book. It is an addition to the library of those doing surgery of the foot as well as a reference for those who encounter only occasional problems in foot care.

—MORTON H. LEONARD, M.D., El Paso

Recent Deaths

DR. V. V. FRIEND

Dr. Victor Vernon Friend, 61, died in Houston March 28, 1966. A practicing surgeon in Houston for 27 years, Dr. Friend also was in general practice there until 1953.

Born July 23, 1904, in Houston, Dr. Friend's parents were John and Myrtle Boyer Friend.

He attended Austin High School and the University of Texas, both in Austin, and received his medical degree in 1937 from Chicago Medical School. After serving his internship at West Side Hospital in Chicago, he served residencies at West Side and at the Wright Clinic and Hospital in Houston.

Dr. Friend also did University of Texas postgraduate work in Houston in 1952-1953.

His medical organizations included the American Medical Association, Texas Medical Association, Harris County Medical Society, Southern Medical Association, Postgraduate Medical Assembly of South Texas, International College of Surgeons, and the American Society of Abdominal Surgeons.

Dr. Friend was a member of the Baptist church and the AF& AM. A 32nd degree Scottish Rite Mason, he also belonged to the Shriner Arabia Temple.

His hobbies included gardening, reading, and coin collecting.

In 1936 Dr. Friend married Beatrice Bates of Altus, Okla. She

DR. FRIEND

survives as do a son, Victor Vernon Friend, Jr., of Houston, and a brother, Stafford Friend of Denver, Colo.

DR. W. H. HATFIELD

Dr. Walter Heron Hatfield, 77, a physician in McAllen since 1933, died March 7, 1966, after an illness of several months.

Dr. Hatfield was city physician of McAllen and a board member of the Hidalgo County Crippled Children's Clinic.

Born Aug. 26, 1888, in Covington, Ky., he was the son of Dr. Walter S. and Elizabeth Heron Hatfield. He attended school in Covington and the Ohio Northern University in Ada, Ohio, and received his medical degree from Hahnemann Medical College, Philadelphia, in 1911. His internship was served at J. Lewis Crozer Home for Incurables and Homeopathic Hospital in Chester, Pa. He also did further work at Hahnemann Hospital, Philadelphia, and at a hospital in New York City.

Before opening his practice in McAllen, Dr. Hatfield was a general practitioner in Cincinnati, Ohio.

During World War I Dr. Hatfield served in the US Army Medical Corps with the rank of first lieutenant. Five of his sons served in the armed forces in World War II. One son was a crew member of an Air Force transport plane which went down over the South Pacific in 1958.

The boys' mother, Luetta Crawley Hatfield, died in 1957.

A member of several veterans' organizations, including the American Legion and Veterans of World War I, Dr. Hatfield also belonged to the Rotary Club, Scottish Rite Masons, Elks, and two Shrine groups.

His medical organizations included the Texas Medical Association, American Medical Association, Hidalgo-Starr Counties Medical Society, Texas Academy of General Practice, and Hahnemann Medical College Alumni Association.

DR. HATFIELD

In the April issue of *Texas Medicine* the photographs of Dr. Francis C. Luckett (left) and Dr. Walter B. West inadvertently were transposed during the printing operations of the journal. Dr. Luckett died Nov. 30, 1965, in a Luling hospital. He had practiced medicine for 48 years in Fentress. Dr. West, pictured above on the right, died of an apparent heart attack in a Fort Worth hospital on Feb. 15, 1966. He had practiced in Fort Worth since 1932.

Dr. Hatfield is survived by his wife, Mrs. Gladys Hatfield of McAllen; five sons, Walter Heron Hatfield, Jr., of San Juan, Arthur Crawley Hatfield of Guymon, Okla., James E. Hatfield of McAllen, Major (retired) Robert Aston Hatfield of Okinawa, and Taylor Curtis Hatfield of Keyes, Okla.; his daughter-in-law, Mrs. Elizabeth Norton Hatfield of Vacaville, Calif., whose husband, Edwin Corey Hatfield, was killed; one stepson, Robert F. Barnes of McAllen; 15 grandchildren and five great grandchildren.

DR. W. C. DURINGER

Dr. William Commodore Duringer, 83, a colorful pioneer physician of Fort Worth, died March 12, 1966, in Fort Worth.

Born on a farm near Burleson, Tex., on Nov. 12, 1882, he attended school at Crowley and was graduated magna cum laude in 1907 from the Fort Worth School of Medicine, Medical Department of Fort Worth University (now part of Baylor University College of Medicine). For several years after his graduation, he was professor of clinical surgery at the college.

Retired from active practice since 1948, Dr. Duringer was one of the oldest members of the Tarrant County Medical Society. He was an honorary member of the Texas Medical Association.

Dr. Duringer, one of the youngest men ever to be admitted to the American College of Surgeons, practiced medicine with his uncle, the late Dr. W. A. Duringer, and later with his son-in-law, the late Dr. J. H. Brown. Until his retirement from private practice, he was also general surgeon for the Rock Island Railroad and division surgeon for the Southern Pacific Lines.

As a captain in the US Army Medical Corps during World War I, Dr. Duringer developed many surgical tools using concepts still employed today in plastic surgery. During World War II he worked long hours in his practice, because of the critical doctor shortage, treating as many as 100 patients daily. In addition to his rigorous private practice, he served on the procurement and assignment committee in drafting Tarrant County doctors for war service. Long hours of work without rest left an irreparable mark on his health, and he found it necessary to retire a few years after the end of the war.

A 50-year Mason and Shriner, Dr. Duringer published his autobiography, "A Pioneer Doctor's Story," in 1964.

Dr. Duringer's wife, Lillian, died in 1950. His survivors include two daughters, Mrs. E. P. Haltom and Mrs. Peggy Floore; a sister, Mrs. Roscoe Smith, and three grandchildren, Heard Floore, Jr., Susan Floore, and Dorothy Duringer Haltom, all of Fort Worth.

DR. A. L. MITCHELL

Dr. Archie Lane Mitchell, 72, died Feb. 20, 1966, in Houston of auricular fibrillation. An outstanding exponent of the advancement of pediatrics, Dr. Mitchell practiced pediatrics in Houston from 1927 until his retirement in 1965, having previously practiced in Galveston during 1926-1927.

Born Sept. 18, 1893, in Marshall, Tex., he was the son of Archie and Elizabeth Cock Mitchell. After attending Marshall High School and the University of Texas, he received his medical degree from the University of Texas Medical Branch in Galveston in 1924.

He served his internship at John Sealy Hospital in Galveston and his residencies at St. Louis Children's Hospital, St. Louis, Mo., and New Haven Children's Hospital, New Haven, Conn.

During his practicing years in Houston, Dr. Mitchell served on the staffs of Texas Children's, St. Luke's Episcopal, Hermann, St.

DR. MITCHELL

Joseph's, and Memorial Baptist Hospitals.

A diplomate of the American Board of Pediatrics, he was a member of the Harris County Medical Society, Texas Medical Association, American Medical Association, and American Academy of Pediatrics. He was also a member of the Masonic Lodge in Marshall, Tex.

During World War I, Dr. Mitchell served as a first lieutenant, and in World War II, as a member of the US Navy, he held the rank of commander.

In 1949, he married Bennie Jordan Parks of Leesville, La. Beside Mrs. Mitchell, he is survived by a daughter, Mrs. Susan Mitchell Eeds of Houston.

DR. W. R. COOKE

Dr. Willard Richardson Cooke, a retired professor emeritus of obstetrics and gynecology at the University of Texas Medical Branch in Galveston, died March 16, 1966, in an Austin hospital following a lengthy illness. He was 77.

Prominently known in medical circles throughout the nation, Dr. Cooke had served on the UT Medical Branch faculty for more than 47 years. He received his medical degree there in 1912 and joined the obstetrics-gynecology staff the following year, becoming chairman and professor in 1924.

Son of the late Dr. Henry P. and Caroline Richardson Cooke, Dr. Cooke was the seventh generation physician in his family. His father also was a professor at the UT Medical Branch and was one of the first deans of medicine there.

Dr. Cooke was born at Galveston on Sept. 6, 1888, and since retirement in 1959 had resided at Austin.

Author of a well-known textbook on the diseases of women, "Essentials of Gynecology," Dr. Cooke served as president of the American Association of Obstetricians and Gynecologists in 1944.

DR. COOKE

He also served as president of the Galveston County Medical Society, Eighth District Society, the Texas Association of Obstetricians and Gynecologists, and as vice president of the American Gynecological Society. He was an emeritus member of the Texas Medical Association. A diplomate of the American Board of Obstetrics and Gynecology, he was a member of the Central Association of Obstetrics and Gynecology.

A member of the Episcopal church, he belonged to the Galveston Boat Club, Sigma Chi, Phi Rho Sigma, and Alpha Omega Alpha.

Dr. Cooke once estimated that he delivered more than 5,000 babies and taught some 3,000 doctors their skills during a career largely devoted to teaching. His ex-residents founded in 1947 the Willard R. Cooke Club, which meets annually in his honor.

Dr. Cooke is survived by two sons, Henry Austin Cooke of Hollywood, Calif., and John Roger Cooke of Austin. His wife, the former Aline Ruth Austin of Galveston, died in 1955.

DR. C. A. HOEFER

Dr. Carl Archibald Hoefer, 70, who practiced ophthalmology in Dallas, 1941-1957, died on March 11, 1966, in Los Angeles, Calif.

Dr. Hoefer was born Sept. 29, 1895, in Friend, Neb., where he attended school. He received both bachelor of arts and bachelor of science degrees from the University of Nebraska in Lincoln, and received his medical degree from the University of Nebraska College of Medicine, Omaha, in 1924.

His internship was served at the Nebraska Methodist Hospital in Omaha, and following postgraduate work at the University of Pennsylvania Graduate School, Philadelphia, he served his residency at Wills Eye Hospital, Philadelphia, 1938-1940.

A diplomate of the American Board of Ophthalmology, Dr. Hoefer was a member of the Dallas

DR. HOEFER

County Medical Society, Texas Medical Association, and American Medical Association. He also belonged to the Catholic church.

Before opening his ophthalmology practice in Dallas, Dr. Hoefer maintained a general practice in Nebraska.

During World War I he served in the US Navy as a bandmaster on two battleships.

Dr. Hoefer married Beulah Martin of Omaha in 1923. Mrs. Hoefer, who now resides in Pasadena, Calif., survives, as do a sister, Mrs. Loretta Tochterman of Pasadena, and a brother, Harold F. Hoefer, M.D., of Hollywood, Calif.

DR. J. L. CLARK

Dr. Joseph Lawrence Clark, 56, died March 16, 1966, at his home in Ennis. His wife, the former Ann Voyner of St. Louis, Mo., who was critically ill at the time of his death, died the following day.

Born in Ennis, Jan. 27, 1910, to Dr. Lawrence Edward and Mary Bell Olson Clark, he attended schools in Ennis and received a bachelor of science degree from Southern Methodist University in Dallas in 1932, where he was a member of Phi Beta Kappa.

Dr. Clark received his medical degree from Washington University School of Medicine, St. Louis, Mo., in 1936, and served his internship at St. Paul's Hospital in Dallas.

A general practitioner in Ennis since 1937, Dr. Clark was a member of the Ellis County Medical Society, Texas Medical Association, Southern Medical Association, American Medical Association, and American Academy of General Practice. He also belonged to the Masons and the Methodist church.

His research activity was concerned with psychosomatic influences in hypertension.

Dr. and Mrs. Clark had been married since July 10, 1937.

Surviving Dr. Clark is a brother, Fred Clark, of Ennis.

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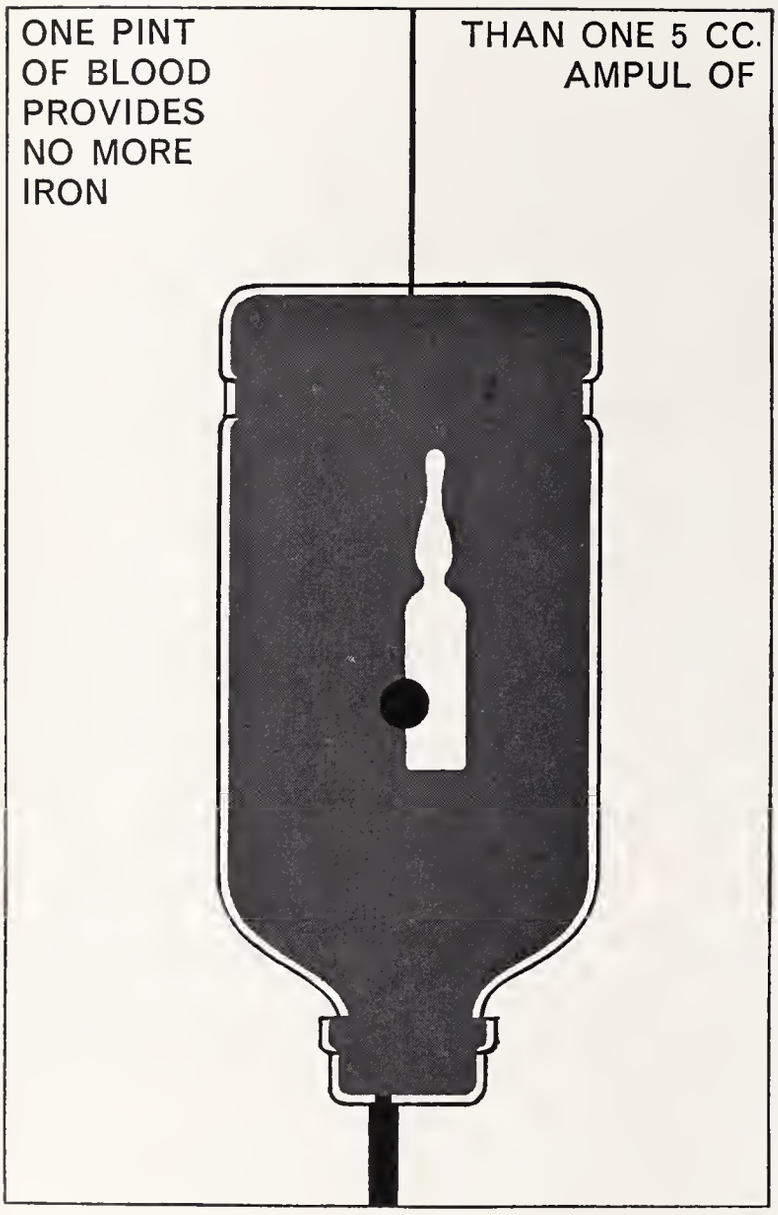

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he initial response is usually maintained vithout dosage increases; indeed, initial losage is often reduced for maintenance urposes.

alicylate or steroid therapy can usually be liminished or, in some instances, eliminated.

ontraindications
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COMING MEETINGS

American Medical Association, Annual Meeting, Chicago, June 26-30, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Austin, Public Relations Conference, Oct. 1, 1966; Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

MAY

American Association for Thoracic Surgery, Vancouver, B. C., May 16-18, 1966. Dr. H. T. Bahnson, Presbyterian University Hospital, Pittsburgh, Pa., Sec.

American College of Obstetricians and Gynecologists, Chicago, May 2-5, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American Gastroenterological Association, Chicago, May 26-28, 1966. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C., Sec.

American Thoracic Society, San Francisco, May 23-25, 1966. F. W. Webster, 1790 Broadway, New York 19, Exec. Sec.

Gastrointestinal Research Forum, Chicago, May 26, 1966. Dr. Frank Brooks, University of Pennsylvania Medical School, Philadelphia, Pa., Sec.

International College of Surgeons, Houston, May 1-5, 1966. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Texas Hospital Association, Dallas, May 14-18, 1966. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

JUNE

American Gynecological Society, Hot Springs, Va., June 2-4, 1966. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Neurological Association, Washington, D. C., June 13-15, 1966. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Cleveland, Ohio, June 20-23, 1966. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

United States-Mexico Border Public Health Association, Saltillo, Coahuila, Mexico, June 6-10, 1966. Dr. Jorge Jiménez-Gandíca, 501 U. S. Court House, El Paso, Sec.

Texas Division, American Cancer Society, Houston, June 7-8, 1966. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966, Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 8-10, 1966. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill., Sec.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. G. Whittlesey, 219 E. 42nd St., New York. Sec.

American College of Allergists. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Gastroenterology, Philadelphia, Oct. 23-26, 1966. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, Dr. Robert A. Peterman, 3 Craig Court, Totowa Borough, N. J., Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, February, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American College of Surgeons, Annual meeting, San Francisco, Oct. 10-14, 1966. Dr. Samuel P. Harbison, 230 Lothrop St., Pittsburgh, Sec.

American Congress of Physical Medicine and Rehabilitation, San Francisco, Aug. 28-Sept. 2, 1966. Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland. Mayo Clinic, Rochester, Minn., Sec.

American Heart Association, New York City, Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Hospital Association, Chicago, Aug. 29-Sept. 1, 1966. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Medical Women's Association, Rochester, N. Y., July 7-9, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Philadelphia, Oct. 1-5, 1966. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Washington, D.C., Sept. 16-22, 1966. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis, Ind., Sec.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Group, Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Private Clinics and Hospitals, Dallas, Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La., Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Mrs. R. F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston, Pres.

Southwestern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland Ave., Birmingham, Ala., Exec. Dir.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec., 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore, Md.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building. Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of General Practice, Houston, Sept. 12-14, 1966. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 11, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Heart Association, Houston, Sept. 16-18, 1966. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Fort Worth, Sept. 29-Oct. 1, 1966. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas State Division, International College of Surgeons, Dr. D. H. Smith, 1501-A N. De Leon, Victoria, Sec.

Texas Surgical Society, Houston, Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Spring, 1966. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

Blackford Memorial Lectures, Denison, Sept., 1966, Dr. Fred W. Shelton, 600 N. Highland Ave., Sherman, Chm.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 18-20, 1966. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Southwest Regional Cancer Conference, Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

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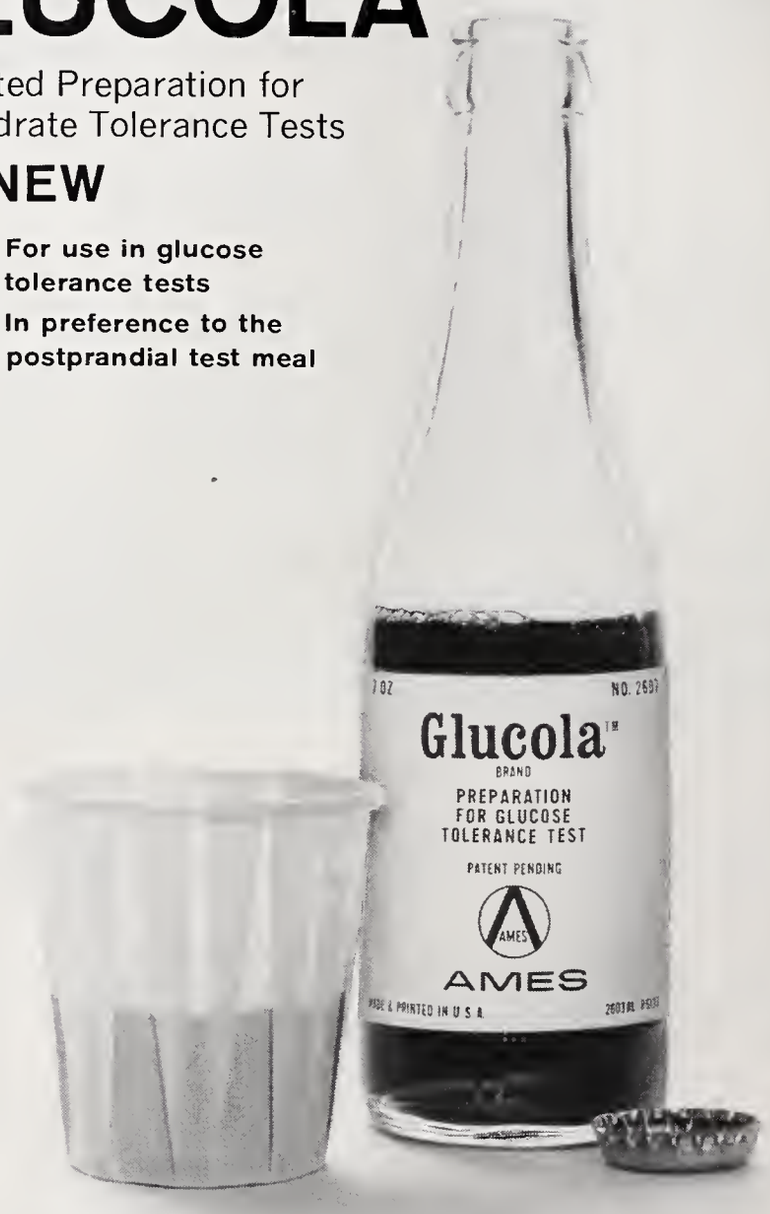

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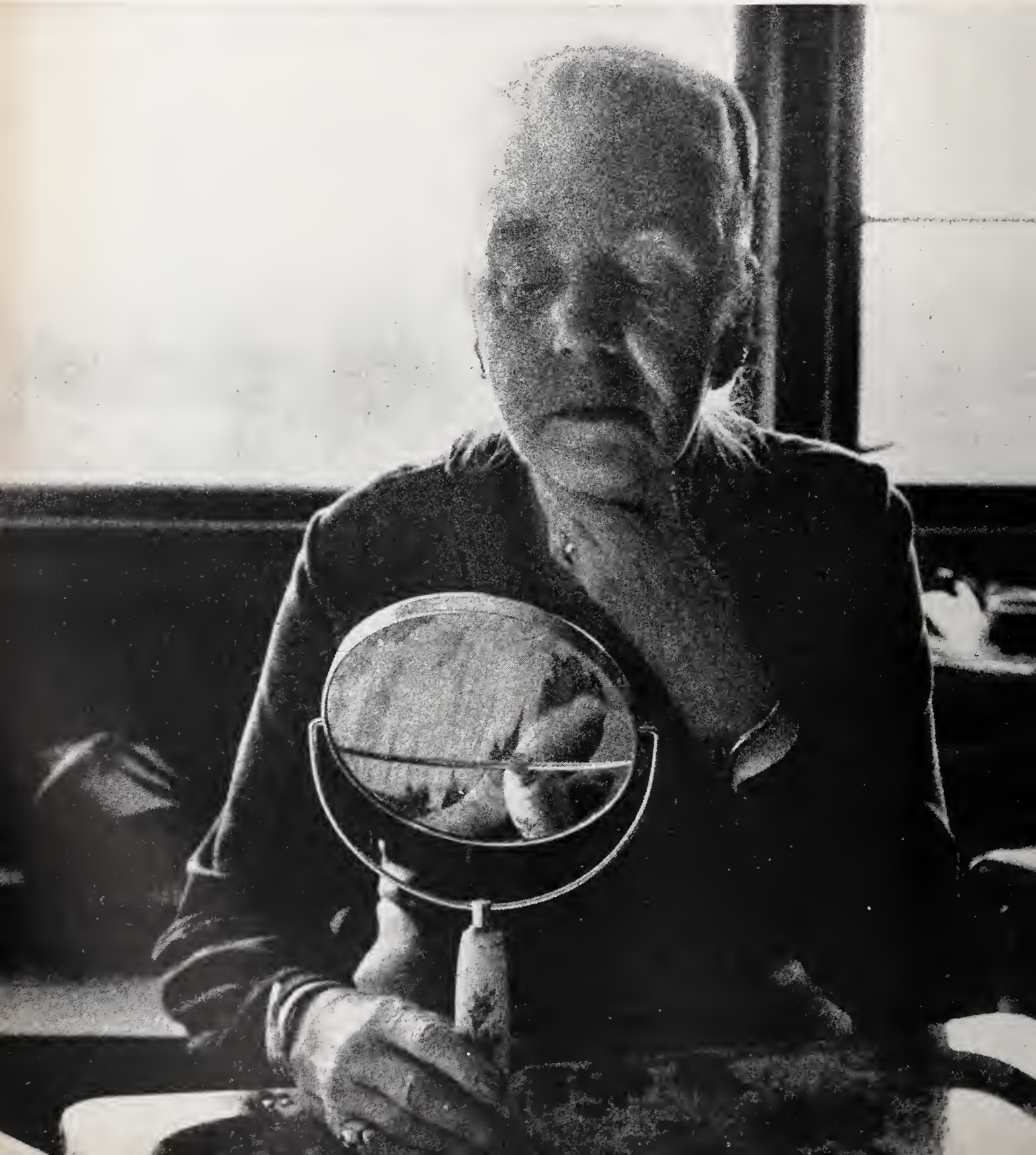

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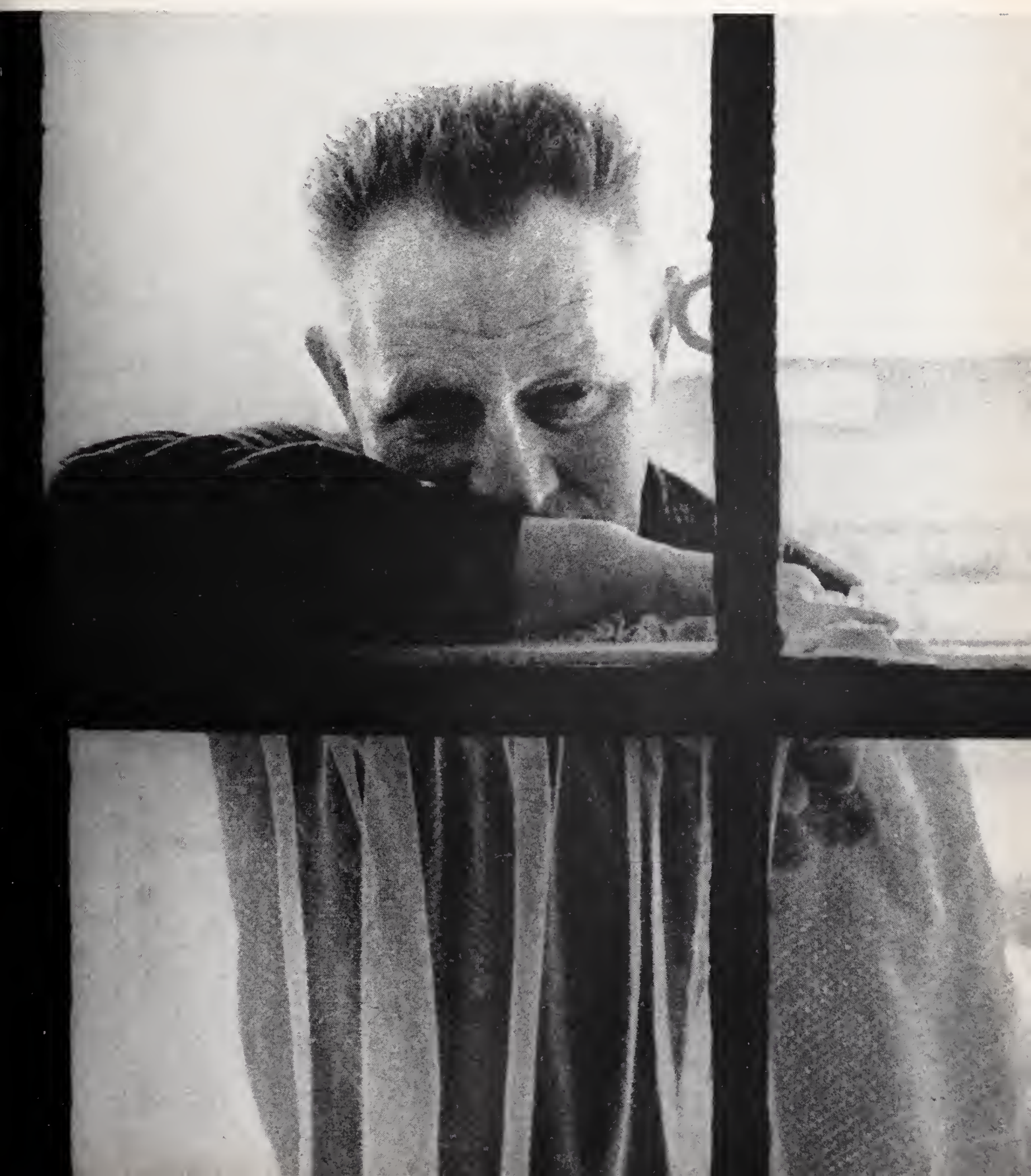

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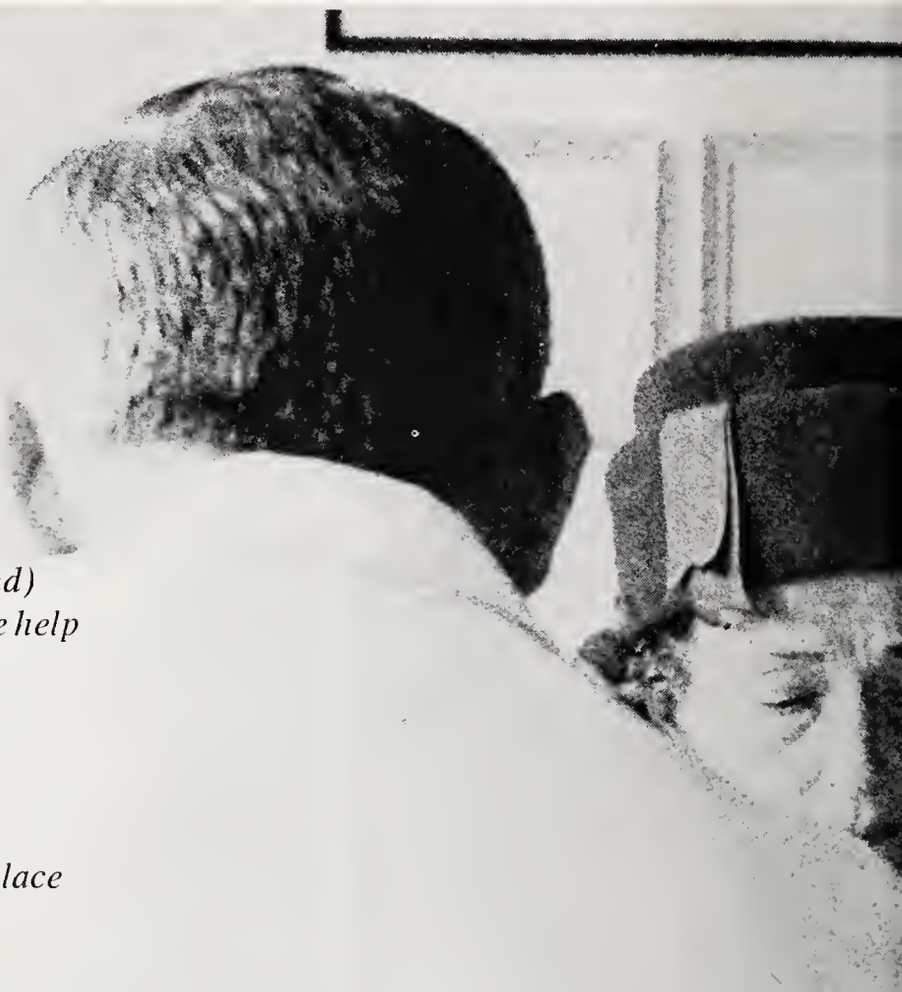

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McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

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Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

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Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

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Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

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Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.

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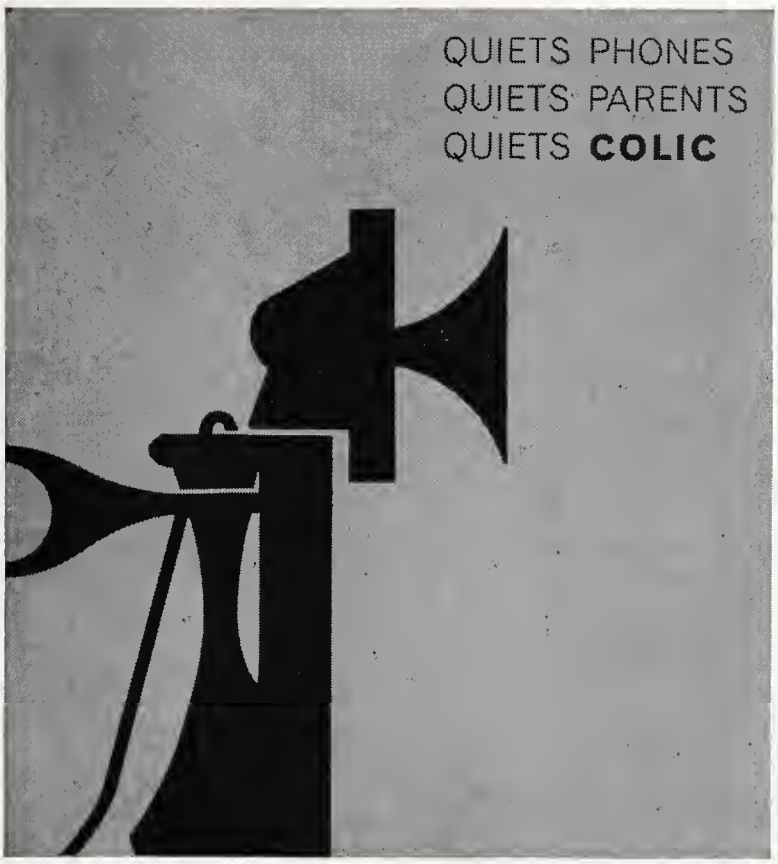

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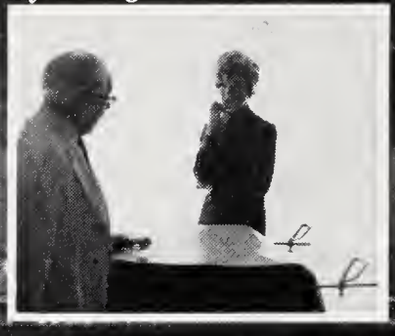

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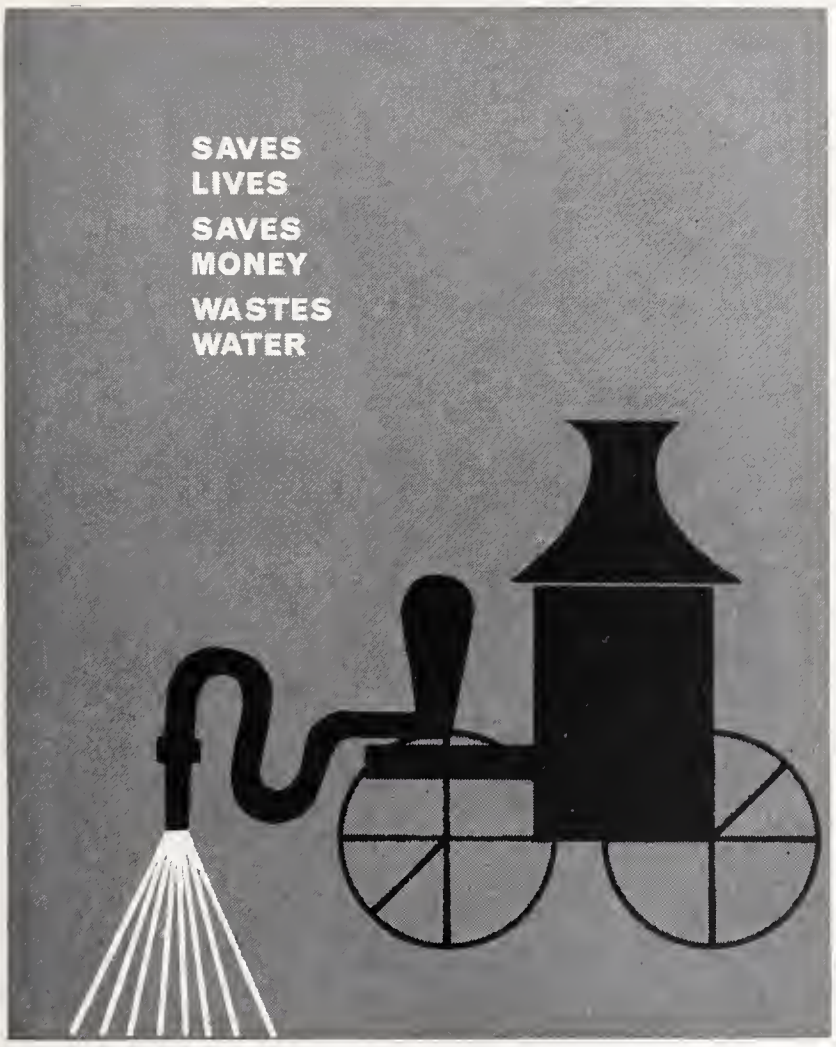

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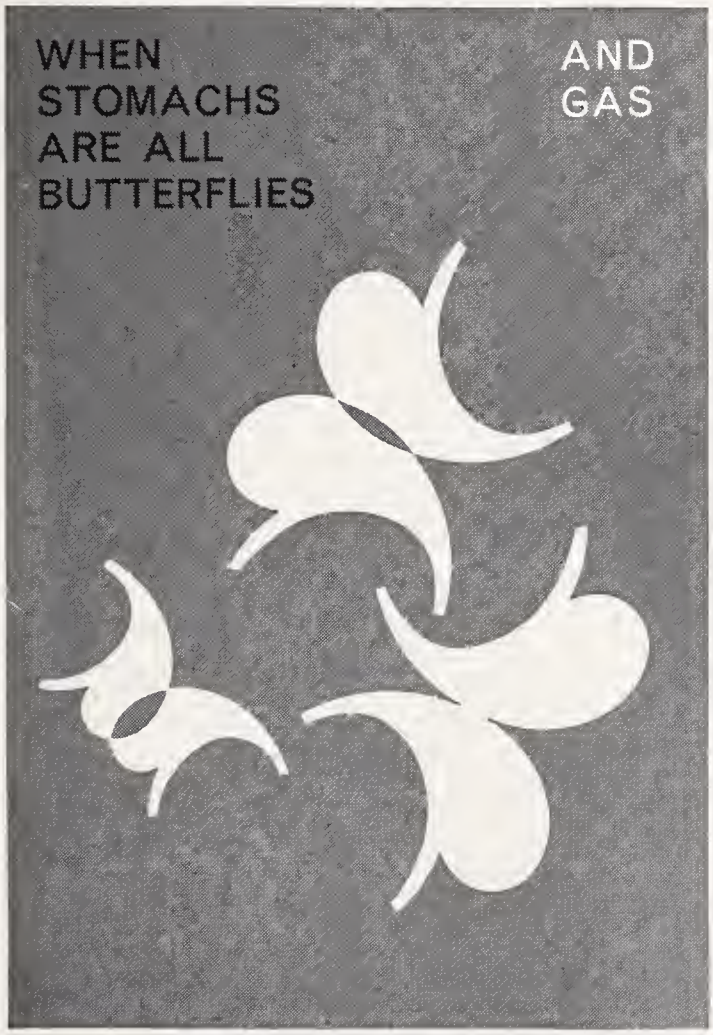

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WANTED, INTERNIST WITH INTEREST IN CARDIOLOGY. Must be board eligible or certified. To be associated with well established four man group. Salary open, no investment, early partnership. Population of 175,000 with fine churches and schools. Write University Medical-Surgical Clinic, 511 College Avenue, Lubbock, Texas.

GENERAL PRACTITIONER to assume well-established above average practice. Small modern hospital to open July 1, 1966. Located 100 miles from San Antonio in trade area of 3500. Good school, churches, and home available. Will lease or sell. Reply to William S. Katzenstein, M.D., 701 College Street, Junction, Texas 76849.

NEEDED: Board eligible or certified internist to join 9 man specialty group in Corsicana, Texas. Three internists are presently in group. Guaranteed salary and early partnership. Contact Louis E. Gibson, Medical Arts Clinic, Box 841, Corsicana, Texas. Telephone Tremont 4-8231.

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WANTED: ONE INTERNIST, ONE GENERAL PRACTITIONER AND ONE PEDIATRICIAN to join established group in Dallas, either Board qualified, or with at least two years residence, not over 35 years of age. Must have Texas license or be able to get Texas license by reciprocity. Salary open, depending on training. Partnership in two years. Group has own fully accredited hospital. Anyone interested, apply to Box 939, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701. Full details concerning group and the opportunities it offers will be furnished on inquiring.

GENERAL PRACTITIONER WANTED. Write or inquire to Medical and Surgical Clinic, 112 Denver Trail, Azle, Texas. Phone Area Code 817, CEdar 7-9811.

PHYSICIAN WANTED FOR ACTIVE GENERAL PRACTICE in suburb of central Texas city of 100,000. Unopposed practice with great growth potential. New clinic under construction. Should net $27,000 first year or will guarantee salary. All medical specialists in area and two large hospitals available. Contact Box 959, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED ASSOCIATES beginning July 1, 1966, in urology, orthopedics, cardiology, opthalmology, otolaryngology in a new clinic-hospital. Address replies to 4809 College, Suite 104, Lubbock, Texas.

PHYSICIAN WANTED: General practitioner or internist to join group operating accredited 100 bed hospital and clinic in Dallas, Texas. Salary negotiable. Write Gillon M. Cole, M.D., 233 West 10th St., Dallas, Texas.

MODERN MEDICAL-DENTAL CENTER, 3721 South Presa. Space available eye, psychiatry, pediatrics or general practice. Six doctors and three dentists with large separate practices. No rent for the first few months to help you get started. Write Dr. R. M. Dufner, 3721 South Presa, San Antonio, Texas, for further information.

WANTED: ONE GENERAL PRACTITIONER AND ONE INTERNIST to join 8 man group in progressive Texas Panhandle city of 12,000. Write or call C. E. Rush, M.D., Hereford, Texas.

GENERAL PRACTICE—Need man who is willing to help with obstetrical practice in group located in Central Texas. Partnership available after three years if mutually satisfactory arrangements can be worked out. Presently offering percentage with minimum guarantee. Please reply to Box 972, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

ASSOCIATE WANTED: Well established doctor who has been operating a three man clinic will be without an associate in the near future. Prefer a young general practitioner. Opportunity to advance in general surgery. May start on salary, interest, or percentage or anyway desired without expense. Living expenses furnished if necessary until well established. If interested, address inquiry to P. O. Box 3669, Odessa, Texas.

WANTED: Medical doctor to practice in fine small town. 1,751 population, (1960 census) with trade area radius of 20 miles, with 4500 population in trade area. Now have one practicing D.O. Contact, Nixon Area Chamber of Commerce, P. O. Box 156, Nixon, Texas 78140.

WANTED—General practitioner, ENT, internist, and pediatrician for small but rapidly growing group on the Texas Gulf Coast. Presently with four doctors, expanding to eight, and two dentists. Complete lab, x-ray, and small operating room. Full partnership after one year if mutually agreeable. Good schools, churches, recreational facilities, and good climate. Please reply to Box 974, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas.

GENERAL PRACTITIONER WANTED: Ten beds hospital clinic, 45 miles north of Austin; trade area of 3,500. Laboratory, x-ray and treatment rooms. Well established practice, excellent opportunity for aggressive individual. For further information, write Florence Clinic, Inc., Florence, Texas. Call O. T. Gray, Secretary, ST 3-2410, Florence, or Harold Bullard (ST 3-2262 or ST 3-2705).

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INTERNAL MEDICINE: (g) Assn 13 spec; 3 in dept; exc sal 1st w/fringes, prtnrshp 5 yrs; med schl city. (h) Assn 27 men; 6 man dept; outstandg oppor; med cntr. (i) Assn 7 spec; mad cl bldg; early prtnr nettg area $35,000; call twn.

OB-GYN: (j) Assn surg, ped & internist; w/45-bd JCAH; min $18,000 1st, then %-prtnrshp net $35,000; agric area. (k) 3rd man in dept; assn lge grp; sal 2 yrs then prtnr; med schl city.

PEDIATRICS: (l) Assn w/3 GP's; awn 33-bd hsp; exc facils; area $20,000 1st; residentl area nr Haustan. (m) Assn expandg grp; to $20,000 guar 1st yr; early prtnrshp; med cntr. (n) Assn 2 peds, 1 Cert; sal open; manufac area.

SURGERY: (o) Assn w/neuro surg, estab 11 yrs; ample growth potentl; area $25,000 1st; univ city. (p) Assn w/Cert plas surg; outstandg pract, long estab; med schl city.

UROLOGY: Jn 3-man urol grp; all Cert urol; sal open; caunty seat.

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WANTED: GENERAL PRACTITIONER to associate with established practitioner in thriving suburb of Dallas. No investment required. Well equipped offices, lab, x-ray. Nearby industrial site under development, already employing 1,000 and destined to have 10,000. Excellent hospital facilities 20 minutes away in Dallas. Will consider guarantee, salary, percentage, or renting and sharing office and equipment, to the right person. Ample opportunities to have whatever size and type practice desired. Extremely desirable fringe benefits. Contact Allen Maurice Fain, M.D., 621 Hustead and Fain Drive, Duncanville, Texas.

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GENERAL AND TRAUMATIC SURGEON to join eight man group. Should exceed $18,000 first year. Possibility of joining group after three years. Thirty-six years of age or younger. Two clinics ideally located in progressive Houston, Texas. Inquire: B. A. Lawrence, M.D., Glen Valley Clinic, 7630 Bellfort, Houston, Texas 77017.

WANTED—YOUNG ENERGETIC GENERAL PRACTITIONER in Texas gulf coast area to join group of three other general practitioners. Salary open, with ample time off. Good recreational facilities for hunting and fishing. Please reply to Box 979, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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GENERAL AND THORACIC SURGEON, F.A.C.S., with 20 yrs. experience. Grad. Havana Univ., Texas lic'd; outstanding univ. training; 2 yrs. rot. int.; 4 yrs. res. in surg.; 4 yrs asst. surg. One year rot. int. in Texas. Seeks position group. assn. or institution with salary. Reply to Box 872, Texas Medicine, 1801 N. Lamar Blvd., Austin, Texas 78701.

OTOLARYNGOLOGIST, American Board Certified, age 39, family, desires relocation. Texas licensed, university hospital trained, available after July 1, 1966. Seek group association or favorable location for solo practice. Reply to Box 976, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

GENERAL PRACTITIONER. Age 44 with fifteen years experience in general practice including moderate industrial practice seeks position in Dallas or suburbs. Will consider general practice with group, industry, insurance company, or federal or city position. Available July 1, 1966. Reply to Box 963, Texas Medicine, 1801 North Lamar, Austin, Texas 78701.

DESIRE RELOCATION—Solo practice has become far too demanding for me. I would like to join a moderate to large group where the hours are regular and limited to a reasonable duration. Also, where the duties are shared on an equal basis. I have been in practice for 22 years and have limited my practice to general medicine and do not do surgery or O.B. Desire to relocate in or near a major city in the north east or north central part of Texas, where ample courses in post graduate medicine are available. Have Texas license and am in good standing with TMA. Please reply to Box 975, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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FOR SALE OR RENT: Clinic Building, 2500 feet for one or two at Earth, Texas. Completely equipped with EKG, ER, two treatment rooms, recovery room, ER facility, instruments, etc. All less than two years old. Three vacant rooms for second man. Take over $25,000 mortgage. Enough land for your own hospital. Community currently active in this way. Hospitals 17-30 miles. Get it all on one note. Keys at Pound's Pharmacy, Earth. Call owner, C. H. Bausman, M.D., Casey-Bausman Clinic, Post, Texas.

FOR RENT: MODERN CLINIC building, furnished or unfurnished. Suitable for two physicians or one physician and one dentist. Has large reception room, doctor's office, two treatment rooms, recovery room, minor surgery room, colored waiting room, carport and ample parking space in central location downtown Bay City, Texas. Call or write Mrs. B. E. Simons, CI 5-2626, 3104 Avenue I, Bay City, Texas. Would prefer to rent but might consider selling it.

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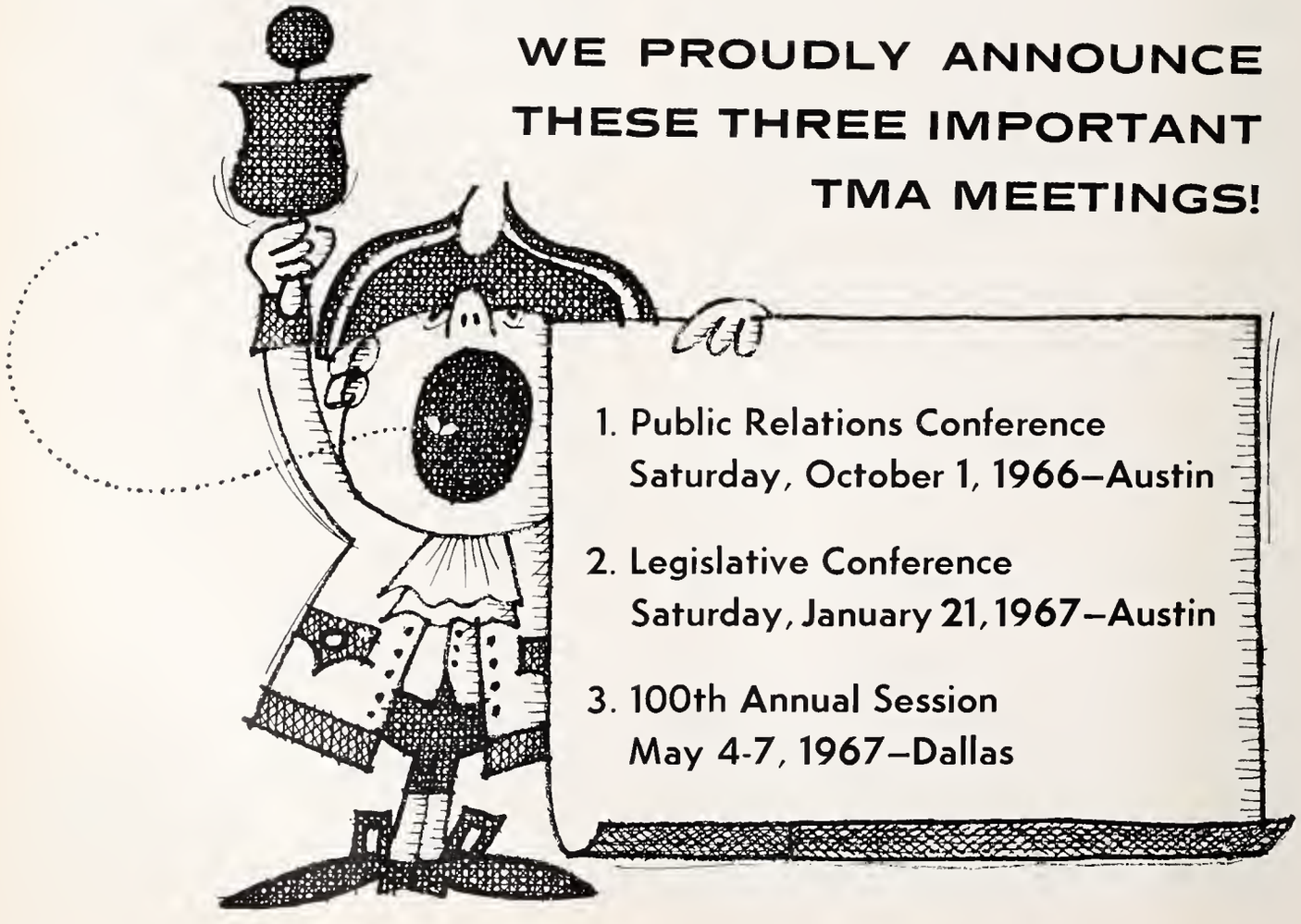

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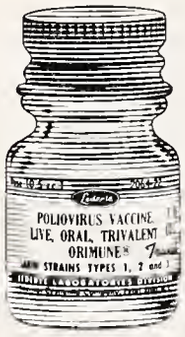

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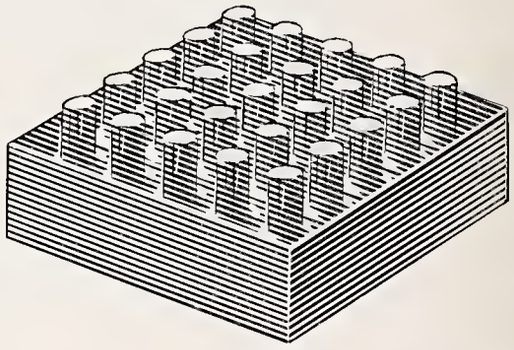

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*Rosenthal, S. R., Nikurs, L., Yordy, E., and Williams, W.: Scientific Exhibit Presented at the Annual Meeting of the National Tuberculosis Association, Chicago, Illinois, May 30-June 2, 1965.

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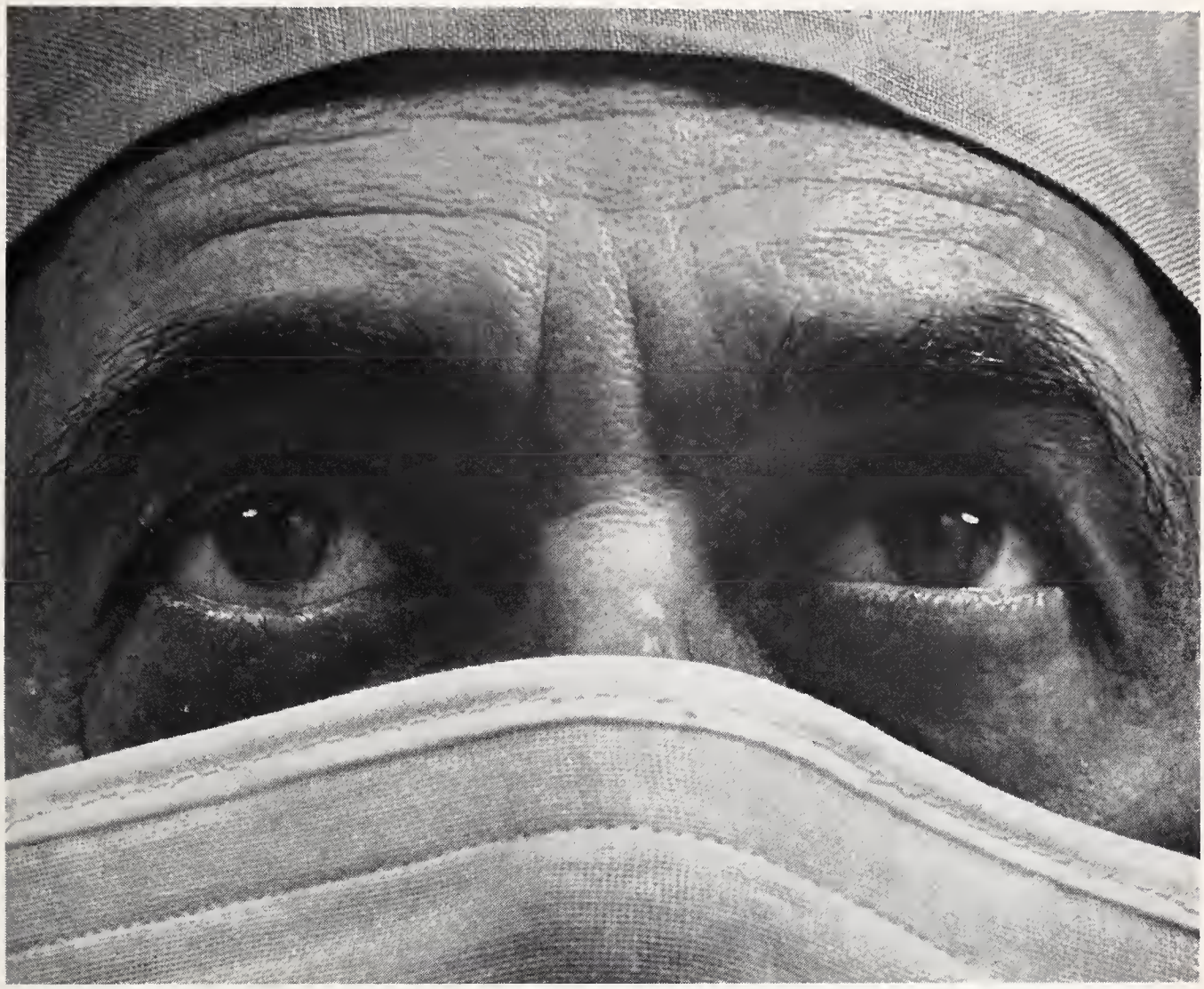

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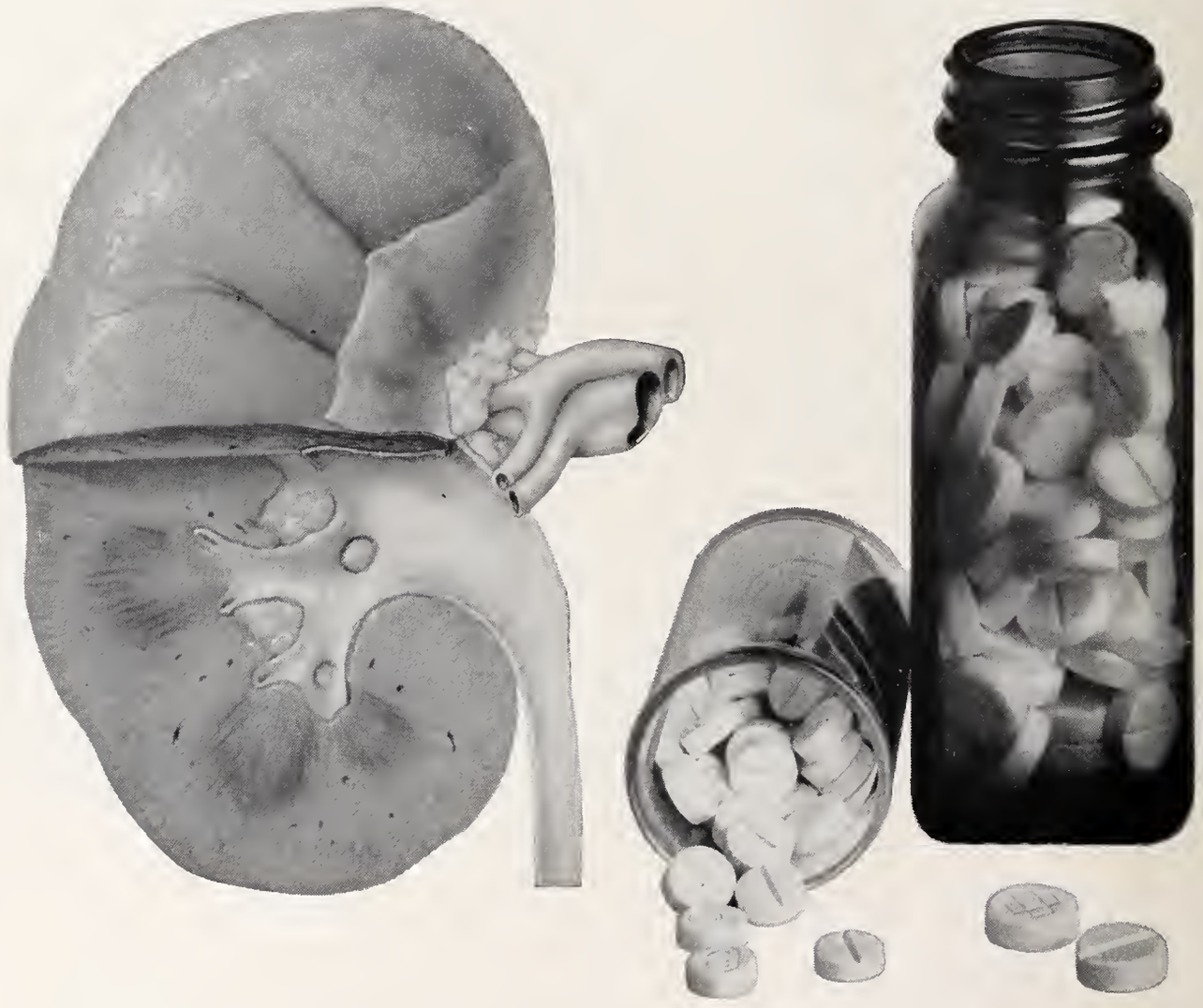

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Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

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Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

One instance each of pancytopenia and leukopenia, occurring when carisoprodol was administered with other drugs, has been reported, as has an instance of fixed drug eruption with carisoprodol and subsequent cross reaction to meprobamate. Rare allergic reactions, usually mild, have included one case each of anaphylactoid reaction with mild shock and angioneurotic edema with respiratory difficulty, both reversed with appropriate therapy. In cases of allergic or hypersensitivity reactions, carisoprodol should be discontinued and appropriate therapy initiated. Suicidal attempts may produce coma and/or mild shock and respiratory depression.

Dosage: Usual adult dose is one 350 mg. tablet three times daily and at bedtime.

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Indications: Indicated in the therapy of acute severe infections caused by susceptible organisms and primarily by bacteria more sensitive to the combination than to either component alone. In any infection in which the patient can be expected to respond to a single antibiotic, the combination is not recommended. Signemycin should not be used where a bacteriologically more effective or less toxic agent is available. *Triacetyloleandomycin, a constituent of Signemycin, has been associated with deleterious changes in liver function.* See precautions and adverse reactions.

Contraindications: Contraindicated in individuals who have shown hypersensitivity to any of its components. Not recommended for prophylaxis or in the management of infectious processes which may require more than 10 days of continuous therapy. If clinical judgement dictates therapy for longer periods, serial monitoring of liver function is recommended. Not recommended for subjects who have shown abnormal liver function tests, or hepatotoxic reactions to triacetyloleandomycin.

Precautions and Adverse Reactions: Triacetyloleandomycin, administered to adults in daily oral doses of 1.0 gm. for 10 or more days, may produce hepatic dysfunction and jaundice. Adults requiring 3 gm. of Signemycin initially should have liver function followed carefully and the dosage should be reduced as promptly as possible to the usual recommended range of 1.0 to 2.0 gm. per day. Present clinical experience indicates that the observed changes in liver function are reversible after discontinuation of the drug.

Use with caution in lower than usual doses in cases with renal impairment to avoid accumulation of tetracycline and possible liver toxicity. If therapy is prolonged under such circumstances, tetracycline serum levels may be advisable. In long term therapy or with intensive treatment or in known or suspected renal dysfunction, periodic laboratory evaluation of the hematopoietic, renal and hepatic systems should be done. Formation of an apparently harmless calcium complex with tetracycline in any bone forming tissue may occur. Use of tetracycline during tooth development (3rd trimester of pregnancy, infancy and early childhood) may cause discoloration of the teeth. Reversible increased intracranial pressure due to an unknown mechanism has been observed occasionally in infants receiving tetracycline. Glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and definite allergic reactions occur rarely. Severe anaphylactoid reactions have been reported as due to triacetyloleandomycin. Photosensitivity and photoallergic reactions (due to the tetracycline) occur rarely. Medication should be discontinued when evidence of significant adverse side effects or reaction is present. Patients should be carefully observed for evidence of overgrowth of nonsusceptible organisms including fungi, which occurs occasionally, and which indicates this drug should be discontinued and appropriate therapy instituted. Steps should be taken to avoid masking syphilis when treating gonorrhea.

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References: 1. Editorial: JAMA *191*:592 (Feb. 15) 1965. 2. Meilman, E., in Moyer, J.H.: Hypertension, Philadelphia, W.B. Saunders Company, 1959, p. 395.

BRISTOL THERAPEUTIC SUMMARY
For complete information consult Official Package Circular.

Indications: Essential hypertension.

Warnings: Small-bowel lesions (obstruction, hemorrhage, perforation) have occurred during therapy with enteric-coated formulations containing potassium, with or without thiazides. Such potassium formulations should be used with Salutensin only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs.

Contraindications: Salutensin is contraindicated in severe depression.

Precautions: Azotemia, hypochloremia, hyponatremia, hypochloremic alkalosis and hypokalemia (especially with hepatic cirrhosis and corticosteroid therapy) may occur, particularly with pre-existing vomiting and diarrhea. Potassium loss, which may cause digitalis intoxication, responds to potassium-rich foods, potassium chloride or, if necessary, stopping therapy. Serum ammonia elevation may precipitate coma in precomatose hepatic cirrhotics. Discontinue therapy two weeks before surgery or if myocardial irritability, progressive azotemia or severe depression occur. Exercise caution with patients with peptic ulcers or renal insufficiency (if severe, Salutensin is contraindicated).

Side Effects: Hydroflumethiazide: Purpura plus or minus thrombocytopenia, hyperuricemia, leukopenia, hyperglycemia, glycosuria, malaise, weakness, dizziness, fatigue, paresthesias, muscle cramps, skin rash, epigastric distress, vomiting, diarrhea and constipation. *Reserpine:* Depression, peptic ulceration, diarrhea, Parkinsonism, nasal stuffiness, dryness of the mouth and, with overdosage, agitation, insomnia and nightmares. *Protoveratrine A:* Nausea, vomiting, cardiac arrhythmia, prostration, excessive hypotension and bradycardia. (Treat bradycardia with atropine and hypotension with vasopressors.)

Usual Dose: 1 tablet *b.i.d.*

BRISTOL LABORATORIES
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Salutensin®

Each tablet contains:
protoveratrine A, 0.2 mg.;
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heart disease or psychic tension?

"Heart symptoms"—chest pain, tachycardia, arrhythmia—invariably alarm and preoccupy the patient, though they may be completely without organic basis. Such symptoms often are somatic masks of psychic tension, arising from constant encounters with stressful situations.

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Valium® (diazepam)

2-mg, 5-mg, 10-mg tablets

In preseribing: Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Preeautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effeets: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

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Phenobarbital	7.5 mg.

(WARNING: MAY BE HABIT FORMING)

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Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.
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Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of

one mid-evening

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300 mg FILM COATED TABLETS

demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg of demethylchlortetracycline HCl.

Tablets: film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

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SALMONELLA CARRIERS "PRESENT A FORMIDABLE FIFTH COLUMN."[2]

Furoxone exerts "outstanding activity against Salmonella sp."[3] and "has enhanced the physicians' armamentarium in that it now affords a relatively effective, safe bactericidal agent, which may be used to treat the active disease as well as the carrier state".[2]

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Proper antibacterial therapy for individual cases should help in reducing the total number of cases in an epidemic situation.

Side effects are infrequent. A few hypersensitivity reactions to Furoxone have been reported including a fall in blood pressure, urticaria, fever, arthralgia and a vesicular or morbilliform rash. These reactions subsided promptly following withdrawal of the drug. Primaquine-sensitive patients may develop a mild reversible hemolytic anemia. Nausea, emesis, headache or malaise may occur occasionally. To obviate alcohol-disulfiram type reactions, advise against use of alcohol-containing drugs during therapy and four days thereafter.

Do not give to infants under one month of age.

Composition: Furoxone Liquid contains, per 15 cc. tablespoonful, furazolidone 50 mg., pectin 225 mg., and kaolin 3.0 Gm. Furoxone Tablets each contain 100 mg. of furazolidone.

References: 1. Editorial: J.A.M.A. *189*:691 (Aug. 31) 1964. 2. Foertsch, J. H.: J. Oklahoma Med. Assn. *57*:449 (Oct.) 1964. 3. Paul, H. E., and Paul, M. F.: The Nitrofurans—Chemotherapeutic Properties, in Schnitzer, R. J., and Hawking, F. (Eds.) Experimental Chemotherapy, Vol. 2, New York, Academic Press, 1964.

Originators and Developers of The Nitrofurans
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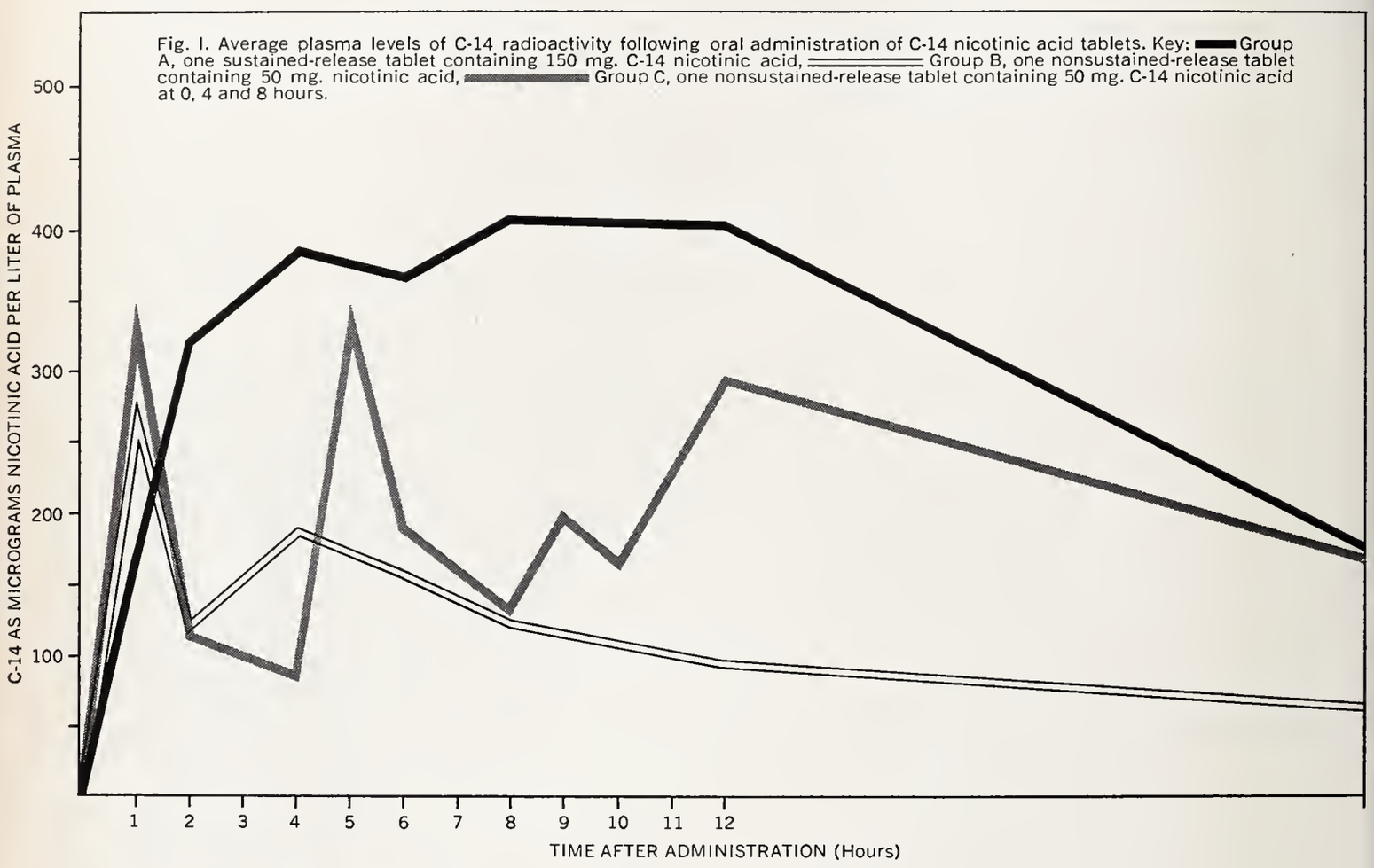
NORWICH, NEW YORK

Sustained circulatory, respirator
and cerebral stimulation for th

Fig. I. Average plasma levels of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: ▬ Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, ═══ Group B, one nonsustained-release tablet containing 50 mg. nicotinic acid, ▬ Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.



(fewer absent doses by absent-minded patients)

Human volunteer subjects were administered Geroniazol TT tablets with the nicotinic acid component made radioactive with C-14. Plasma and urine samples were analyzed. (See Figures I and II) The radioactive tracer study substantiated the previous clinical evidence that the release of nicotinic acid from the Geroniazol TT tablet produced a gradual rise in plasma levels to a plateau for a total of 12 hours and more.

Such proven sustained activity makes the management of geriatric patients much easier by minimizing the possibility of neglected doses through absent-mindedness or senile confusion. Therapy *can* be continuous on a daily dose of only one Geroniazol TT tablet every 12 hours.

The gradual release of nicotinic acid in Geroniazol TT will provide the well-known peripheral vasodilatation needed in patients with deficient circulation and with a minimum amount (if any) of "flushing." Also, cerebrovascular circulation is complemented by pentylenetetrazol, long-established as a cerebral and respiratory stimulant.

Geroniazol TT improves the typical, unfortunate, signs of senile confusion. Patients become more alert,

ged and debilitated

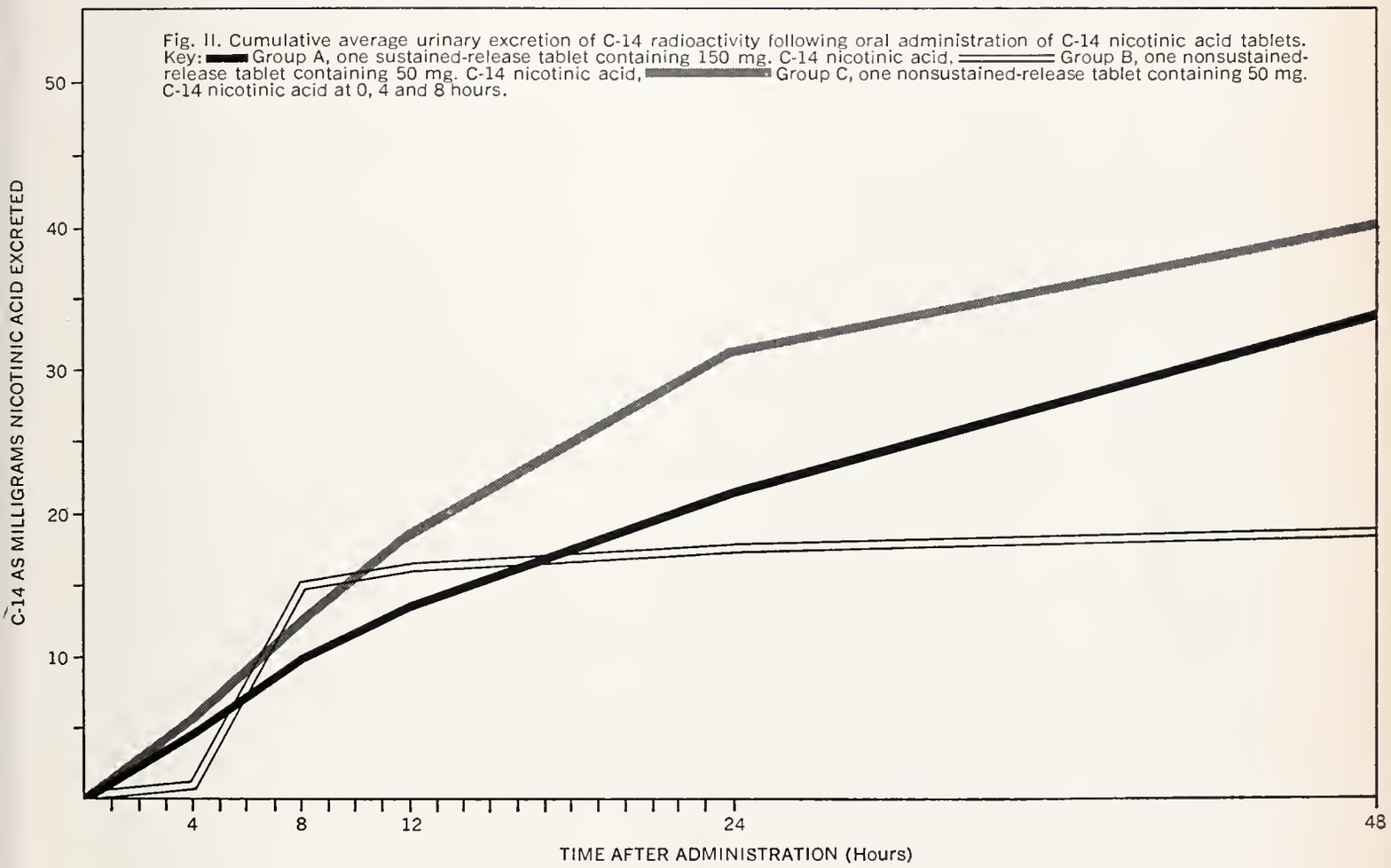


Fig. II. Cumulative average urinary excretion of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MILLIGRAMS NICOTINIC ACID EXCRETED
50
40
30
20
10
4
8
12
24
48
TIME AFTER ADMINISTRATION (Hours)

less confused and moody. Personal care, memory, emotional stability, social attention improve. Fatigue, apathy and irritability are reduced.

A prescription for 100 tablets of Geroniazol TT will permit your patients to enjoy the benefits of time-prolonged nicotinic acid/pentylenetetrazol therapy, at an economical price. Dosage is only one tablet every 12 hours.

Contraindications: There are no known contraindications.

Precautions: Exercise caution when treating patients with a low convulsive threshold.

Side Effects: Side effects are rarely encountered, however due to the vasodilatation effect of nicotinic acid, transitory mild nausea, flushing, tingling and pruritus are possible.

Dosage: One tablet every 12 hours.

Supplied: Prescribe bottles of 100 tablets, to take advantage of recent price reduction.

References: 1. Report by Nuclear Science & Engineering Corp., Pittsburgh, Pa., in files of Philips Roxane Laboratories. 2. Connolly, R.: W. Virginia Med. J. *56*:263 (Aug.) 1960. 3. Curran, T. R., and Phelps, D. K.: Am. Pract. & Digest Treat. *11*:617 (July) 1960.

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nicotinic acid 150 mg., pentylenetetrazol 300 mg.
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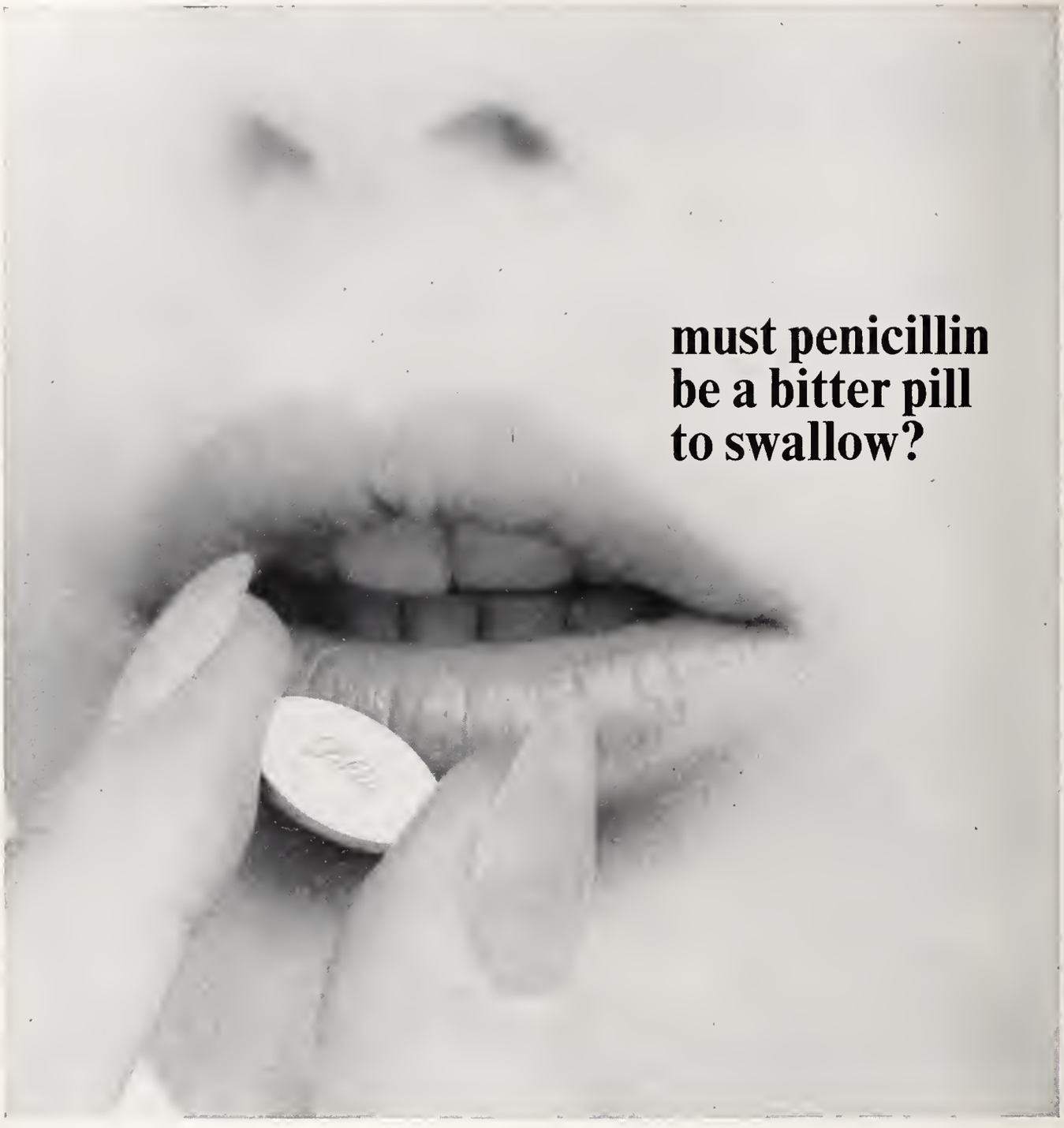

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Indications: V-Cillin K is an antibiotic useful in the treatment of infections caused by streptococci, pneumococci, and sensitive strains of staphylococci.

Contraindications and Precautions: Although sensitivity reactions are much less common after oral than after parenteral administration, V-Cillin K should not be administered to patients with a history of allergy to penicillin. As with any antibiotic, observation for overgrowth of nonsusceptible organisms during treatment is important.

Usual Dosage Range: 125 mg. (200,000 units) three times a day to 250 mg. every four hours.

Supplied: Tablets V-Cillin K, 125 or 250 mg.; also, V-Cillin K, Pediatric, 125 mg. per 5-cc. teaspoonful, in 40, 80, and 150-cc.-size packages.

V-Cillin K® **Six-Second Barrier to Bitterness**

Potassium Phenoxymethyl Penicillin

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TEXAS MEDICINE

VOLUME 62 NUMBER 6 JUNE 1966

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"Like Begets Like"

THERE IS A PROVERB for everything, and nearly any proverb can be bent to the advantage of the user. Admitting this, I would (for present purposes) recast "Like begets like" into "Collectivism begets collectivism."

It may be more difficult to demonstrate as well that "Individualism begets individualism"; however, many in the Texas Medical Association, including the House of Delegates, hope that all physicians in the state will voluntarily return to the strictly individual relationship between doctor and patient.

Though tempted to give a history of Medicine's dive into collectivism to avoid collectivism, I shall refrain in the interest of space. Nevertheless, I do ask each of you to meditate upon the likelihood that good begets good, bad begets bad, war begets war, collectivism begets collectivism, and tyranny begets tyranny.

Rather, I would emphasize a thing which doctors of medicine have always wanted: a personal, private, individual relationship between themselves and each of their patients. Physicians are individualists at heart because none is worthy to aid the suffering until he has attained that height of skill, art, and training which makes him so thoroughly responsible for decisions in life-and-death matters that he needs no "boss." As his own boss, he works for the hardest taskmaster imaginable—one he dare not try to fool—and the one best fitted to keep him everlastingly aware of his responsibilities.

This sense of responsibility and trustworthiness in physicians of the past has inspired a like sense in each oncoming generation to become as proficient as is humanly possible. This can be attained, of course, only by individual struggle for perfection; it does make the physician a strict individualist.

It is not surprising, therefore, that the TMA House of Delegates instructed its delegates to the

June meeting of the AMA to present a statement and resolution which proposes succinctly a plan designed to prove that "Individual responsibility begets individual responsibility":

"The duty to maintain quality in medical care lies by oath and ethics with the physician. As its obligation to the public, the medical profession must fulfill its responsibility to maintain the quality of care. This duty is best served by a personal relationship in which the physician is responsible directly to the patient for care and for medical services rendered. The patient, in turn, assumes as his personal obligation the responsibility for paying the physician for professional services rendered. The patient should be accorded unlimited freedom to employ whatever mechanism he finds most suitable in financing the costs of his medical care.

"This doctrine of individual responsibility is fully compatible with the American way of life, and the traditional approach to financing the purchase of goods and services. In keeping with the tenets of free choice, it should be emphasized that the principles of individual responsibility are voluntary."

The resolution calls for endorsement of the ethics and the principles of individual responsibility in the rendering of medical services by physicians, except when this principle conflicts with the law of the land; declares that the principles of individual responsibility are well sustained by an adequate statement or report by the attending physician, recognized as an ethical means for use by those physicians who voluntarily desire to do so; and urges that all AMA members be informed of this action so that those who voluntarily wish to do so may employ the principles of individual responsibility in preserving an ethical doctor-patient relationship, and in maintaining the highest quality of medical care.

—JOHN K. GLEN, M.D., Houston,
Vice President,
Texas Medical Association.

Radiologists' Dilemma

IT IS WRITTEN in the second chapter of Revelation that an angel came unto St. John at the Island of Patmos and directed him to write the church in Ephesus saying, "I know thy works, and thy labor, and thy patience . . . Nevertheless I have somewhat against thee . . ."

In 1965 and 1966, radiologists have likewise been approved—and chided—by general medical organizations. Our contributions to patient care and the consultative services we provide for referring physicians have been applauded. Our atypical financial arrangements with hospitals have been condemned. These arrangements have not only jeopardized the integrity of our specialty as a branch of medical practice, but they have also provided a precedent that is a potential threat to all physicians.

The American Medical Association has clearly recognized that if radiology can be fiscally transmuted into a part of hospital service, then other branches of medicine can be converted by similar alchemy. The AMA House of Delegates said it well in October, 1965:

> Hospital-based medical specialists are engaged in the practice of medicine. The fees for the services of such specialists should not be merged with hospital charges. The charges for the services of such specialists should be established, billed and collected by the medical specialist in the same manner as are the fees of other physicians.

This opinion has been echoed by medical associations over the nation. About two-thirds of the state medical societies, including the Texas Medical Association, have iterated similar policies, as have many county medical societies.

Radiologists began working toward the goals set forth in these policies late in 1964. After an agonizing reappraisal of our situation, we recognized that, to fulfill our medical role in patient care, we needed to modify our arrangements for practice in hospitals. We understood that that single change could solve the problem of low recruitment compounded by a mushrooming volume of patient service. Twenty-five percent of our openings for residency training in radiology were unfilled. Since 1946, our clinical workload has been increasing at a rate of 12 percent per year. The USPHS reported that 100 million Americans required radiologic examination or treatment in 1961. By 1964 this increased to 108 million. Studies at the UCLA Medical Center and Johns Hopkins revealed that time-consuming, complex, special radiologic studies are increasing at a rate of 30 percent annually. A radiologist can accom-

plish from four to six of these procedures per day. The USPHS National Advisory Committee on Radiation has estimated that by 1970 a minimum of 20,000 radiologists will be needed, and at the present rate of accession we shall be fortunate to have 9,000 radiologists.

This basic medical situation has caused radiologists to strive to alter relationships with hospitals to align them with those of other physicians. We believe that we can recruit young physicians into a field that is definitely a branch of medicine more easily than we have been able to recruit them into a limbo between medical practice and hospital service. We are striving to practice in hospitals on the same terms as other members of the medical staff.

Some hospital administrators and boards of trustees have indicated that radiologists will not be permitted to practice with the same responsibilities and prerogatives as other medical staff members. If this occurs, and if we are not permitted to enter hospitals in which we are practicing on July 1, so be it. Assuming July 1, 1966, is like any other Friday, approximately 150,000 patients will be referred to radiologists to be examined or treated in hospital departments of radiology. We shall be available, not only to care for these patients and to consult with the physicians who refer them, but also to continue our present services in teaching, research, and the administration of hospital departments of radiology. In the future, however, we plan to be available only on a private practice basis, not as hospital employees.

So, if we are "locked out" of hospitals on July 1, then we would stress that this is a situation not of our making, nor our desire. If we are not permitted to practice as you do on July 1, then we believe it is incumbent upon administrators and board members who make this impossible to make necessary explanations to patients, the public, and to you. Radiologists are eager and willing to practice—not on any special basis or with any special advantages—but also with no special disabilities and disadvantages.

We believe that the effort we are making is in the best interests of patient care and medical practice in general. The policy positions taken by general medical organizations support this thesis. We need your personal support now.

—J. E. MILLER, M.D., Dallas.

COMMENTS

Certainly, it is the feeling of most of the profession that *all* physicians should be on organized hospital staffs, and that *none involved in patient care* should be hospital employees. Surely, the phrase "hospital-based specialties" was coined by enemies of these men and women whose work requires them to spend more time in hospitals than the remainder of us. Hospitals should never be required to hire physicians to see charity patients or clinic cases. These people, too, fall into the category of "ours to serve."

Hospitals provide operating rooms and instruments for those of us who do surgery. For the use of these rooms, these instruments, and the personnel who man them, the patient is charged a fee. I never have seen, nor can I now see, the difference between this arrangement and those for radiologists and pathologists. In my opinion, the following statement by the AMA is self-explanatory: "The charges for services of such specialists should be established, billed and collected *by the medical specialist* in the same manner as are the fees of other physicians."

—WALTER WALTHALL, M. D., San Antonio,
Chairman, Board
of Councilors, TMA.

An Essay On Contemporary Leprosy

LEPROSY is a disease rarely included in differential diagnosis by today's physician. The appearance at the La Tuna, Texas, Penitentiary of a young patient from the state of Sinaloa, Mexico, with lepromatous leprosy refreshed memories of an earlier public health problem with migrant Mexican laborers from Sinaloa, an endemic area for the "diffuse lepra" variety of Hansen's disease.

"Aole Kanawai Ma Keia Wahi." [In this place there is no law.] This expression was the watchword during the early days of lawlessness and desperate abandon at Kalawao, Molokai, following the edict of Kamehameha V. This culminated in the isolation of some 200 ravaged, despondent souls on that hostile shore in January, 1866. This outcry was symbolic of the despair that had reechoed throughout 2,000 years of experience with the great destroyer, leprosy.

Hansen's disease, although declining, still claimed an estimated more than 10 million victims in 1959.[1] Advances in chemotherapy have begun to revolutionize attitudes toward

the disease. Superstition and fear are being slowly dissipated by scientific knowledge; however, much about leprosy remains a mystery.

The causative organism has been identified since 1874, yet the first animal transmission was achieved in 1958.[2] Koch's postulates have yet to be fulfilled. The incubation period and the mode of transmission for the disease are unknown. The bacillus does not grow on artificial media, but can be cultured on the footpads of mice and in tissue culture. Therefore, organism sensitivity to chemotherapeutic agents cannot be ascertained and the evolution of resistance cannot be detected by usual laboratory techniques. Consequently, selection of specific sulfone drugs and duration of treatment are based on clinical observation.

How can one be sure when a patient is permanently cured? Rarely, individuals treated for five to ten years, who have become bacteriologically negative according to smears of cutaneous tissue fluid and nasal septum scrapings, have been known to relapse. Some patients in direct contact with leprous family members never contract the disease. One woman developed the illness at age 69 after having lived with her afflicted husband for almost 50 years. Such capricious clinical behavior is unprecedented with other infectious diseases.

The historic paper published by Paget[3] and associates in 1943 represented the first significant advance in the long, dark history of therapy in Hansen's disease.

The illness seen today, in the sulfone era, is remarkably and mercifully different. A patient with classic, advanced, untreated lepromatous leprosy has such dramatic configuration that his disease cannot be overlooked. Paradoxically, in the earliest stages of the disease, correct diagnosis is the exception, especially in geographical areas where the disease is rare. The average duration of illness at the time of initial recognition is about five years. Thus, the disease is usually advanced when first recognized in this country, and the more ominous lepromatous type is more likely to be overlooked. The anesthetic macule, hallmark of the tuberculoid type, attracts the patient's attention; it is usually recognized by the alert physician.

Lepromatous leprosy may be manifested by subtle symptoms of considerable duration. It ravages skin and nerves, yet spares vital organs. Nasal obstruction and epistaxis are common first symptoms. Sometimes a curious disturbance in sensation will attract the patient's attention. The first definite cutaneous signs will be the occurrence of one or more macules or plaques. The lepromatous macule has an indefinite border; it may be hypopigmented or hyperpigmented and is often erythematous. The lepromatous plaque is elevated from the surrounding skin. Diffuse infiltration of skin and mucosa without evidence of distinct nodules, plaques, or macules is also common. The infiltrated skin thickens and natural lines become exaggerated and characteristic corrugations develop, contributing to the so-called leonine facies.

Infiltration around the hair follicles causes alopecia, especially noticeable on the outer aspects of eyebrows. The nasal mucosa is involved in a significant proportion if not in all cases of lepromatous leprosy. Ulceration may be followed by septal destruction with resultant facial deformity. Pendulous ear lobes, thickened skin, alopecia, septal destruction, and perhaps advanced keratoconjunctivitis combine to present the characteristic facial features of the advanced lepromatous leprosy patient. Ulceration of the nasopharynx is less common. Soft palate, uvula, and aryepiglottic folds may be swollen, hard, and smooth. Occasionally, ulceration may occur. When the larynx is involved, the voice may be reduced to a harsh whisper. Cases of obstruction requiring tracheostomy once were extremely common but have become much less frequent today because of sulfone treatment. At Kalaupapa I observed that there were only 8 of the 215 elderly persons with functioning tracheostomies.

Ulceration may occur in any mucocutaneous area in which lepromatous infiltration has occurred. The "Lucio" phenomenon of cutaneous necrosis is seen frequently in patients from Sinaloa. Such ulcers discharge many bacilli, and must be distinguished from those of the trophic type since the latter do not discharge the Hansen's bacilli. Lepromatous eye lesions are common. Diffuse infiltration and occasional nodules occur in the upper lid, sclera, cornea, iris, and ciliary body. The cornea is most vulnerable, and all stages of keratitis may be seen.

Lepromatous leprosy is the contagious type and presents the most significant therapeutic and diagnostic problems. Resistance to the infection is low. The lepromin skin test (Mitsuda) is negative. This is interpreted as an indication of lack or absence of antibody response. Sulfone therapy can arrest most of the lepromatous processes, but there may

be irreversible nerve damage. Arrest of the infectious process does not assure termination of symptoms. Peripheral nerve involvement may progress to an advanced stage before symptoms bring the patient to medical attention. This is the principal continuing problem in contemporary leprosy therapy—how to manage the sequelae of peripheral nerve damage.

In the tuberculoid variety, the most frequent early sign is appearance of a macule (leprid) in which there is impairment of sensation. Sometimes the patient will not be aware of his illness until a lepra reaction occurs with widespread erythematous plaques, fever, and neuritis. Except during these acute reactions, bacilli are extremely rare in the cutaneous tissue fluid of the tuberculoid type. When sought with diligence they are most likely found in the margins of macules. Usually these patches are hypopigmented; they may be erythematous. The typical tuberculoid lesion is raised, usually pebbled in appearance, and extends irregularly by centrifugal spread leaving an atrophic central area. It is probable that in a leprid, some degree of hyperesthesia precedes anesthesia; color changes follow the anesthetic phase. Upon testing there is usually inability to detect light touch; a pin may be inserted without pain; the patient is unable to distinguish between hot and cold.

Polyneuritic involvement occurs in both lepromatous and tuberculoid leprosy but occurs earlier and is more severe in the tuberculoid type. In many instances the patient comes to the physician because of persistent tingling or numbness in part of an extremity. Examination may reveal atrophy of the muscles of the hand with flattening of thenar and hypothenar eminences. Weakness of interosseous and lumbrical muscles and perhaps early contracture of the fourth and fifth fingers will be detected. In these cases there may be no skin eruption; if the process has been prolonged it may be difficult to determine the type of leprosy.

Muscular changes in the hand and foot are usually accompanied by trophic decalcification of bones. In extreme cases, fingers and toes become shortened and even disappear without the occurrence of an open wound.

Thus we see the pattern of modern day leprosy. Patients are detected when peripheral neuropathy develops. Treatment with sulfones usually arrests the process, but significant neuropathic changes may have occurred already. These irreversible neural lesions and sequelae of neurologic damage cause today's problems. Anesthetic limbs are predisposed to injury. Prevention and treatment of the recurring plantar ulcer are problems. The ulcer process may be insidious and abetted by atrophy of the small muscles of the feet and gradual resorption of the phalanges and metatarsals. Normal gait is altered and the damaged, insensitive foot is continually traumatized at the same point of weight impact. It is rare for bone destruction to proceed above the ankle. However, a Charcot's joint may develop due to unheeded trauma to the anesthetic knee. A similar situation occurs in the hands. Osteomyelitis is a common component of trophic ulceration of both hands and feet, a result of peripheral nerve damage that occurred months or years before.

Another equally devastating complication affects the eyes. This is distinct from active lepromatous leprosy which can involve the eye directly with iridokerato-cyclitis progressing to blindness. Sulfone therapy can arrest this process, but insidious sequelae of neural damage may result in corneal anesthesia and paralysis of the small protective muscle of the eye. Parenthetically, damage to peripheral facial nerves may result in severe asymmetry due to uneven wasting of facial muscles. At times this may produce a most grotesque countenance. With corneal anesthesia and inability to close the lids, exposure keratitis becomes a problem. Pupillary adhesions progressing to the point of occlusion may occur, together with increasing corneal opacity. Early recognition and continuous effort to protect the eyes against physical trauma may prevent much of this difficulty.

With the changing picture of leprosy, effort must be directed toward protecting against deformity secondary to trophic change. These problems represent significant challenges to the orthopedic surgeon, plastic surgeon, neurologist, and ophthalmologist. The ultimate solution to this problem can be effected by wider education of physicians to facilitate earlier diagnosis. Early institution of sulfone therapy before the point of irreparable nerve damage is important.

Far from being a lawless, disorderly process leprosy is indeed orderly, albeit progressive and relentless if not treated with vigor and vigilance. With earlier case finding, rapid institution of therapy and vigilant prophylaxis against the secondary ef-

fects of anesthesia and trophic change, the prospect of conquest is at hand.

—Lt. Col. Robert H. Moser, El Paso.

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letters to TEXAS MEDICINE

Insurance Programs Resembling Official TMA Plans May Be Confusing

It is apparent that considerable confusion and misunderstanding exist regarding the insurance programs officially sponsored and approved by the Texas Medical Association. Disability income insurance appears to be involved to the greatest degree.

Your Association approves four insurance programs. The TMA Disability Income (Accident and Health), Major Medical, and Accident Group Plans are administered by Charles O. Finley and Company and underwritten by Lumbermens Mutual Casualty Company.

The fourth approved program is the life insurance plan through Great American of Dallas.

Other than the preceding, in the categories as listed, no agent has the authority or right to infer that his policy is connected with the Texas Medical Association in any way.

There are several disability income policies available which are known as "franchise plans." These plans are placed on the market with the inference that they are sold only to "Members of the Association" or "Members of County Medical Societies," etc. These plans have been presented to a busy physician and because of the reference to a medical organization they have been confused with the approved TMA Disability Program.

A physician may certainly purchase any policy he chooses; however, if he has any doubt and if his basic interest is in the officially sponsored and approved program, he should ask the agent if the particular plan being presented is the approved TMA plan.

Much work has been done by your Committee on Association Insurance Programs to make our Disability Income Program the best group plan of this type available. Benefits are broad, the premium rate is most favorable, and the overall success of this plan is unsurpassed. If you want this plan, be certain that the application you complete is headed "Application to Lumbermens Mutual Casualty Company for Texas Medical Association 'True Group' Disability Income Program."

Should you have any doubt about a disability plan you may now have, it would be wise to examine your policy. If it is not issued by the Lumbermens Mutual Casualty Company, you are not insured under the officially sponsored and approved plan nor are you availing yourself of the many group benefits available to our members through this program.

—Laurance N. Nickey, M.D., El Paso,
Chairman, Committee on Association
Insurance Programs,
Texas Medical Association.

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
*U.S. Pat. No. 3,062,714

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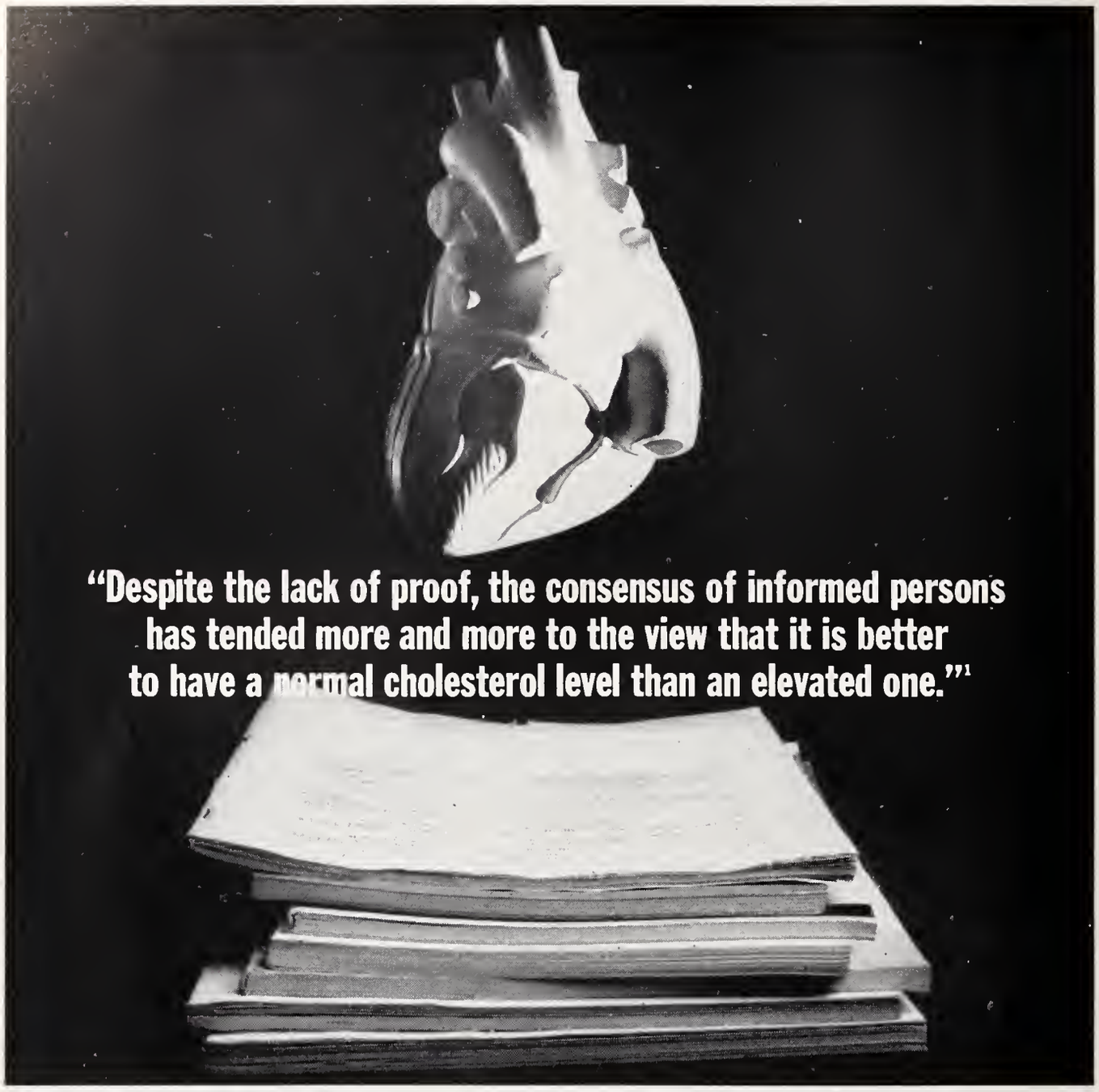
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Physicians Benevolent Fund

At the Austin meeting of the Texas Medical Association in April, a resolution presented to the House of Delegates called for a $1.00 per year dues increase to be earmarked for the Physicians Benevolent Fund.

This well-meaning resolution was wisely turned down by the reference committee, with the comment that the testimony emphasized that mandatory participation in a benevolent program would be offensive to a high percentage of our membership and would eliminate altruistic participation.

The Reference Committee did, however, suggest a substitute resolution, which resolves:

"That the House of Delegates calls attention to the membership of the Texas Medical Association that the Physicians Benevolent Fund is worthy of bequests, memorial donations, and personal contributions, and . . .

That the Texas Medical Association through its component county societies encourage all members of the Texas Medical Association to voluntarily . . . support the endowment of the Physicians Benevolent Fund."

The Fund is now actively supporting needy physicians or their widows or both. I know of no better way to pay tribute to members of our profession or to their families than to send a donation to the Physicians Benevolent Fund. It seems to me that our wives, too, are in a wonderful position to promote this fund.

James D. Murphy M.D.

President
Texas Medical Association

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meet the authors

DONALD D. HARVILLE is a resident in dermatology at the Mayo Graduate School of Medicine; his coauthor, **ROBERT R. KIERLAND** is head of the Section of Dermatology, Mayo Clinic, Rochester, Minn. **Cutaneous Malignancies, p. 42.**

JOHN L. KESTEL, JR., is clinical instructor in dermatology at Southwestern Medical School and chief of the Division of Dermatology, Methodist Hospital, Dallas. **Giant Pigmented Nevus with Vitiligo, p. 52.**

ROBERT H. LE PERE practices thoracic surgery in San Antonio. His coauthor, **ROBERT C. HARDY,** is a neurosurgeon. **Surgical Improvement of Collateral Circulation to the Brain, p. 55.**

DAVID J. TURELL, Houston, is associated with the St. Joseph's Hospital cardiac work evaluation unit, which is the first established in Texas. **Cardiac Work Evaluation, p. 66.**

BRUCE D. FALLIS, professor of pathology, Southwestern Medical School, Dallas, is guest editor of a new series of **Clinicopathologic Conferences** beginning in this issue, p. **76.**

STANLEY J. ZIMMERMAN and his coauthors are staff members of the Department of Medicine and Surgery, Baylor University College of Medicine, Houston. **Acute Hyperparathyroidism, A Surgically Correctable Cause of Coma,** p. **71.**

ROBERT H. LE PERE and **PAUL KLINGER,** San Antonio, specialize in thoracic surgery. **Mechanical Pacemaker Failure, p. 59.**

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Cutaneous Malignancies

REVIEW ARTICLES

A PHYSICIAN has a great responsibility for the diagnosis of skin cancer because the skin is readily accessible for examination and because skin biopsy is a relatively simple and safe procedure. Furthermore, the opportunity for cure is far greater for a skin cancer than for an internal malignancy.

Predisposing Factors

There are many predisposing factors in the development of skin cancer, and they may be classified as environmental factors and host factors.

Environmental Factors.—Sun exposure is the greatest single predisposing factor in skin cancer.[1] A definite relationship has been shown between sun exposure and squamous cell and basal cell epitheliomas (Fig. 1a). These cancers are much more frequent among persons living where solar radiation is more intense.[2] There is also a greater incidence of melanoma in the south than in the north, although a relationship to sun exposure has not been proved.

Ionizing radiation predisposes to the development of skin cancer. Approximately 1,000 roentgens (r) are required to produce minimal changes of radiodermatitis. The carcinogenic effect of radiation is dose-dependent, but the total dose and dose-time factors necessary for the production of cancer are unknown.[3]

Arsenic ingestion predisposes to cutaneous as well as internal malignancy. Prolonged exposure to tar and tar products predisposes to skin malignancy, as exemplified by "chimney-sweep cancer" in Great Britain.[4] Such exposure to tar in this country rarely produces cancer. Scar-forming trauma, such as occurs in chronic discoid lupus erythematosus and after burns (Fig. 1b), and chronic irritation also are predisposing factors.

Host Factors.—Persons with fair complexion and blue, gray, or green eyes are much more sensitive to the damaging effects of sun and have a much greater incidence of squamous cell epithelioma, basal cell epithelioma, and malignant melanoma than do persons with dark complexion and brown eyes.

Genetic predisposition to skin cancer also exists. Xeroderma pigmentosum and albinism are two recessively inherited conditions; common to both are light sensitivity, dry skin, photophobia, large freckles, keratotic patches, and a tendency to form skin cancers.[5] Hair and eye pigmentation is normal in xeroderma pigmentosum whereas there is almost complete lack of pigmentation of hair and eyes in albinism. Development of skin cancer occurs much sooner and is much more extensive in xeroderma pigmentosum than in albinism. Patients with xeroderma pigmentosum have basal and squamous cell epitheliomas as well as malignant melanomas, whereas patients with albinism usually have only squamous cell epitheliomas. Skin cancer is rare among albino persons living in the temperate zone if proper precautions are taken for protection against sun exposure.

Precancerous Dermatoses

Precancerous lesions of the skin can exist for many years without apparent change or with slow change. Histologically, these lesions may exhibit carcinoma in situ without showing clinical evidence of malignancy. Invasive cancer, usually squamous cell epi-

Clinical aspects of premalignant and malignant dermatoses and the environmental and host factors contributing to their development are reviewed. Primary emphasis is on diagnosis, but general principles of treatment are outlined.

DONALD D. HARVILLE, M.D.
ROBERT R. KIERLAND, M.D.

thelioma, eventually develops in 20 percent of such lesions.[6]

Bowen's disease is a precancerous dermatosis which histologically shows carcinoma in situ. The lesions occur with equal frequency on exposed and unexposed skin, and are more often single than multiple. They consist of sharply demarcated, polycyclic plaques with various degrees of erythema and scaling. There may be ulceration. These lesions are often confused with those of psoriasis or other papulosquamous dermatoses. Graham and Helwig[7] reported that the median diameter in 212 lesions of Bowen's disease in 155 patients was 1.3 cm. Lesions of Bowen's disease tend to enlarge slowly or to remain unchanged, but invasion and metastasis may occur after many years.

Actinic keratoses occur exclusively on exposed areas of the body or on areas previously exposed to ionizing radiation. These lesions histologically show carcinoma in situ with a picture similar to that of Bowen's disease. Persons with light complexion, especially those who work out of doors, are especially susceptible. Actinic keratoses are scaly, verrucous, or horny plaques with varying degrees of erythema and brownish pigmentation. Most lesions are from 0.5 to 2 cm. in size, and they arise on weatherbeaten or chataigne skin. Cutaneous horns are an exaggerated expression of such keratoses.

Patients who develop *arsenical keratoses* have frequently had arsenic treatment previously in the form of Fowler's solution, usually for conditions such as asthma and certain benign dermatoses. Arsenical toxicity may also result from exposure to chemicals containing arsenic. Arsenical changes may occur on exposed and unexposed areas but keratoses have a predilection for palms and soles. These lesions, except for those on the palms and soles, are clinically similar to those of Bowen's disease. Graham and Helwig[7] are of the opinion that all cases of Bowen's disease may be caused by arsenic ingestion or absorption. There is an increased incidence of internal malignancy in association with the arsenical keratoses. Carcinomas of the bronchus and of the urinary bladder are the most common internal malignancies.

The lesions of *leukoplakia* consist of white, irregular, usually thickened plaques which may or may not ulcerate (Fig. 1c). They may occur on the buccal mucosa, palate, tongue, or vulva. Frequently, the area of leukoplakia has been a site of chronic irritation, such as may occur with malocclusion of the teeth or poorly fitted dentures. Leukoplakia frequently occurs at the site where a pipe is held in the mouth and excessive smoking seems to contribute to its development. Leukoplakia of the tongue frequently occurs after syphilitic glossitis (Fig. 1d).

The lesions of *leukoplakic vulvitis* have essentially the same appearance as those of leukoplakia in the mouth. The cause is unknown. Lesions may occur on the labia minora, clitoris, inner aspects of the labia majora, and, less commonly, in the vagina. Leukoplakia is sometimes superimposed on kraurosis vulvae (a phase of lichen sclerosus et atrophicus), but kraurosis vulvae per se is not a premalignant condition. Both kraurosis vulvae and leukoplakia are more common after menopause, and it has been suggested that hormonal deficiency may contribute to their development.

Histologically, the lesions of leukoplakia are similar to those of actinic keratosis and

may show a picture of carcinoma in situ. The lesions have the same potential for becoming malignant as do those of actinic keratosis.

Erythroplasia of Queyrat is manifested by a dry, velvety, red, sharply demarcated plaque on the glans penis, with or without extension onto the inner aspect of the prepuce. The lesion occurs almost exclusively

Fig. 1. a. Chataigne skin with actinic keratoses and squamous cell epitheliomas.
b. Squamous cell epithelioma arising in a burn scar.
c. Leukoplakia of the vulva.
d. Leukoplakia and squamous cell epithelioma arising in syphilitic glossitis.
e. Squamous cell epithelioma arising de novo.

in uncircumcised males. Histologically, a picture of carcinoma in situ similar to that of Bowen's disease is seen.

Malignant Neoplasms

Squamous cell epithelioma may arise de novo (Fig. 1e) or from precancerous dermatoses, such as actinic keratosis, arsenical keratosis (Fig. 2a), and Bowen's disease. In general, squamous cell carcinoma arising de novo tends to be more malignant in its course than does squamous cell epithelioma arising from precancerous lesions or keratoses. Clinical differentiation between a precancerous dermatosis and invasive squamous cell epithelioma may be difficult or even impossible. For this reason, skin biopsy must be relied on for the answer. More advanced lesions of squamous cell epithelioma are usually ulcerated with infiltrated, woody, pearly borders. The border is wider and more infiltrated and inflammatory than is the border of basal cell epithelioma. There may be severe inflammation and secondary infection. Regional nodes should be checked carefully for metastases. Exposed areas, especially the lower lip, cheek, ears, nose, upper extremities, and forehead, are common sites of squamous cell epithelioma.

Adenoid squamous cell epithelioma (adenoacanthoma) is a histologic type of squamous cell epithelioma which tends to form

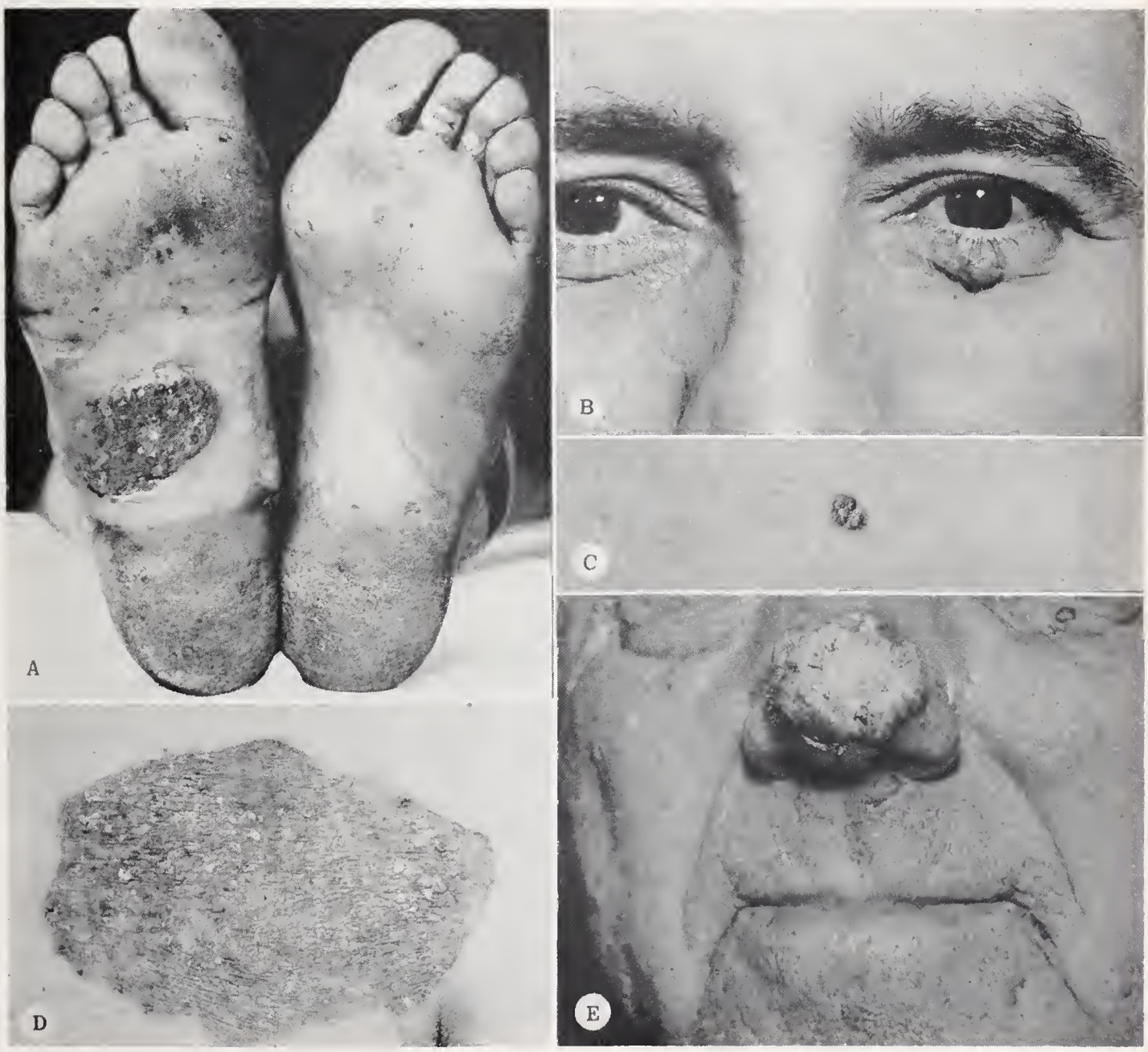

Fig. 2. a. Squamous cell epithelioma (right sole) and multiple keratoses in patient previously treated with arsenic.
b. Basal cell epitheliama of the eyelid.
c. Pigmented basal cell epithelioma resembling malignant melanoma.
d. Superficial epitheliamatasis.
e. Keratoacanthoma.

pseudoglandular structures, and to metastasize by the perineural lymphatic vessels. Local recurrences of the tumor are common after excision.[8,9]

Basal cell epithelioma causes damage by local extension and destruction of tissue. Metastasis is rare, having been reported in less than 100 cases, and, when it occurs, usually only involves regional lymph nodes. Clinically, basal cell epithelioma occurs in several morphologic types of skin lesions. The earliest lesion is usually a pearly, translucent nodule which slowly enlarges (Fig. 2b). Ulceration (rodent ulcer) frequently develops in the larger lesions. Basal cell epitheliomas may also appear as shiny, flat, grayish or yellowish plaques which have a somewhat sclerodermoid appearance (morphea form). Another type of lesion is a flat, scaly plaque with a thin, pearly, serpiginous border (superficial epitheliomatosis). Some basal cell epitheliomas are pigmented and resemble melanoma (Fig. 2c).

In *superficial epitheliomatosis,* multiple, erythematous, scaling plaques with fine, pearly borders occur over the skin, but in contrast to the usual basal cell epithelioma, they occur on unexposed skin (Fig. 2d). These may vary from a few centimeters to palm size or larger. There is usually some scaling and crusting and, frequently, some superficial ulceration. Such lesions often occur after the ingestion of arsenic.

The *basal cell nevus syndrome* is a dominantly inherited condition in which there are multiple nevoid basal cell epitheliomas, cysts of the jaw, and congenital anomalies of the ribs, spinal column, and skull.[10] Basal cell epitheliomas usually appear about the time of puberty and behave much as do other basal cell epitheliomas. The central part of the face is the most common site of involvement, but the lesions occur anywhere on the body. There may be few or hundreds of lesions; their aggressiveness is greater in adulthood than in childhood. Disfigurement often results from damage to the eyelids and other facial tissues. A peculiar type of punctate pit (0.5 to 1.0 mm. in diameter) occurs on the palms and soles of patients with the basal cell nevus syndrome.

There is considerable controversy as to whether or not a basal cell epithelioma ever transforms into a squamous cell epithelioma. Clinically, most lesions which have been classified as basal-squamous cell epithelioma have had the clinical features of basal cell epithelioma. Lesions having histologic features suggestive of squamous cell epithelioma are more likely to metastasize than are those with histologic features of basal cell epithelioma.

Keratoacanthoma, a skin tumor which has been recognized only for the past 14 years, probably is not a true malignancy. Two types occur: (1) hereditary (multiple self-healing epitheliomas of Ferguson and Smith), and (2) acquired lesions, mainly on exposed areas and in association with premalignant and malignant dermatoses.[11] The familial type of keratoacanthoma occurs at an earlier age, frequently by puberty, and may develop on unexposed as well as exposed areas. Clinically, the lesions are similar in both types. They begin as hemispheric papules which enlarge to 1 or 2 cm. in four to five weeks. The skin over the lesion is shiny and smooth with a central umbilication which is covered by a crust (Fig. 2e). When the crust is removed, a central horny plug is seen. Most keratoacanthomas will heal spontaneously, if left alone, in two to six months. Some of the lesions will recur if excised.

Whether keratoacanthoma ever transforms into squamous cell carcinoma is not settled. Experimental keratoacanthoma-like lesions in animals frequently do transform into invasive carcinomas.[12] Histologically, keratoacanthoma closely resembles low-grade squamous cell carcinoma. However, the overall pattern is distinctive.

In *malignant melanoma* (Fig. 3a, b and c), early diagnosis is of paramount importance since 61 percent of patients survive for five years when their lesion is discovered when it is less than 2 cm. in diameter.[13] On the other hand, only 16 percent of patients survive five years when their lesion is larger at the time of diagnosis. Approximately 80 percent of malignant melanomas are said to arise in a preexisting nevus. Clues to diagnosis of malignant melanoma include recent onset, recent growth, or recent darkening of a "mole." Variation of color pattern also points to probable malignancy. Irregular configuration with spreading border, especially when satellite nodules are present, is indicative of malignant melanoma. Lesions which can be confused with malignant melanoma inlcude early keratosis, pyogenic granuloma, benign nevus, and post-traumatic hemosiderin deposits, especially when the latter are located under nails.

The question of what to do about benign

nevi (moles) always arises. Routine removal of nevi from the palms and soles is no longer considered necessary.[14] A lesion on the palm or sole should be removed only if its features suggest malignant melanoma. Benign nevi elsewhere should be removed only if they are subjected to chronic irritation, or for cosmetic reasons. The routine excision of benign nevi is impractical since the average adult has 15 to 20 pigmented nevi and removal would be time-consuming for all physicians.[15] Furthermore, the incidence of melanoma per mole is only approximately 1.8 per 1,500,000.[15]

Spindle cell and *epithelioid cell nevi* (juvenile melanoma) usually occur before puberty, although they also occur in adults, and the face and lower extremities are the most common sites. The lesions vary from a few millimeters to 3 cm. in diameter, and usually are isolated, red-brown, hairless papules or nodules (Fig. 3d) which may resemble angioma or pyogenic granuloma. Most of these lesions were formerly diagnosed as malignant melanomas, and this accounts for the reported high incidence of cure of malignant melanoma in children. Malignant melanoma in children has the same prognosis as it does in adults.

The *melanotic freckle of Hutchinson* (lentigo maligna) is a dark-brown to black, macular lesion occurring mainly on the face of older patients. It tends to be stable for many years or to enlarge slowly. The lesion needs no treatment unless it is raised or nodular areas appear within it, the latter being evi-

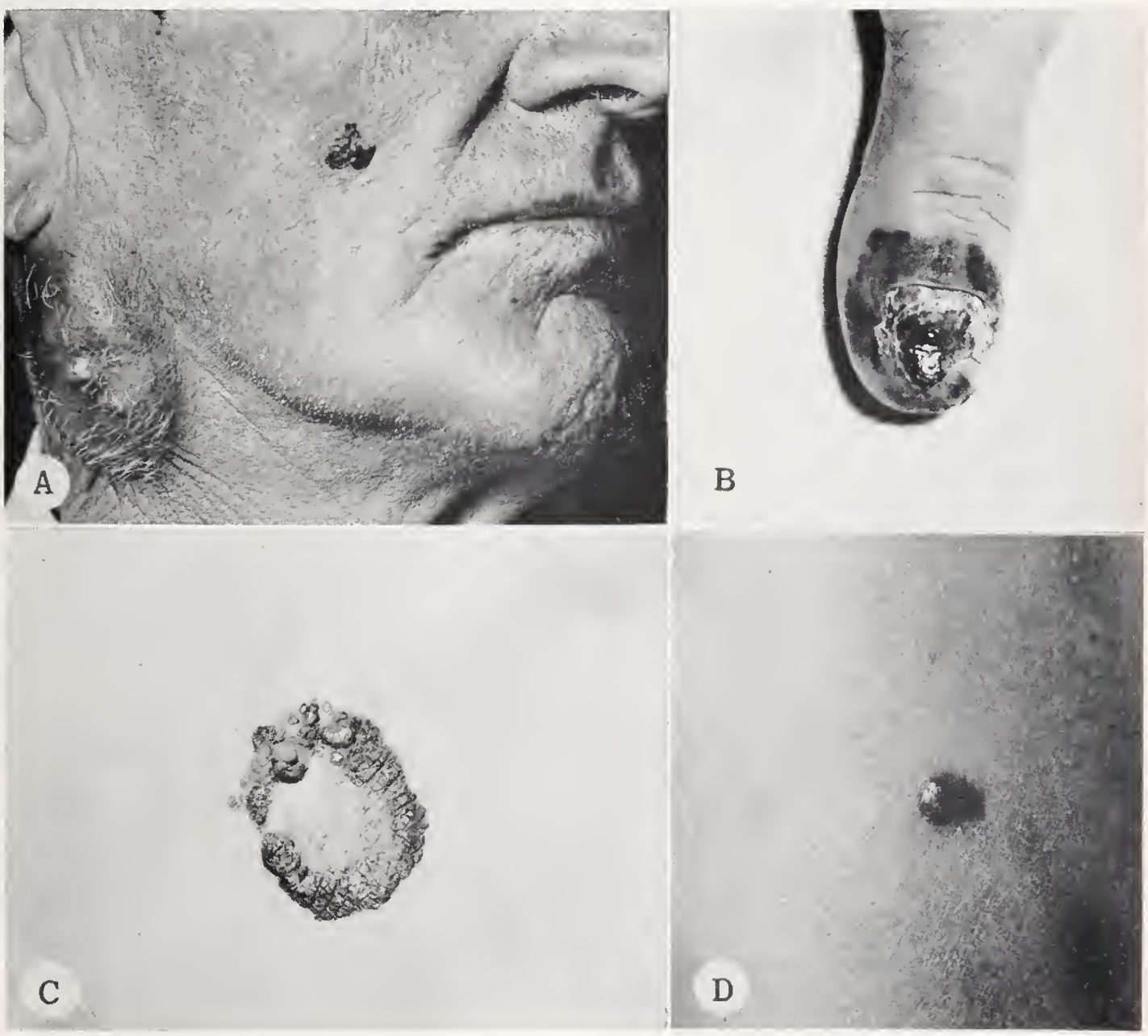

Fig. 3. a. Malignant melanoma of the face with cervical nodal metastasis.
b. Melanotic whitlow.
c. Malignant melanoma on upper arm.
d. Juvenile melanoma on arm.

dence of a malignant transformation. Even when malignant melanoma develops in this lesion, the prognosis is relatively good.

Paget's disease of the breast consists of an eczematous or scaling, erythematous, usually sharply marginated plaque involving the areola of the nipple and, later, the surrounding skin[16] (Fig. 4a). There may or may not be a palpable mass in the involved breast, but histologic evidence of ductal carcinoma is found almost universally. Radical mastectomy is the indicated treatment. Biopsy should be done in the operating room to facilitate surgical treatment immediately after diagnosis is made from the frozen section.

Extramammary Paget's disease has a clinical appearance similar to that of mammary disease, but the lesion may be more vegetative and macerative (Fig. 4b) due to its location in the intertriginous areas. It most frequently is seen in the groin and perineum; less often, the axillae, lower part of the abdomen, thighs, and buttocks may be involved. There has been considerable controversy as to whether malignant cells arise in the epidermis or elsewhere. In an analysis of 58 cases of extramammary Paget's disease, Graham and Helwig[7] found that in 55 per-

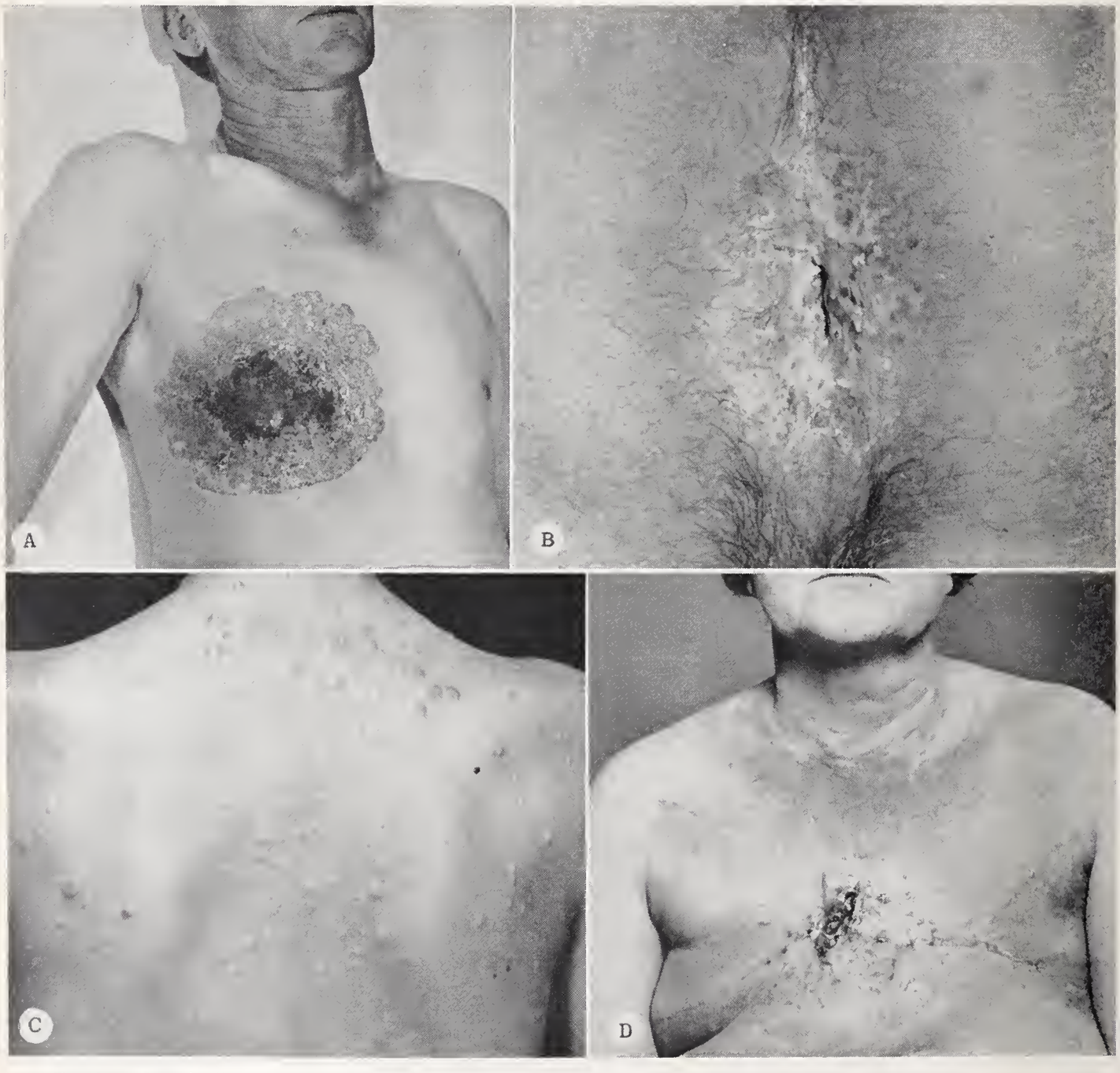

Fig. 4. a. Paget's disease in a man.
b. Extramammary Paget's disease involving perianal area.
c. Multiple nodules of metastatic adenocarcinoma from an unknown primary site.
d. Carcinoma erysipelatoides involving mast of chest around mastectomy scars.

cent there was carcinoma of the skin adnexa, regional carcinoma (usually in the rectum), or carcinoma elsewhere.

Both mammary and extramammary Paget's disease tend to be confused with eczema. Eczematoid lesions in those areas where Paget's disease occurs should be biopsied if there is any doubt of the diagnosis.

Carcinoma of the dermal appendages, except in association with Paget's disease, is rare. Carcinomas of the sebaceous glands and sweat glands do occur occasionally.

Of all cutaneous cancer, 1 to 3 percent is metastatic from underlying internal cancer (Fig. 4c). Involvement may be by direct invasion or by lymphatic or vascular spread. The skin is occasionally involved by metastasis from cancers of almost any organ, but carcinoma of the breast is the most common metastatic malignancy involving the skin. Metastatic lesions in the skin may take several clinical forms. Nodular lesions vary from a few millimeters to several centimeters in size, and frequently have a translucent appearance with varying degrees of telangiectasia. Indurated scleroderma plaques of various sizes (a few centimeters to large sheets) also occur, and may be stony hard. Perhaps the most elusive type of metastatic involvement of the skin is the inflammatory type known as carcinoma erysipelotoides because it closely mimics erysipelas (Fig. 4d). This lesion consists of a cellulitis-like, irregular area of erythema. There is usually some increase in warmth and there may be some degree of induration.

Sarcomas are relatively rare in comparison to the previously described epidermal malignancies. Sarcomatous degeneration of neurofibromas (von Recklinghausen's disease) has been said to occur in from 5 to 16 percent of cases, but the actual incidence is difficult to estimate.[17] Other types of sarcomas involving the skin are fibrosarcoma, liposarcoma, and rhabdomyosarcoma. Clinically, sarcomas usually appear as subcutaneous, rapidly growing, hard masses.

Dermatofibrosarcoma protuberans usually begins as a firm, fibrous plaque which may remain unchanged for years but which eventually enters a phase of rapid growth to form a multinodular mass.[18] The tumor spreads by local extension and is difficult to eradicate surgically. Local recurrences are frequent but metastasis is rare. Histologically, the lesions are often confused with low-grade fibrosarcoma.

Kaposi's sarcoma is a multicentric malignant neoplasm which tends to progress into angiosarcoma. Bluefarb[19] discussed the nature of the disease extensively. Clinically, the lesions are symmetrically arranged, bluish to reddish nodules or plaques appearing most commonly on the feet and legs, but also occurring anywhere on the body and in internal organs as well. Lesions may undergo spontaneous regression. Multiple foci of the disease arise simultaneously in widely separated areas of the body.

The *Stewart-Treves syndrome* refers to lymphangiosarcoma in a lymphedematous extremity. It occurs most commonly in an edematous arm after radical mastectomy, but also develops in the lower extremities in association with lymphedema of various causes. Clinically, skin nodules appear and may resemble metastatic carcinoma. It is important to distinguish lymphangiosarcoma from metastatic carcinoma because amputation of the involved extremity is indicated in the former, whereas it would be contraindicated in the latter.

All of the *lymphomas* and *leukemias* may have manifestations in the skin.[20] These are of two types: (1) nonspecific, in which the lesions are the result of systemic toxicity of the disease without infiltration of the skin by the malignant cells of the specific disease, and (2) specific, in which the skin is infiltrated by the malignant cells of the specific disease. Nonspecific skin manifestations run the gamut from mild to severe generalized pruritus to generalized erythroderma and exfoliative dermatitis. Urticaria, purpura, bizarre bullous eruptions, eczematous eruptions, prurigo nodularis, and even herpes zoster may occur. Perhaps the most familiar is the severe pruritus which may accompany Hodgkin's disease. Specific lesions of the lymphomas are papules, nodules, or tumors which usually are pink to plum-colored (Fig. 5). These may occur anywhere on the body. Skin lesions may appear before any systemic sign of disease develops; therefore, they are important in diagnosis.

Mycosis fungoides is a somewhat controversial condition which nevertheless has clinically recognizable patterns. The disease may be confined to the skin for many years without affecting the patient's general health and without involvement of internal organs, or it may undergo change into malignant lymphoma with involvement of internal organs and a rapid demise. In a fully developed case there is a multiplicity of morphologic lesions, including faint, scaly, poor-

ly defined macules; infiltrated erythematous plaques; and frank tumors with or without ulceration (Fig. 6). Diagnosis is made by punch biopsy, preferably of an infiltrated plaque or tumor. Several biopsies may be required since the lesions may be in different stages of development and not all may show the characteristic histologic pattern.

Treatment

Histologic diagnosis is essential to the proper treatment of skin cancer; a punch biopsy or, in some cases, a shave biopsy is usually satisfactory. Sometimes, multiple biopsies are necessary to establish a diagnosis. If a malignant melanoma is suspected, an excision biopsy should be done or, if the lesion is too large for excision biopsy under local anesthesia, the patient should be taken to the operating room and a punch or wedge biopsy be taken for frozen-section examination. If malignant melanoma is found, the lesion can be surgically excised with an adequate border. It has been suggested, but not proved, that punch biopsy may spread a malignant melanoma unless the lesion is then immediately excised.

The several modalities for the treatment of skin cancer give equally good results, and the therapist should use the one with which he is most experienced. Surgical excision, electrodesiccation, chemosurgery, chemotherapy, surgical planing, radiotherapy and radium implantation all have application to the treatment of certain types of skin cancer. Lesions near the eyes and nasolabial folds are usually best managed with radiation therapy. Bowen's disease and superficial epitheliomatosis are best treated by surgical excision since they respond poorly

Fig. 5. a. Leukemia cutis. b. Hodgkin's disease with nodular infiltrations in the skin. c. Lymphosarcoma.

to radiotherapy. Most actinic keratoses and arsenical keratoses can be successfully treated by electrodesiccation and curettage.

Malignant melanoma is resistant to radiation therapy, and wide surgical excision with regional lymph node dissection is the treatment of choice. If the lesion is on an extremity, regional perfusion with a chemotherapeutic agent may be indicated.

Paget's disease of the breast is an indication for radical mastectomy; extramammary Paget's disease is best treated by surgical excision.

Sarcomas in general are radioresistant. The best hope for cure is in cases in which the lesions are on the extremities; in these cases amputation is usually indicated.

X-ray or electron-beam therapy is the treatment of choice for lymphomas which involve only the skin. Leukemias and lymphomas involving the internal organs are usually best treated by systemic chemotherapy.

The treatment of mycosis fungoides depends on the stage of the disease. Prior to the development of tumors and infiltrated plaques, it is usually best treated nonspecifically and symptomatically with topical application of steroids and antipruritic lotions and with ultraviolet light; such treatment may suffice for many years. When the disease progresses to the stage of infiltrated plaques or tumors or both, radiation therapy to local lesions or whole body irradiation is usually indicated.

Fig. 6. Mycosis fungoides.

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Giant Pigmented Nevus With Vitiligo

CASE REPORTS

ABOUT 200 CASES of giant pigmented nevi have been reported, though more exist. To my knowledge, the following is the first reported case of giant nevus within which are areas of vitiligo. Both pigmentary disorders are relatively uncommon and apparently are unrelated to each other, but the appearance of vitiligo within the nevus raises questions as to its significance or influence.

Case Report

Since birth, a seven-year-old Indian boy has had an elevated brown-to-black lesion covering a large area of his left shoulder and left upper back (Fig. 1). This lesion is covered with coarse, dark hairs. The involved area has remained the same size in proportion to the child, and has not changed appreciably since birth except for the sudden appearance of depigmented areas within the past year. They have also appeared on other parts of the body (scalp, trunk, and extremities). The depigmented areas have continued to increase in number and size since their onset. The nevus and the areas of depigmentation are asymptomatic. The child has no unusual illness and is on no regular medications. No other members of his family have any known pigmentary disorders or illnesses.

The nevus is 15 cm. in greatest diameter; and some areas are darker than others. Many of the round, oval, and irregular depigmented (hypopigmented) areas within this nevus and elsewhere on the body have the characteristic band of hyperpigmentation on the peripheral edges that are seen in vitiligo. In most areas of vitiligo, including those within the nevus, there is depigmentation of the hairs. This results in the striking appearance of light-colored hairs surrounded by darker hairs within the nevus. In a few areas, especially within the nevus, small islands of hyperpigmentation occur within the vitiligo (Fig. 1). The largest area of vitiligo is on the lower border of the nevus, and is the most irregular in outline.

Otherwise, the patient is a medium-to-dark complexioned boy who is in good health. There is no local or general adenopathy and no other pigmentary or nevoid changes are seen.

Giant Pigmented Nevus

Giant pigmented nevus (nevus pilosus, bathing trunk nevus, large hairy nevus) is a pigmentary disorder present at birth, and most commonly affects the shoulders, back, or buttock; however, any cutaneous surface may be involved. Most of the nevi are rather heavily pigmented and are covered, to some extent, with excessive, usually dark, coarse hair.

The lesions do not regress spontaneously and may undergo a malignant transformation to a melanoma. In most of the cases reported, the incidence of malignancy is greater than 10 percent. This percentage is relatively high, since usually only cases in which a melanoma occurs are reported. An incidence of 1 or 2 percent may be more realistic.[1] Melanoma may develop in these nevi at any age. For this reason, most authorities recommend removal of these growths if possible. However, a giant pigmented nevus can involve such a large area of the body that removal becomes technically impossible.

Giant pigmented nevi may be associated with other disorders, many of them having features of von Recklinghausen's disease (multiple neurofibromatosis). Other types of tumors such as neurofibroma, fibroma, and lipoma, may occur within the nevi or elsewhere. Other pigmentary changes such as cafe-au-lait spots and areas of hyperpigmentation may develop on scattered areas of

A boy, age 7, has had a giant pigmented nevus on his shoulder and upper back since birth. Recently, patches of vitiligo have developed within the nevus and elsewhere on his body. Coexistence of these lesions has not been reported previously.

JOHN L. KESTEL, JR., M.D.

the body; however, no mention has been made of associated depigmentation. Bony changes, such as certain hypertrophic changes, and those of spina bifida with meningocele, have also been reported. Enlargement of an involved extremity and atrophy of the muscles have also been associated with this condition. Neurological changes, occasionally present, have been explained by involvement of the leptomeninges, especially when the giant nevi occur on the head and neck. There are 20 reported cases with this type of involvement of the skin and leptomeninges.[2]

Fig. 1. Dark, coarse hairs cover most of the giant nevus on the patient's back. The light areas within the nevus, on the back of the neck, and elsewhere on the body are vitiligo. Many hairs in the vitiligo patches are white.

Vitiligo

Vitiligo is a pigmentary disorder characterized by well defined areas of depigmentation which vary in size and shape. While vitiligo may involve any part of the body, it appears most frequently on exposed parts, especially the hands. Usually a dark brown band at the edge of the lighter areas gives a definite contrast between the patch of depigmentation and the surrounding normal skin. This dark border is not found when depigmentation results from other causes such as inflammation. An area of vitiligo may not be complete, resulting in partial depigmentation within the involved portion. Hair in the involved spots may or may not be white. Other family members may be affected, suggesting hereditary influence; the genetic pattern is not constant but at times suggests a dominant gene.

On microscopic examination, areas of vitiligo show a lack of melanin in the epidermis. Melanocytes are present in normal numbers, but lack the ability to produce melanin. This is in contrast to the nevus, in which there is an increased number of melanocytes in the epidermis, dermis, or both. Melanin (as granules) is usually increased, causing the dark appearance of most nevi (Fig. 2).

Vitiligo is a benign condition and little or no therapy is generally recommended. Varying degrees of repigmentation may follow the

prolonged oral administration of psoralens; trimethylpsoralen, one of the newer forms, seems to be more potent and less toxic than others.

Summary

A case of a giant hairy nevus on the back has been presented because of the sudden appearance of vitiligo within the nevus and on other body areas. Vitiligo has not appreciably changed the nevus from a practical standpoint. Nevus cells are still present in the areas of vitiligo that have developed within this pigmented growth. The only alteration produced by the occurrence of vitiligo within the nevus is diminished pigmentation.

Treatment recommended is surgical excision. Although no proof exists, treating the vitiligo with trimethylpsoralen might induce malignant change in the nevus. If malignancy should develop, this drug could be implicated as a causative factor. Treatment of the vitiligo should be deferred until the nevus has been entirely removed.

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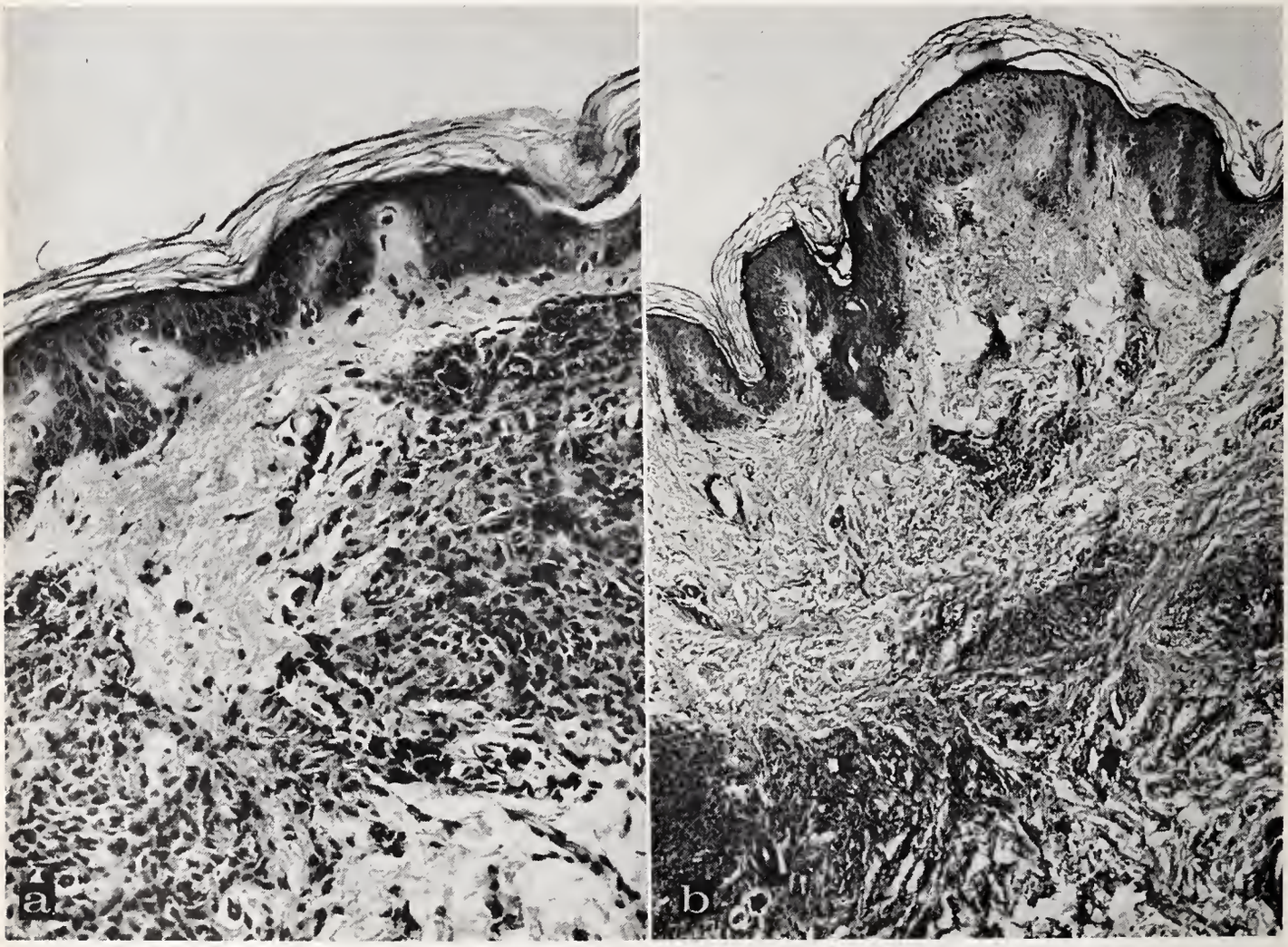

Fig. 2. a. Photomicrograph of the giant nevus tissue shows accumulations of nevus cells in the dermis, especially in its middle and lower portions. Considerable melanin (dark-staining small granules) is seen in the dermis, as opposed to its relative absence in vitiligo tissue. X 206.

b. Tissue from an area of vitiligo within the giant nevus, and adjacent to the specimen shown in Fig. 2a, shows the nevus cells present in the dermis, but considerably less pigment (melanin) is seen. X 78.

Surgical Improvement of Collateral Circulation to the Brain

Robert H. LePere, M.D., and Robert C. Hardy, M.D.

A palliative surgical procedure to increase collateral circulation to the hemicerebrum after occlusion of the major blood supply gave clinical improvement in two cases. It might be considered for patients having complete occlusion of the internal carotid artery and severe stenosis of the external carotid on the affected side.

CLINICAL MEDICINE

MANY TECHNICAL procedures have been devised for increasing the cerebral vascular circulation; most deal directly with the vascular obstruction.[1] The best results surgically have been achieved when the vascular occlusion is extracerebral and incomplete. Early removal of complete obstructions of the extracranial vessels gives good results in a fair proportion of cases. Generally, however, when the complete obstruction is of long duration little surgical help has been offered to these patients. The following reports describe a somewhat different technique which gave two patients considerable benefit.

Case Reports

Case 1.—Three weeks before being seen, a 50-year-old teacher had blurring of vision and paroxysmal blindness limited to the right eye. This had been associated with severe pressure headaches, generally in the occipital region, lasting up to an hour after the attacks. Bilateral carotid angiography indicated a complete block of the internal carotid artery on the right just at the bifurcation. There was narrowing of the external carotid artery on that side (Fig. 1a). There was only minimal narrowing of the internal and common carotid artery on the left side.

Surgery was performed on Oct. 10, 1962. The internal carotid artery was completely thrombosed and no backflow could be obtained upon opening the vessel. However, it appeared that if the circulation through the external carotid artery could be improved, then we might expect increase in the collateral circulation through the ophthalmic branch into the internal carotid artery intracerebrally. Thus the external carotid artery was opened longitudinally and a pinpoint opening observed in the vessel immediately at its origin. A long, oblique patch of knitted Dacron was oversewn in the incision extending from the common carotid well up into the external carotid. A good pulsation was obtained in the external carotid artery following the onlay graft.

The patient was much improved, though not completely relieved. She is teaching again, though she still has symptoms of cerebral vascular insufficiency. Bilateral carotid angiography was performed again in January, 1963, and showed a good lumen in the external carotid artery (Fig. 1b), and an increase in collateral blood flow.

Case 2.—A 54-year-old man had numbness in the right side of the face, transient diplopia, and numbness of the fingers of the right hand. His symptoms were of recent origin. Arteriography Oct. 22, 1962, showed complete occlusion of the left internal carotid artery above the bifurcation. The external carotid artery on the left was narrowed just at the bifurcation (Fig. 2). The opposite carotid artery was normal in the neck, but arteriosclerotic changes were noted intracranially. The vertebral arteries were normal. The patient underwent surgery on Oct. 24, 1962. The internal carotid artery was completely blocked distal to the bifurcation. No backflow could be obtained. The external carotid artery was patched with a long, oblique segment of knitted Dacron and a good pulsation was obtained distally in the vessel

(Fig. 3). This patient, too, has considerable relief of symptoms. Bilateral angiography repeated in January, 1963, showed good filling bilaterally. On the left side there was improvement of the circulation via the external carotid artery and its collaterals (Fig. 4). The intracerebral arteriosclerotic changes remained constant on the right. This patient was benefited, though his symptoms were not completely relieved. He has had recurrence of similar symptoms on the opposite side, and has further evidence of intracerebral arteriosclerosis on that side.

Discussion

The ophthalmic artery is a branch of the intracerebral internal carotid artery. It enters the orbit via the optic foramen with the dural sheath of the optic nerve (Fig. 5). It supplies the contents of the orbit, giving off a central artery to the retina, and a posterior ciliary artery or arteries, anastomosing with the anterior ciliary branches derived from muscular branches. Six branches of the artery pass beyond the orbit. These six arteries—the supratrochlear and the supraorbital to the forehead, the dorsal nasal to the face, the lacrimal to the eyelid and via zygomatic branches to the cheek and the temporal region, and the anterior and posterior ethmoidal to the nasal cavity—anastomose freely with branches of the external carotid artery.[2]

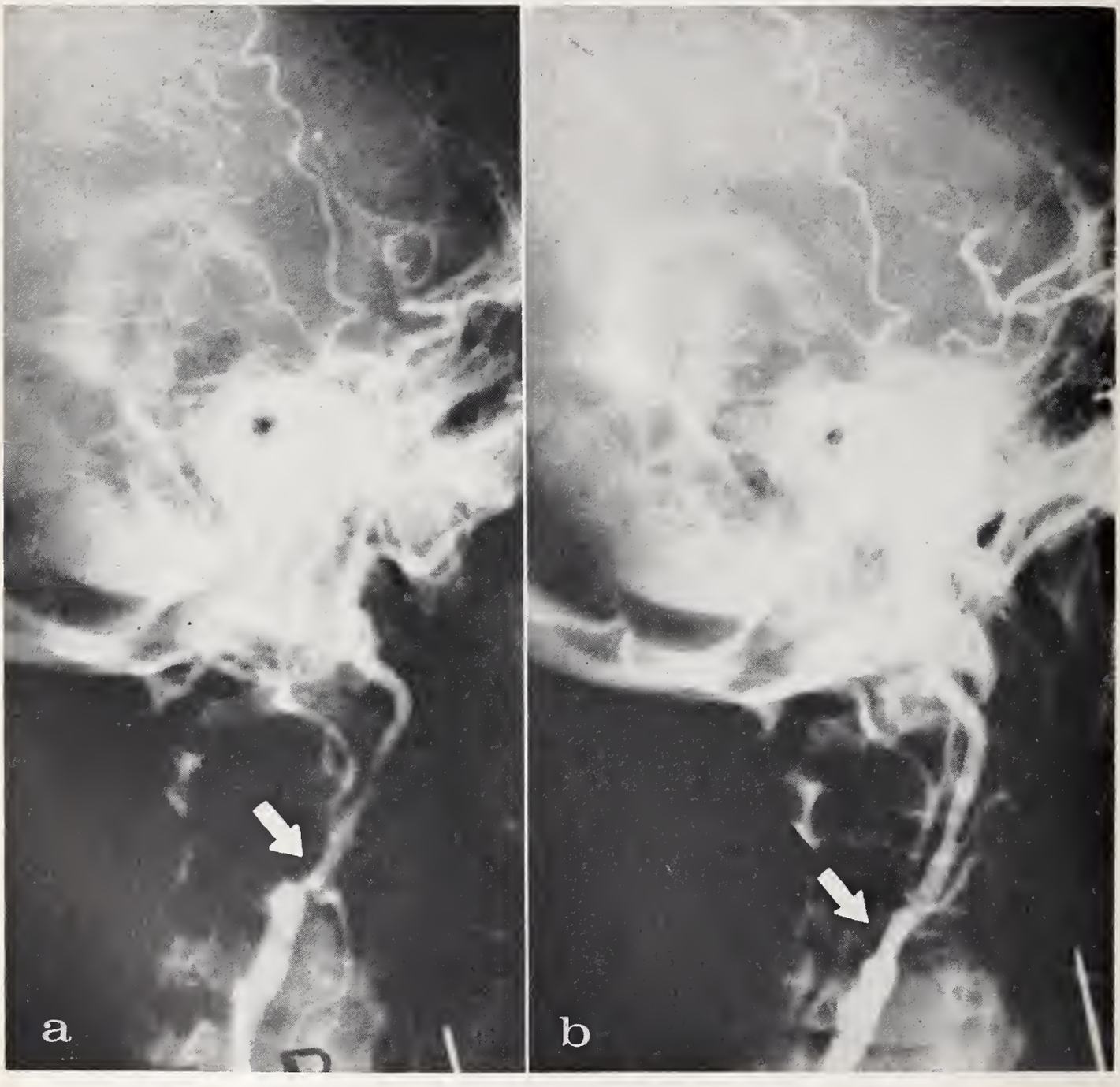

Fig. 1a. Preaperative arteriagram, case 1, revealing complete acclusion af the internal caratid artery on the right, and stenasis af the external caratid.
b. Arteriagram dane three manths pastaperatively, case 1.

Fig. 2. Preoperative arteriogram, case 2, revealing complete occlusion of the internal carotid on the left with narrowing of the external carotid.

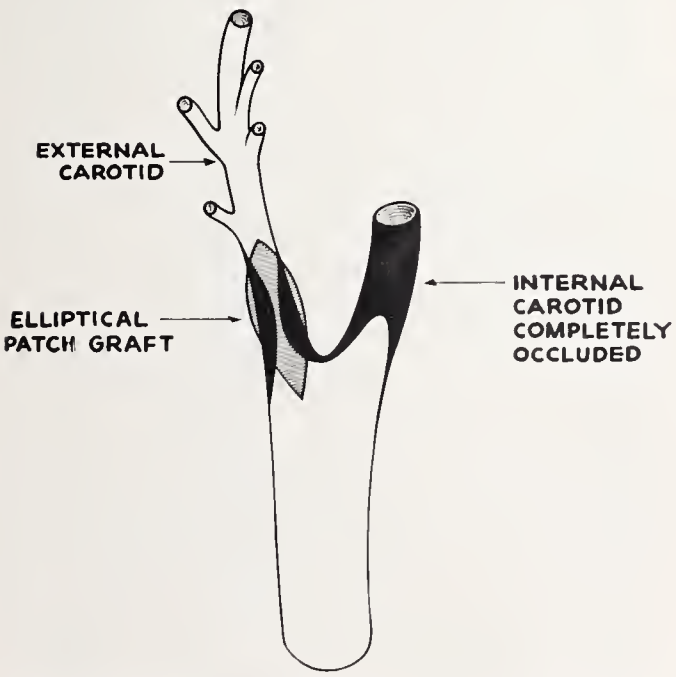

Fig. 3. Diagram of external carotid patch graft procedure performed on both patients.

Fig. 4. Arteriogram three months postoperatively, case 2.

It is recognized that the application of patch grafts to the external carotid artery is limited in scope, finding application only wherein the internal carotid is permanently obstructed in association with an external carotid artery which is narrowed more than about 60 percent. Increasing the blood flow through the external carotid artery theoretically should provide a significant collateral circulation through the ophthalmic branches. In cases of sudden, complete occlusion of the internal carotid with resultant symptoms, providing increased collateral blood flow in this manner also might give temporary help to a patient with a precarious blood supply to the brain while other collateral channels are developing.

ADDENDUM: A third patient, one with

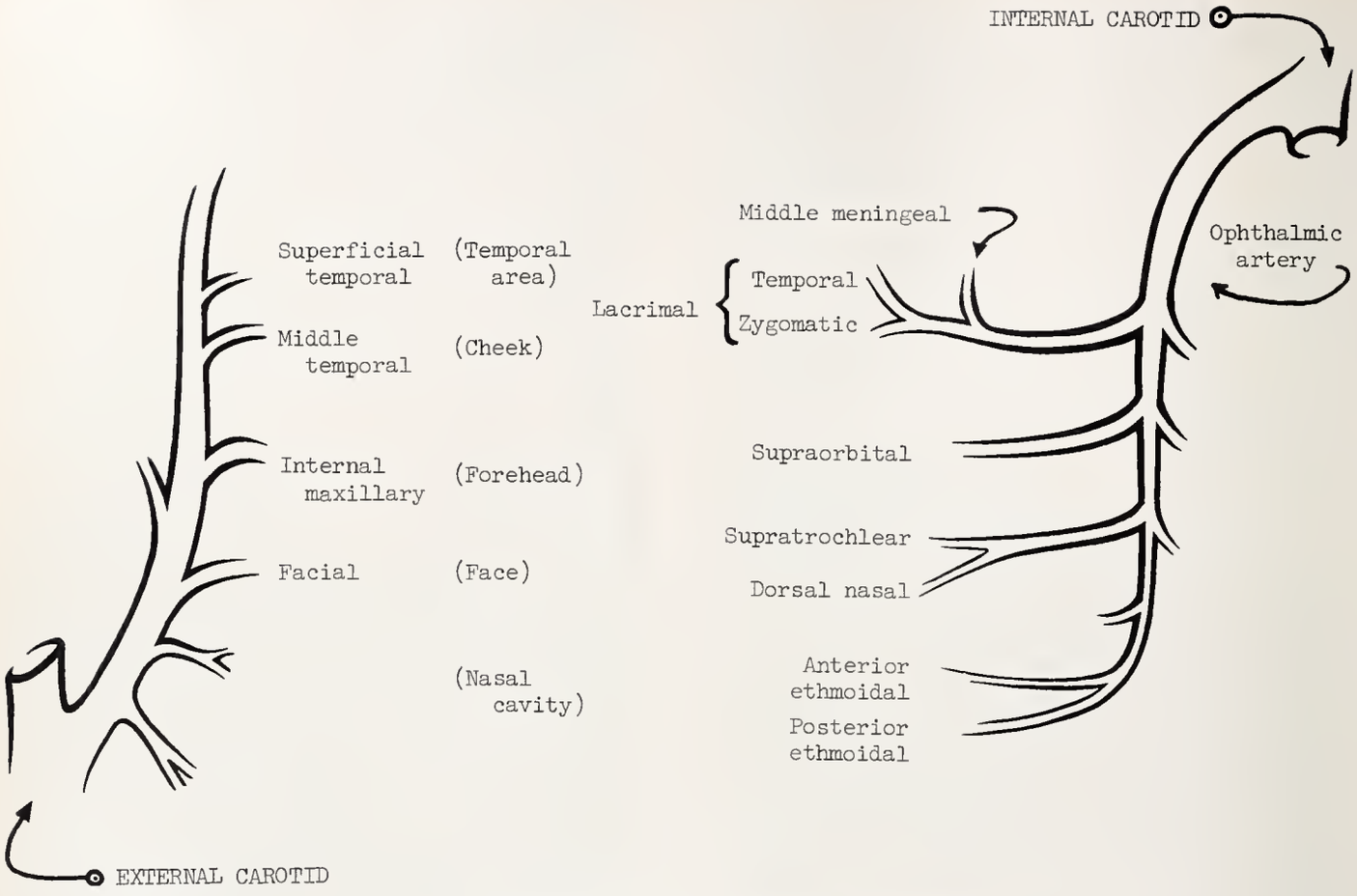

Fig. 5. Ophthalmic-external carotid artery anastomosis.

complete bilateral internal carotid artery occlusion and bilateral stenosis of the external carotid arteries, has been benefited following bilateral application of the external carotid patch grafts.

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Pacemakers Not Influenced by Outside Electric Current

Heart patients who have been fitted with implantable electronic pacemakers need not fear that the pacemaker will be affected by proximity to diathermy machines, neon signs, household appliances, radios, TV sets, or other electrical or electronic apparatus that generate radio frequency emissions, reports the National Heart Institute.

Researchers said that implantable electronic pacemakers manufactured and commercially available in the United States have been repeatedly tested and found completely free of susceptibility to outside radio frequency interference. The electronic design and the natural shielding afforded by body tissues prevent any external radio frequency sources from affecting the pacemaker's performance.

Mechanical Pacemaker Failure

Robert H. LePere, M.D., and Paul Klinger, M.D.

During 40 months, 16 cardiac pacemakers were implanted in 11 patients. Multiple complications required 28 surgical procedures to correct disruption of pacing. One patient died shortly after an operation to replace a unit. It is believed that pacemaker implantation with intracavitary bipolar electrode is the present procedure of choice.

CASE REPORTS

TO IMPLANT AND maintain 16 mechanical pacemakers in 11 patients who had complete heart block, 28 various procedures were required in 40 months. One patient required three pacemakers (Fig. 1) and three patients required two each. There were 16 instances of disruption of the mechanical stimulus, causing recurrence of the original symptoms and requiring reoperation of some type before pacemaker-induced stimulation of the heart could be obtained (Table 1). To date, the longest uninterrupted pacemaker function in any one patient has been 40 months.

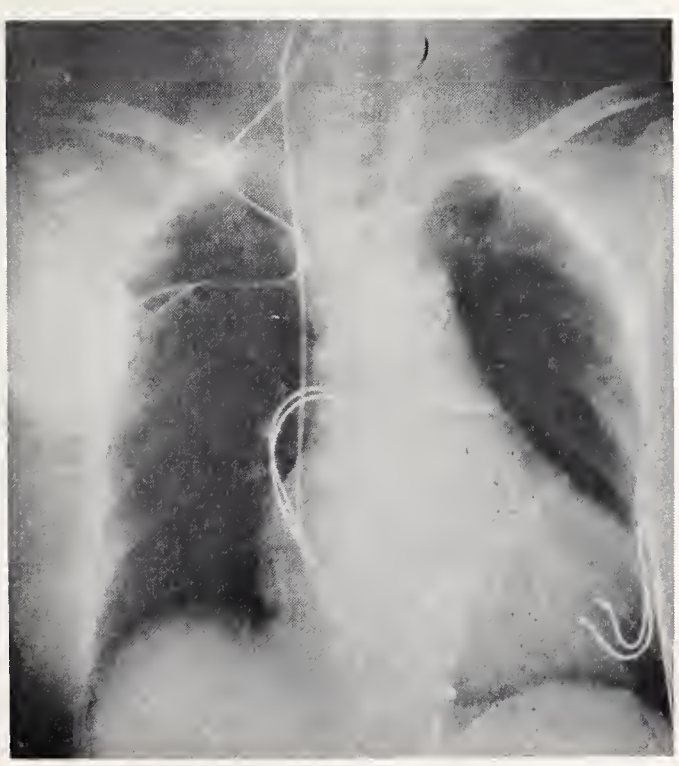

Fig. 1.

Broken Electrodes

Broken electrodes caused disruption of heart pacing in three instances. In two other patients whose pacemakers had been replaced previously, fracture of the myocardial portion of the residual inactive electrodes was seen on roentgenograms.

One year postoperatively, a 73-year-old man dropped a church bench across his abdomen, breaking one, or probably both, electrode wires and causing the resumption of Stokes-Adams attacks. A complete new unit was implanted; it has been trouble-free for 27 months.

In a 64-year-old woman, disruption of both electrodes was discovered about three months postoperatively (Fig. 2). Both electrode wires were tightly coiled around the battery in its abdominal pocket. When the abdominal pocket was opened to remove the broken wires and battery, both electrode leads sprang out. This woman was the most obese in the group, and her unusually pendulous abdomen swayed as she walked. Undoubtedly the pacemaker battery unit twisted with each swaying motion until the electrodes were torn apart. The electrocardiogram

TABLE 1.—*Complications Requiring Re-operation.*

Broken Electrodes (requiring complete new unit)	3
Electrode Insulation Disruption (requiring complete new unit)	1
Battery Component Failure (requiring new battery)	4
Increasing Myocardial Resistance (requiring increased amperage)	4
Insulation Repair and Adjustments	4
Total	16

(Fig. 3) revealed good amperage of the pacemaker stimulus without initiation of the QRS complex. In comparison with the postoperative electrocardiogram, the stimulus is of higher amplitude following disruption.

A 70-year-old man had intermittent impulse failure occurring with position change 20 months following implantation of his first unit. A roentgenogram showed sharp angulation in two areas of the myocardial portion of the electrodes and a fracture across one electrode and possibly the other (Fig. 4). A new unit was implanted, however, the patient died in his sleep on the sixth postoperative day. Postmortem examination revealed plaque occlusion of the left anterior descending coronary artery.

Fractures in the myocardial portion of the dead electrodes have shown up in follow-up roentgenograms two years (Fig. 5) and three years (Fig. 6) following implantation of the patients' first pacemakers. Both had to have replacement pacemakers previously for other causes.

Fig. 2.

Broken electrodes are reported as a frequent cause for pacemaker failure, and in some instances have resulted in fatalities.[1-6]

Electrode Insulation Disruption

Eight months following implantation of her second cardiac pacemaker, a 72-year-old woman had sudden recurrence of Stokes-Adams attacks. The insulation about the electrode wires was filled with tissue fluid. The leak could not be located, and a new battery failed to pace with the shorted electrodes. This required a complete new pacemaker—the patient's third.

Fluid leakage into the insulation of both electrodes was also seen at postmortem examination in the one patient who died. It is not believed that the pacemaker failed prior to death, though the leakage probably would have caused failure in the future. Nicks were seen in the insulation of both electrodes near the area of exit from the thoracic cage, and were probably cut on a sharp edge of a rib.

Battery Component Failure

Loss of pacing occurred in four instances, one of which was probably accompanied by increased myocardial resistance. The battery units required replacement at 16, 13, 11, and 7½ months. The battery failures occurring at 13 and 7½ months were in units in which the amperage previously had been doubled.

A 70-year-old man had resumption of Stokes-Adams attacks 11 months postoperatively. The electrocardiogram revealed typical failure of the pacemaker stimulus to induce the QRS complexes. The battery was replaced and the patient has remained symptom free. Inspection of the battery revealed change in characteristics of the 4.7 capacitor.

The woman in whom the battery component failed twice has had three pacemakers implanted over a two-year period. Because of increased resistance and the need for having to double the amperage previously, when the battery failed at 13 months, a complete new unit was implanted. Examination of the battery by engineers at Medtronic, Inc. indicated that the difficulty lay in failure of the output capacitor. Later, the battery of her third unit had to be replaced at 7½ months due to component failure not yet specifically identified. It is to be noted that the amperage of this unit was doubled nine days after its implantation.

There was one case of runaway pacemaker occurring 16 months after implantation in a 71-year-old woman. When removed, the battery had fluid beneath the silastic seal. A new battery corrected the situation, but during the period of runaway pacemaker, the electrocardiogram (Fig. 7) showed an intermittent rate varying from 40 to 180 per minute associated with anginal pain and dyspnea during the bouts of ventricular tachycardia. The QRS complexes follow even at the most rapid rates of stimulation. Engineers at Medtronic, Inc. reported failure of the oscillator transformer. In this unit the amperage had been doubled 14 months earlier.

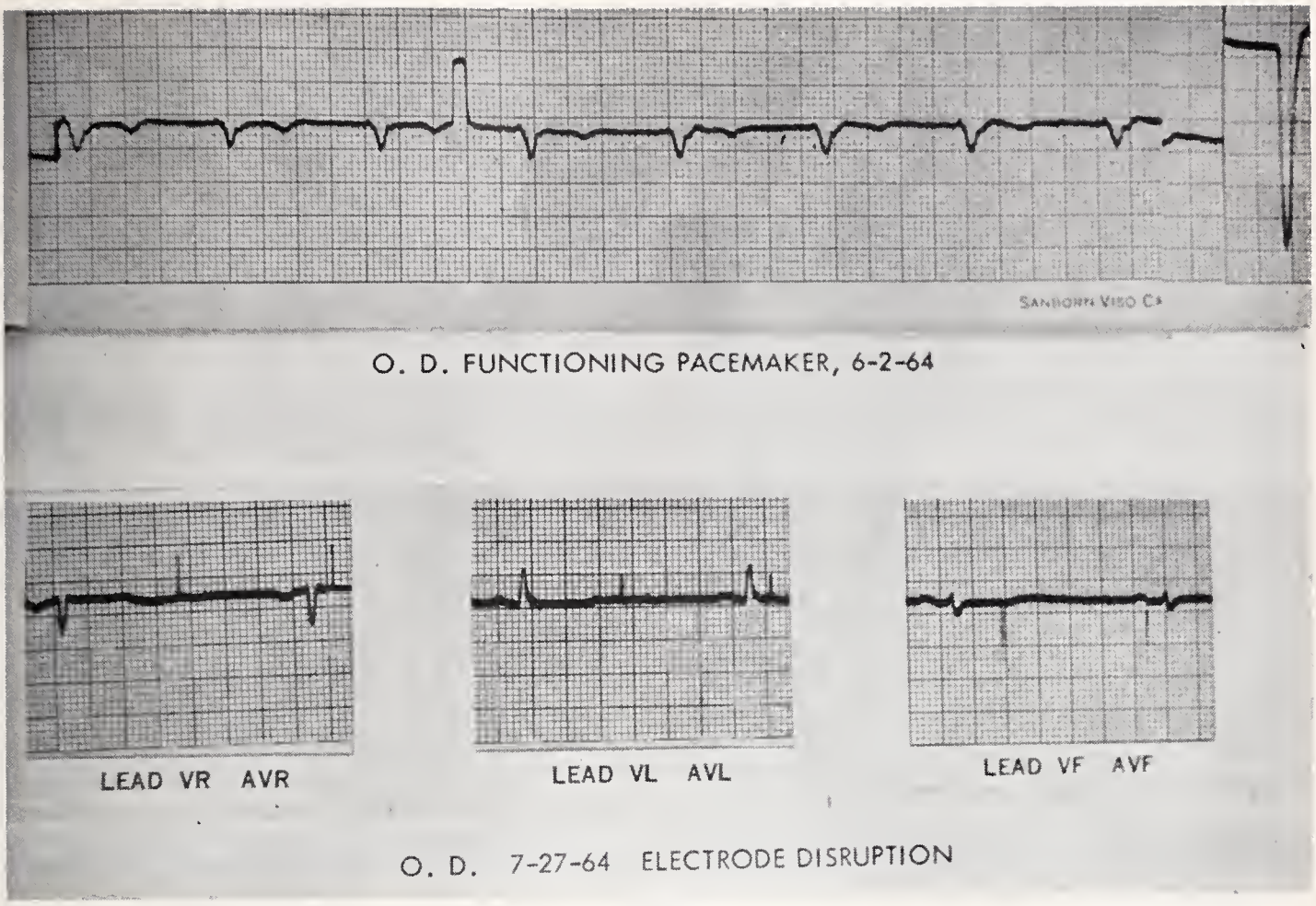

Fig. 3.

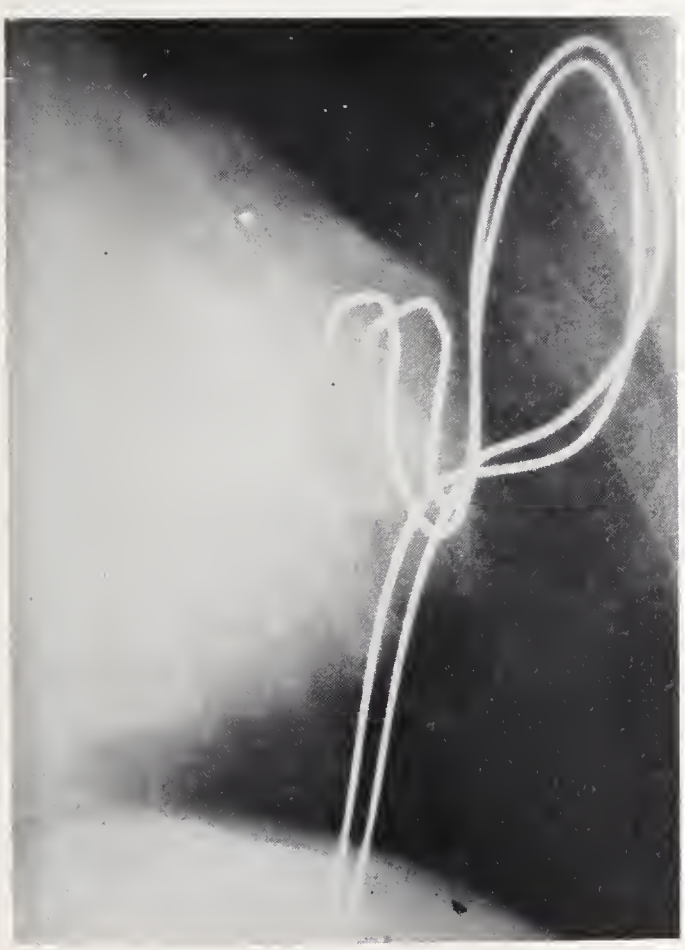

Fig. 4.

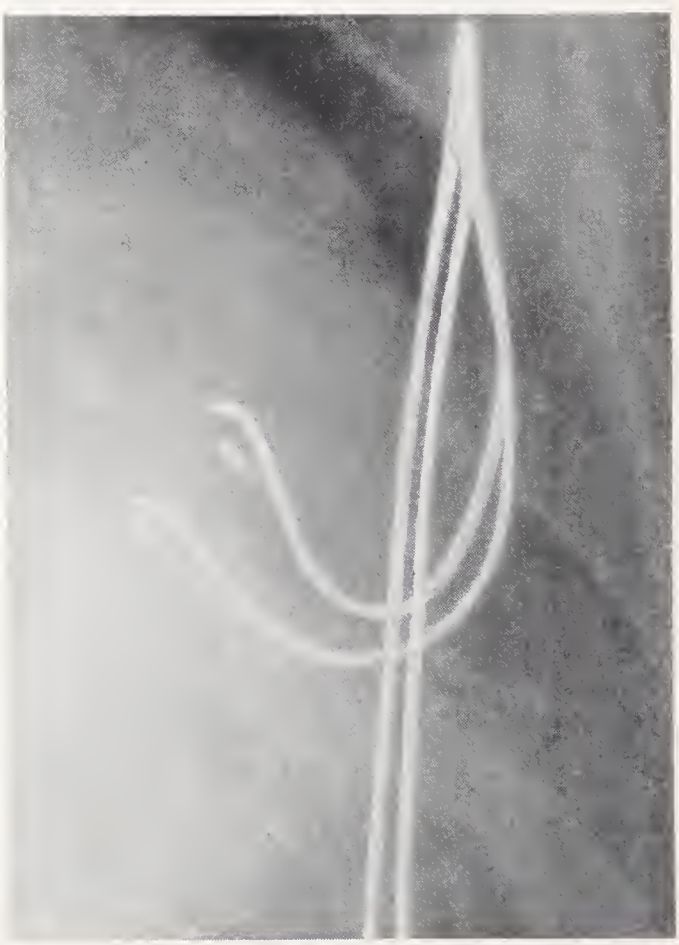

Fig. 5.

Failure of the power unit has been reported.[1,3,6-8] Seven cases of arrhythmia with three fatalities are reported by Spear and associates.[4] One of these was a pacemaker tachycardia.

Fig. 6.

Increasing Myocardial Resistance

In four instances, cessation of pacemaker function was attributed to increasing myocardial resistance which was corrected by doubling the amperage.

One instance, which is questionable, was attributed to increasing resistance one year following implantation in which response was obtained by doubling the amperage. However, 26 days later the pacemaker failed again, and a complete new pacemaker was implanted since it seemed doubtful that battery replacement only would have corrected the situation. The engineers at Medtronic, Inc. suggested that failure of the output capacitor was the source of trouble. After 22 days, the second pacemaker failed as a result of increasing myocardial resistance, but it responded to doubling of the amperage.

Another instance of increasing myocardial resistance occurred in a 71-year-old woman 18 days following implantation, and responded to doubling of the output. Another occurred four months after implantation in a 72-year-old woman whose symptoms were corrected again by doubling the amperage.

According to Dressler and Jonas,[9] in the weeks following implantation of the myocardial electrodes an inflammatory reaction progresses to the formation of scar tissue. This increase in resistance to impulse passage is associated with a steadily increasing refractory time, both reaching a plateau

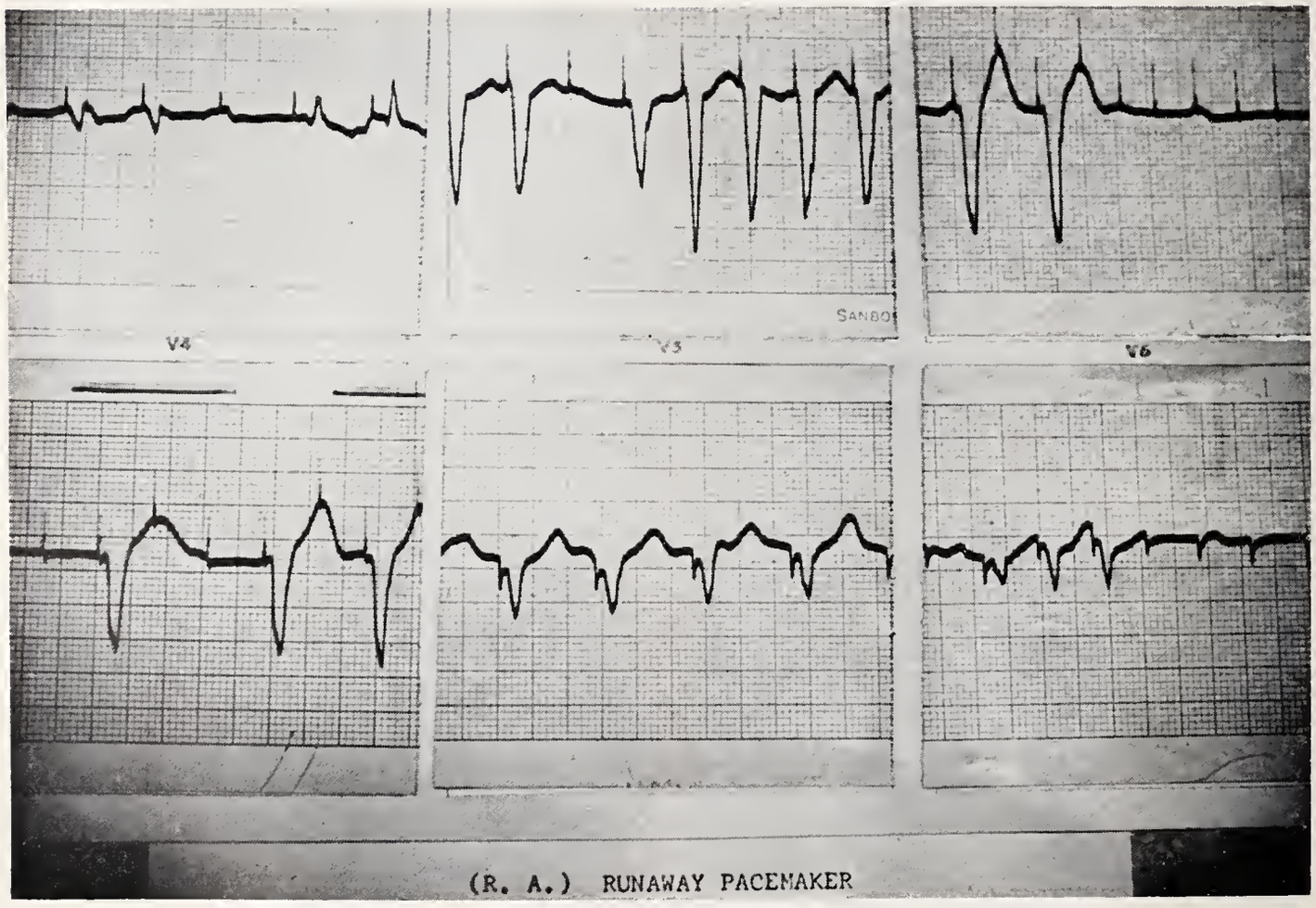

Fig. 7.

about three weeks postoperatively and lowering the current available to stimulate the myocardium. Increasing resistance at the myocardial electrodes is reported, though less frequently, with present pacemakers.[1,2,4,7]

Insulation Repairs and Adjustment

One pacemaker failed due to leaking of the insulation which was reapplied following doubling of the amperage in the safety extension of the battery. There were two instances of loss of position of the bipolar intracavitary right ventricular lead with an axillary unit, occurring in the same patient one and seven days postoperatively. Additional loss of pacing on the tenth day was corrected by doubling the amperage.

In this patient, pacemaker failure ensued six days following doubling of the amperage by shorting out the resistance in the safety extension. The patient was rehospitalized and the safety extension was exteriorized; it contained fluid which short-circuited the pacemaker. This was remedied by drying and re-insulating the extension.

A third pacemaker (axillary implantation with intracavitary catheter) was implanted in the same patient, and there was loss of position of the intracavitary electrode at 24 hours with resultant interruption of pacemaker stimulation. This required reoperation with repositioning of the catheter under fluoroscopic guidance. The same thing happened seven days postoperatively, and the procedure was repeated. At ten days postoperatively, loss of pacing again occurred, and was corrected by doubling the amperage. The patient has had her intracavitary implant for ten months without further difficulty except for battery replacement at seven and one-half months.

Sowton and Davies[7] reported that malposition of the catheter electrode was the usual cause of pacemaker failure, requiring repositioning once in every 18 patient months.

Other Complications

Several instances of potentially serious complications did not require pacemaker repair.

A 70-year-old woman with the history of heart block for five years complicated by ankle edema, dyspnea, angina pectoris, and numbness in the left arm had intermittent complete heart block and runs of sinus rhythm on repeated electrocardiograms. A fixed-rate cardiac pacemaker was implanted March 18, 1963, with doubling of the amperage required April 16, 1963, because of loss of pacing with ECG evidence of continued function. Afterward, the pacing improved but there were occasional periods of bradycardia in the range of 40 to 50 beats per minute, and occasional periods during which the patient's own pacemaker would take over. Maintenance doses of corticosteroids gave fairly good response until July 23, 1964, when the patient was hospitalized with the runaway pacemaker. The battery was replaced, and the patient has been asymptomatic with constant artificial pacemaker QRS response.

Dreifus and Uricchio[10] reported a patient who developed postoperative atrial flutter with a pulse of 280 per minute and four-to-one ventricular response with pacemaker stimulus at 60 per minute. The second and sixth beats were fusion complexes, suggesting competition between the patient's own and the artificial pacemaker stimulus. The atrial flutter was controlled by digitalization. Dressler and Jonas[9] reported 11 instances in which the pacemaker stimuli combined with the patient's to form a ventricular parasystole.

A 64-year-old woman had heart block for six years and syncopal attacks for five years. Acute cholecystitis and severe ileus complicating congestive heart failure developed 48 hours following implantation of her pacemaker. She was treated medically, and after a prolonged convalescence she recovered.

A 72-year-old man had external pacemaker electrodes in place for four days preoperatively, causing necrosis of two areas of the chest wall. A postoperative infection cleared on intensive antibiotic therapy and did not interfere with the pacemaker function.

The patient who required three pacemakers nearly died following the first implant. She had had syncopal attacks and congestive heart failure with hypertensive cardiovascular disease and angina pectoris for three years. At the time of her operation she was having eight to ten Stokes-Adams attacks daily. After the first pacemaker implantation April 3, 1963, intestinal obstruction developed, complicating congestive heart failure. This was treated medically, and cleared with gastrointestinal suction and symptomatic treatment. She sustained myocardial infarction, and one week postoperatively an embolus lodged in the right brachial artery and another in the left internal carotid siphon. She was treated conservatively with anticoagulants and regained all function with the exception of the pulse deficit in the right brachial artery. She has made a complete recovery from multiple procedures and is able to be up and about unaided.

The one fatality was a 70-year-old man whose second pacemaker was implanted June 6, 1965, 20 months following his first. There was fracture of the myocardial electrodes seen on roentgenogram, and it is suspected that there may have been battery component failure as well. Roentgenograms revealed moderate exhaustion of the mercury cells (Fig. 8). On the sixth postoperative day he was found dead. Autopsy revealed a plaque dissection with occlusion of the left anterior descending coronary artery.

Glass and associates[11] reported a case in which hypotension developed when cardiac rates above 50 per minute were utilized. This patient died on the eighth postoperative day and was found to have bilateral staphylococcal lung abscesses and pyelonephritis.

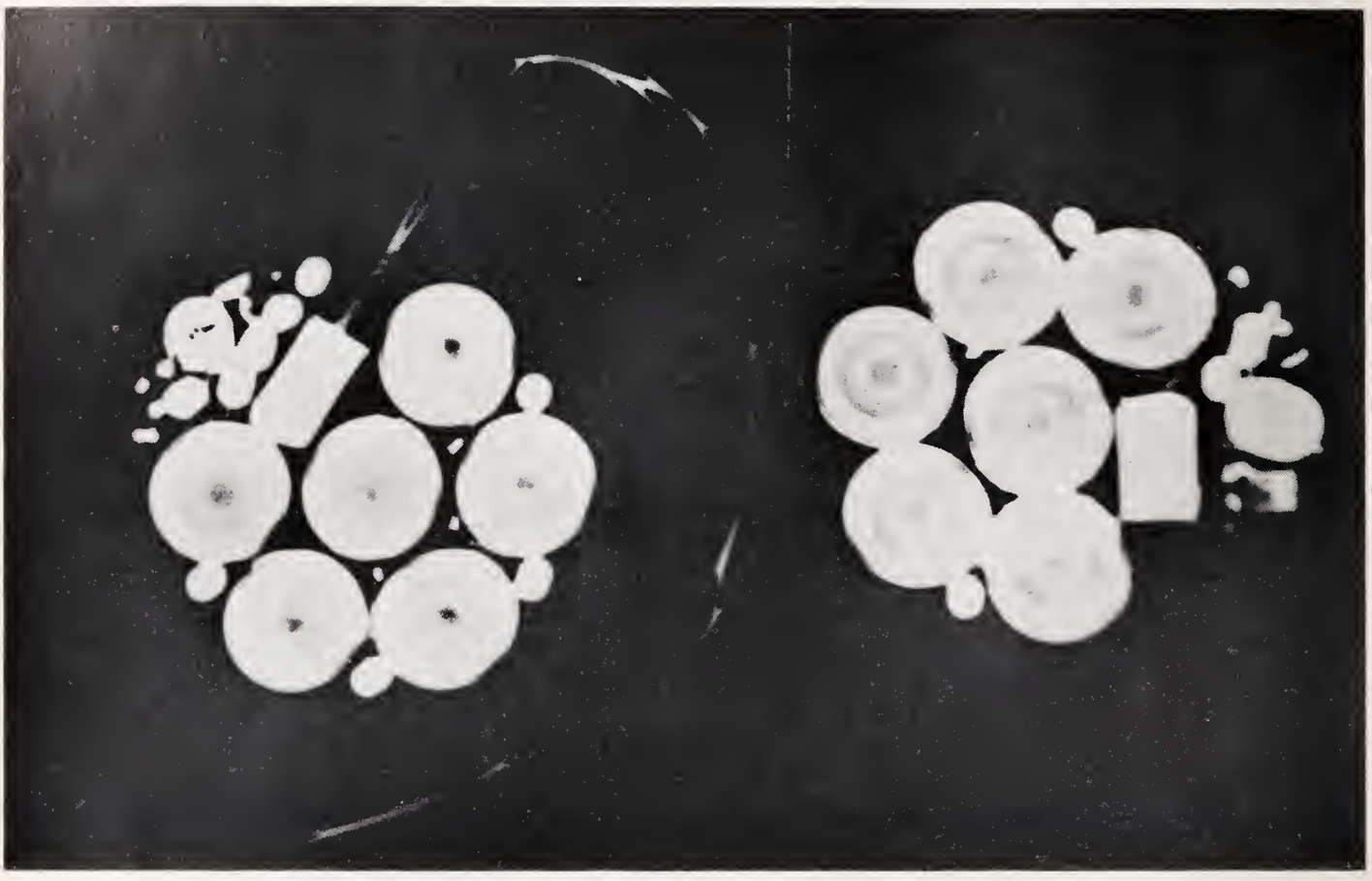

Fig. 8. Simultaneous x-ray of used battery (left) compared with a new one.

Tavel and Fisch[8] reported ventricular tachycardia and what appeared to be ventricular fibrillation associated with three postoperative syncopal attacks. The artificial pacemaker stimulus was 58 per minute. When the rate was increased to 75 per minute, the ventricular tachycardia ceased.

Norman and others[3] reported 15 deaths during a 32-month interval, with eight occurring in the first two weeks. One occurred from malposition of intracardiac pacemaking catheter. One was caused by myocardial infarction one day postoperatively. On the second day, one patient died from a collapsed left lung. On the second postoperative day one patient died from bronchopneumonia and pulmonary insufficiency. At seven days, ventricular tachycardia was presumed to be the cause of one death. At three days, six days, and two weeks, deaths occurred from undetermined causes. One death occurred three months postoperatively due to broken electrode, and another, four months postoperatively, was caused by pulmonary infarction with subacute endocarditis. One death four months postoperatively was believed to have resulted from mechanical pacemaker failure. Causes of two deaths, at 5 and 11 months were undetermined.

Trimble and others[12] reported one death from ventricular fibrillation on the fifth day, probably on the basis of coronary artery disease, and one death at seven months, etiology unspecified.

Spear and others[4] reported six deaths in 29 months. Three died during the early postoperative period, two of ventricular fibrillation, and one of perforation of a stress ulcer. Three late deaths were associated with postoperative pacemaker tachycardia, cerebral vascular accident, and bronchopneumonia.

Potassium Levels

Serum potassium levels were obtained generally before the first pacemaker implantation and at the time of pacemaker failure. Potassium was given in each case in which the level seemed below normal, but in no instance did it benefit the patient enough to cause resumption of pacemaker stimulation. Potassium levels varied from 3.7 to 8.5 mEq. per liter. The latter potassium determination was repeated and is presumed to be accurate; it was drawn preoperatively from an 85-year-old woman.

Type of Pacemaker

All the pacemakers utilized to date have been Medtronic, Inc. models 5860 (fixed

rate) and 5870-C (adjustable rate and amperage). There is a safety extension off the body of model 5860 which permits the resistance to be shorted out, doubling the amperage. This has proved useful on several occasions. Additionally, the safety extension can be exteriorized and used for determination of pacemaker amperage and, on occasion, determination of nonfunctioning electrode.

Under local anesthesia, three pacemakers have been implanted in the axilla with an intracavitary right ventricular bipolar catheter. This method was utilized for a woman 85 years old, for a patient aged 72 who was a poor cardiac risk, and for the obese woman of 65 years who had twisted off her abdominally implanted pacemaker.

The other 13 pacemakers were implanted in the conventional manner, utilizing Medtronics model 5860 (and a thoracotomy to implant the myocardial electrodes). Twelve times the electrodes were sewn into the left ventricular myocardium, and once into the right. The right ventricular implant was done as a second procedure through a sternal splitting incision to avoid entering the pleura of a patient who was a poor cardiac risk.

Discussion

In 1951, Callaghan and Bigelow[13] reported pacing for cardiac standstill, and in 1952 Zoll[14] first used pacemaking for heart block. With the advent of the implantable pacemaker, new vistas were opened for the patient with complete heart block, but with certain limitations. The widely accepted units in clinical use today all have implanted power sources (mercury cell batteries, timers, capacitors) and wires connecting the power sources to the heart. The apparatus is being improved constantly, and the units available today are much superior to those formerly used. One manufacturer has reported a decrease in premature pacemaker failure from 15 percent prior to 1962 to less than one percent for current models. Considerable clinical and experimental work continues to contribute toward a better understanding of this subject.[1,8,14-17]

The intracavitary electrode may deserve wider usage. True, there are reported problems. Norman and associates[3] reported three perforations of the right ventricle in 20 patients with intracavitary pacing. However, at thoracotomy no evidence of intrapericardial bleeding was found. They reported one death from malposition of the intracardiac electrode. Sowton and Davies[7] mention perforation of the right ventricle as a complication, but apparently have had no fatality. Spear and others[4] reported three perforations in preoperative intracavitary pacing. The usual complication appears to be malposition of the catheter within the right ventricular cavity with resultant loss of pacing. These complications must be weighed against the obvious advantages: Thoracotomy and general anesthesia are avoided; convalescence is shortened; and the procedure carries little more risk than cardiac catheterization.

ADDENDUM: In the past six months, four additional patients required pacemakers (three were of the intracavitary type). Also, one of the reported patients, in whom a second complete unit already had been implanted, required a battery change in the unit two years and nine months after implantation.

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Cardiac Work Evaluation

REVIEW ARTICLES

OF THE 750,000 patients who have heart attacks each year in this country, a large number are not returning to work. For example, in Texas the Social Security Disability Program is now receiving 25,000 applications yearly from patients who are not yet age 65 and who feel that their cardiovascular disease is severe enough to warrant retirement and a disability pension. Forty-six percent of these patients are 55 years old and under, thereby representing ten years or more of productive life if properly returned to work. It is estimated that more than half of these applicants could be rehabilitated if appropriate action were taken.[1] Supporting this estimate are the findings that 80 to 85 percent of survivors of heart attacks can return to full-time work, 5 to 10 percent can return to part-time work, and only 5 to 10 percent must retire.[2,3]

Physicians today must be aware of the methods of work evaluation and also of ergonomics, the process of measuring the energy costs of activity. Recent studies provide the tools to (1) decisively manage the convalescent activities of the coronary patient, and (2) arrive at a clearly-defined work prescription. Blanket statements, that is, "only light work," or "occasional lifting of small objects," tell the patient and his employer little. Limits can be defined specifically.

With knowledge of work evaluation, anxiety, the primary side effect of myocardial infarction for both patient and physician, can be removed. The patient's fears must be allayed by his physician. The latter will control his own anxieties if he has the means to evaluate the physical capabilities of his post-coronary patient. Without this ability the doctor's own fear of doing damage will force him to direct resumption of activity at a pace slower than ideal and to set work limits that are too strict.

Cardiac Classification

In determining whether a patient can return to work, the most important aspect is classifying him according to the function and therapeutic classification of the American Heart Association (Table 1). A patient's classification establishes his prognosis for survival, for future employment, and helps determine a permissible level of physical activity (Table 2).[2] Furthermore, the classification represents an exact means of integrating the physician's impression of functional ability and the therapeutic implications.

An approximate cardiac classification, accurate in only 75 percent of cases, can be obtained from a careful history, particularly of exercise tolerance, and a physical examination.[4] Therefore, to provide an exact classification, objective exercise testing also must be performed. The history of exercise toler-

TABLE 1.—*Classification of Cardiac Patients.*

Functional Capacity	Therapeutic Class
I. Without limitation of activity. No symptoms.	A. No restrictions of ordinary activity.
II. Slight to moderate limitation. Symptoms with ordinary activity.	B. Restricted from severe or competitive efforts.
III. Moderate to great limitation. Symptoms with less than ordinary activity.	C. Ordinary activity moderately restricted.
IV. Unable to do any physical activity without symptoms. Symptoms at rest.	D. Ordinary activity severely restricted.
	E. Bed and chair rest only.

To help combat loss of manpower and economic drain in Texas because of heart attack, the author describes how a physician may evaluate capability to return to work.

DAVID J. TURELL, M.D.

ance, supported by simple objective tests, along with knowledge of on-the-job physical and emotional requirements, allows the development of a safe work prescription.

Energy Cost of Activity

The energy requirements of all human activity have now been determined in terms of calories per minute, the same kilocalories used in describing food values for diets. One calorie is equal to approximately 200 cc. oxygen consumption per minute. There are many articles which provide the caloric equivalent values for each activity.[5,6] Table 3 demonstrates the calories per minute required for various convalescent activities after a heart attack. A detailed history of home, avocational and leisure activities and correlation with calorie requirement quickly gives the physician an approximate level of energy expenditure below which the patient apparently can perform safely while at work (Table 3).

The reasoning for this approach is as follows: All activities with the same energy expenditure, in general, will evoke similar cardiac outputs, and cardiovascular responses and symptoms in the same subject. Therefore, if a patient can perform safely one activity at a given energy level, he can usually perform safely all other activities of equivalent caloric requirement, provided emotional and environmental stress are also the same. Emotional reactions to work are of utmost importance in evaluation; pulse and blood pressure responses to a tense situation may create as much heart effort as a brisk walk up a hill.[7] Of course there are other variables which have to be considered; for example, a grossly obese patient obviously will use more energy to do a job that requires movement of his entire body. The literature provides tables to estimate the caloric cost of moving body weight,[5] as well as other specialized tables, so that the cost of a job not specifically described can be estimated from knowledge of required body movements.

TABLE 2.—*Relationship Between Functional Capacity, Mortality, Work Capacity, and Employability.*

Functional Classification	I	II	III	IV
% Mortality* (6 year average follow-up)	25	31	53	60
Estimated work capacity—calories/minute.	4.0-6.6	2.7-4.0	1.5-2.7	1.0-1.5
% employed[2] (6 year average follow-up)	88.7	73.7	37.5	12.5

Exercise Testing

The parameters of caloric expenditure, oxygen consumption, pulse rate, minute ven-

TABLE 3.—*Energy Expended in Equivalent Work and Home Aetivities.*

Work	Calories per Minute	Home
Gauge setter	1.2	Sitting
Painter	1.4	Eating
Watch repairer	1.6	Knitting
Typist	1.8	Copper tooling
Turret lathe operator	2.3	Dressing or undressing
Welder	2.5	Washing, shaving
Foreman	3.4	Showering
Bricklayer	3.6	Using bedside commode
Plasterer	4.0	Walking (2.5 mph)
Wheelbarrower	4.7	Using bedpan
Gardener	6.0	Lawn mowing (power mower)
Shoveler	7.5	Lawn mowing (hand mower)

tilation, and cardiac output, are all linearly related between energy expenditures of two to ten calories per minute. Below two calories per minute and above ten calories per minute, these relationships are curvilinear. The two to ten calorie limit encompasses all work done and all amateur sports activity in the United States except for vigorous competitive sports. The linear relationships permit myocardial metabolism or cardiac work to be expressed in terms of pulse and systolic or mean blood pressure (tension-time index of Sarnoff).[8] These facts enable the private physician, in his own office, to do simple exercise testing. He may employ a standardized walk (walking at 2.5 mph requires an energy expenditure of four calories per minute), or a step test such as that employed by the Harvard Fatigue Laboratory or by Astrand;[9] or he can employ the Master "2-step" exercise test, but it must be remembered that this latter test is really a peak effort which involves eight and one-half calories per minute and may not be safe for all patients.[10] Pulse, blood pressure, and electrocardiographic changes must be observed. For the walk, pulse should not exceed 110 beats per minute and should return to normal in two minutes. For the Master test, safe responses are pulse rates less than 135-140 per minute and a systolic blood pressure increment of 10-40 mm. of mercury, with a return of these parameters to within 10 units of normal in two minutes.[11]

In an exercise laboratory, such as the Work Evaluation Unit at St. Joseph's Hospital, Houston, a more sophisticated approach is employed. The unit determines the upper limits of safe work performance without exceeding these limits during the tests, and at the same time obtains an estimate of a sizable cardiac reserve which has not been encroached upon. This requires an estimate of maximal performance which may be made by the use of pulse rate.[1] For every age range there is a predictable maximal pulse which can be achieved during maximal energy expenditure and maximal cardiac output.[9] The patient is exercised on a bicycle ergometer at levels of two, three, and four calories per minute (or three, four, and five) for four minutes during each exercise period. The pulse rate during the fourth minute is obtained and a graph is constructed relating pulse rate to calories. The patient's pulse values at two, three, and four calories are extrapolated to his expected, or predicted, maximal pulse, and the point of intersection predicts the maximum calories which the subject could achieve, although it might be extremely dangerous to permit him to do so. Authorities in work physiology, such as Hellerstein or Astrand, employ this technique, and the latter author has even produced a nomogram to avoid the construction of a graph for each patient.[9] The results are reproducible. These authorities permit a patient to work up to 50 percent of his predicted maximum capacity during peak effort periods on the job, and allow an average energy expenditure of 30 to 35 percent of the predicted maximum during the entire eight-hour day; this provides a large untouched cardiac reserve. The two to four calorie range of the test is equal to the energy costs of 90 percent of jobs in this country.

During this test, electrocardiograph monitoring is utilized continuously during exercise and throughout six-minute recovery periods to insure that the exercise is safely performed. The presence of ischemia is interpreted according to published criteria.[12,13] The search for ischemic changes on the electrocardiogram provides another technique to enhance the reliability of the pulse rate estimates of physical capacity. Another parameter which is used and adds to the reliability of the test is measurement of oxygen consumption. During a six-minute recovery period after exercise, "recovery oxygen" (oxygen debt) can be measured and correlated with the oxygen consumption during work. In normal subjects the total six-minute recovery oxygen above rest does not exceed the increment in oxygen consumption per minute above rest required by the exercise. If the recovery oxygen does exceed this increment it can be assumed that oxygen consumption has been deferred to the recovery period because of cardiac or pulmonary insufficiency during the exercise.

Work Prescription

The results of the exercise tests correlated with the history given by the patient, permits the physician or the work evaluation unit to provide the patient with a work prescription guide (Table 4), which defines specific limits. The use of exact classification and exact caloric figures, based on an authoritative workup, is reassuring to both the patient and his employer, and will do much to reduce industrial reluctance to hire

TABLE 4.—*Classification and Work Prescription Guide.*

Class		I	II	III	IV
Cal/min.		4.0-6.6	2.7-4.0	1.5-2.7	1.0-1.5
A.	Walk	Unlimited			
	Stairs	Unlimited			
	Lifting	60-100 lb.			
	Standing	Unlimited			
B.	Walk	Unlimited	1-2 miles	½-1 mile	
	Stairs	4 flights	2 flights	1-1½ flights	
	Lifting	40-60 lb.	25-40 lb.	15-25 lb.	
	Standing	Unlimited	Unlimited	75% of time	
C.	Walk	1-2 miles	½-1 mile	½ mile	1 block
	Stairs	2 flights	1 flight	1 flight	½ flight
	Lifting	25-40 lb.	15-25 lb.	10-15 lb.	5-10 lb.
	Standing	Unlimited	Unlimited	50-75% of time	25% of time
D.	Walk			2-3 blocks	½-1 block
	Stairs			½ flight	None
	Lifting			5-10 lb.	5 lb.
	Standing			25-50% of time	10% of time
E.	Unemployable				

cardiac patients. As more of them are permitted to return to work the unnecessary court litigations and misuses of the Workmen's Compensation Act involving cardiac accidents on the job will greatly decrease. A cardiac evaluation and rehabilitation program conducted at the Texaco Refinery, Port Arthur, by Wilson, has substantiated these statements.[1]

Studies done by both the Pittsburgh Work Evaluation Unit and the Cleveland Work Classification Clinic have determined that workers who followed the recommended work prescription were in no way harmed by continuing to work. Patients who tended to *overdo* had increased morbidity, when compared to patients who adhered to the work recommendations. Conversely, the Pittsburgh unit has developed evidence to show that the outlook for over-restricted patients or for patients who over-restricted themselves, is decidedly poorer. A similar finding among patients with rheumatic fever, that is, an increased morbidity associated with over-restriction, has been demonstrated in a study by Feinstein and associates.[14]

Vocational Rehabilitation And Guidance

There are many avenues open to the patient seeking reemployment after cardiac injury. He, of course, can return to his old job, with or without restrictions, as prescribed. Possibly a brief conference by his doctor with his employer will produce a reassignment within the same plant. If not, the Department of Vocational Rehabilitation, Vocational Guidance Services, and employment services are available to guide the patient to jobs within his capacity. On occasion, however, patients will require sheltered workshop or Goodwill Industries forms of employment to rebuild exercise tolerance through graduated exercise and work. Some patients may require retraining in a new field based on certain previously acquired skills and aptitudes, and will be eligible for either Veterans Administration or Department of Vocational Rehabilitation programs.

Of course a few patients will be unemployable and some patients will refuse to become reemployed; the latter patients in general have a passive-dependent personality structure and tend to react to heart disease with overconcern manifested by an exaggerated adherence to restrictive regimens. In this group, vigorous social service and psychiatric counseling will have to be done, although the outlook generally is poor.

Summary

All but 10 percent of survivors of myocardial infarction and other forms of heart disease can work after a bout of illness. In many cases return to work is accomplished satisfactorily and with a minimum of difficulty. However, in cases that present a more complex problem, use of exact methods of cardiac classification and exercise testing is invaluable in defining the limits of the patient's exercise tolerance, in providing guidelines to help the patient return to work, and also in allaying the anxieties of both patient and physician. The physician must be alert and wary as to the patient's emotional response to his illness, for it is this response

which most often creates difficulties. Patients who do return to work, in general, do so safely, and lead productive lives which are not shortened by continued work. For those patients whose problems are so complex that cardiac rehabilitation and classification cannot be achieved in the doctor's office, a work evaluation unit is now established at the St. Joseph's Hospital, Houston, and is accepting referrals.

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Dr. Turell, 3618 Yoakum Blvd., Houston 77006.

Doctors Asked To Have Drugs Labeled

Family doctors have been urged to ask pharmacists to label prescription drug containers with the name and strength of contents so that drugs can be identified quickly, especially in emergencies.

The action, taken by the American Academy of General Practice, is designed to (1) help prevent death in children or attempted suicides by enabling those finding them to identify immediately the drug taken and obtain the proper antidote; (2) help persons changing doctors or moving from one place to another to identify for the new doctor a drug being taken; (3) advise patients with allergies as to prescription contents so that they can identify drugs which have caused reactions, and (4) prevent mixups between two or more drugs being taken concurrently or between drugs being taken by different members of a family.

While recognizing the possibility of abuses, the Academy's action was based on the belief that "the advantages, especially in emergency situations such as drug poisoning in children, far outweigh the potential drawbacks." The Academy did stipulate 'under normal circumstances' to enable the doctor to control the situation when he knows his patient might be harmed by such knowledge.

Dr. Edward J. Kowalewski, Akron, Pa., who is chairman of the Academy's Commission on Environmental Medicine, said that the Academy believes the patient has a right to know the nature of his illness and what is being prescribed for it. He added that today's patient is sophisticated enough about drugs and their dangers to use the knowledge wisely.

Acute Hyperparathyroidism
A Surgically Correctable Cause of Coma

Stanley J. Zimmerman, M.D., Elmer K. Sanders, M.D.,
Darryl M. Williams, M. D., and William R. Berry, M.D.

Surgical relief of coma caused by acute hyperparathyroidism is reported. The authors believe the patient to be the 71st and oldest to survive. A work-up procedure for unconscious patients is described.

CASE REPORTS

THE COMATOSE patient often poses a diagnostic problem to the physician. This is particularly true if a good history cannot be obtained. The present case represents an unusual cause of coma and illustrates how definitive surgery can alleviate this condition.

Case History

A 77-year-old Negro woman was admitted to the Ben Taub General Hospital on July 20, 1965, because of progressive confusion and lethargy. She had been seen by a physician four years prior to admission because of dizziness. She was treated for hypertension briefly but did not continue therapy because the dizziness was unchanged. Eight months before admission, she began losing consciousness at times. She was easily aroused and resumed her activity, therefore no medical attention was sought. The family noted some slight changes in her alertness but they did not become concerned until three weeks before admission. At this time she again lost consciousness, fell to the floor, and struck her head. Afterward she complained of stiffness in her neck and was confused. She was admitted to another hospital and was told she had "cervical arthritis." Her mental status continued to deteriorate, and she had urinary incontinence. She was transferred to the Ben Taub Hospital where the physical examination revealed a blood pressure of 140/90, a pulse of 80, a rectal temperature of 99F., and respirations of 18 per minute. There was anisocoria, with the right pupil larger than the left, and there was bilateral arcus senilis. No papilledema was present. The neck was held in an extended position. Skin turgor was poor, and the lungs were clear except for a few diffuse, moist rales. The patient was disoriented and unable to answer questions except for a few slurred phrases. The remainder of the physical examination, including neurological evaluation, was unremarkable.

Laboratory findings included a hematocrit of 43 percent, white blood cell count of 7,800 per cu. mm. with a normal differential. The urine revealed a specific gravity of 1.008 and pyuria on microscopic examination. The blood urea nitrogen was 31 mg. per 100 ml. and the serum creatinine was 1.6 mg. per 100 ml. Blood glucose and electrolytes were within normal limits. VDRL was reactive 1:1. The electrocardiogram showed apparent S-T elevation in leads V_2-V_6 and was interpreted as showing subepicardial injury and the Q-T interval was .30. Chest roentgenogram was thought to be normal and a film of the abdomen revealed a calcified 3 cm. mass in the left flank. Intravenous pyelogram showed the mass was within the kidney. Pressure, chemical content, and cytology of the cerebrospinal fluid were normal.

Shortly after admission, the patient became more confused and was no longer able to respond to questions. Serum calcium at this time was 14.2 mg. per 100 ml., phosphorus 1.6 mg. per 100 ml., and alkaline phosphatase 4.2 King-Armstrong units. Sulkowitch's test of the urine was strongly positive. Repeat calciums were 14.7, 14.7, 17.2, and 14.2 mg. per 100 ml. The diagnosis was acute hyperparathyroidism, and emergency surgery was undertaken for removal of a parathyroid adenoma. A large mass was found in the lower pole of the right lobe of the thyroid gland. The mass was excised and submitted for frozen section study. The tentative pathologic diagnosis was follicular parathyroid adenoma. Because of the poor condition of the patient, only a cursory examination of the neck was made and no other masses could be found. Postoperatively there was no change in mental status. Serum calcium values were 16.0, 14.5, and 17.2 mg. per 100 ml. A urine calcium was 691 mg. per 24 hours. Forty-eight hours following surgery the final pathologic diagnosis of the neck mass was reported as "degenerating thyroid adenoma." The patient was again taken to surgery where a second mass weighing 3.7 gm. was found in the right lower parathyroid gland. Histologic study of the mass revealed parathyroid adenoma. No other masses could be found.

Following surgery the patient became progressively more alert. Serum calcium decreased throughout the following week to 8.9 mg. per 100 ml. Urine calcium was 19 mg. per 24 hours. The ST elevation and shortened QT interval of the electrocardiogram reverted to normal, and the blood urea nitrogen also returned to a normal value.

The patient was dismissed from the hospital, fully ambulatory and alert, 47 days following surgery.

Comment

This case illustrates a rare disease entity, and represents the oldest known patient to survive. In 1962 Gershberg and associates,[1] reported a 77-year-old male with acute hyperparathyroidism; however, this patient died before surgery could be performed.

Serum calcium and urine calcium determinations were elevated, and serum phosphorus determinations were low in our patient. In view of the lack of evidence of vitamin D ingestion, sarcoidosis, thyrotoxicosis, milk alkali syndrome, malignant bone disease, or multiple myeloma, the diagnosis of acute parathyroid intoxication was made and surgical exploration was undertaken. Unfortunately, the frozen section diagnosis was in error and a second surgical procedure was necessary.

Discussion

It is important that a definite approach be established in the care of a comatose patient. The outline in Table 1 is the general procedure used in the work-up of an unconscious patient on the medical service of the Ben Taub General Hospital. The history and physical examination is of primary importance in determining which of the studies is chosen. A patient in insulin shock can be readily recognized and treated in a simple, rapid fashion without an extensive work-up. On the other hand, a patient such as the one presented, who has a perplexing history and an unrevealing physical examination, presents a much greater problem. We have found that a flat plate film of the abdomen is an extremely helpful procedure as has been pointed out by Rosenbaum and associates.[2] In this patient, calcification was seen in the kidney and, although this type of calcification is not typical in hyperparathyroidism, it has been our practice to obtain calcium and phosphorus determinations on all patients with ectopic calcification.

Howell has illustrated the difficulties in identifying certain parathyroid adenomas which resemble thyroid tissue.[3] Acute hyperparathyroidism, which has a mortality rate of 59 percent,[4] presents a medical and surgical emergency.[5] Adenomas of the parathyroid gland account for 85.7 percent of all cases of acute hyperparathyroidism;[5] the majority occur in the inferior parathyroid glands.[6] A careful examination of the neck should be made, and an attempt to identify each parathyroid gland is imperative. Finding a solitary adenoma does not justify the surgeon's refraining from further exploration of the neck since there is a 7 percent incidence of multiple adenoma. Unfortunately parathyroid adenomas may also be found on the pericardium[7] and in the mediastinum.[8] Serum calcium levels will usually decrease within 48 hours following removal of the adenoma and as demonstrated in this case, will serve to indicate whether or not the cause of hypercalcemia has been removed.

TABLE *1.—Procedure in Work-up of Unconscious Patient.*

I. History and physical examination (as complete as possible)
II. Complete blood count, urine, and stool for occult blood (occasionally clot retraction) and prothrombin time
III. Cultures (blood, urine, stool, cerebrospinal fluid)
IV. Chemistries
  A. Required
    1. Blood urea nitrogen
    2. Blood sugar
    3. Carbon dioxide
    4. Chloride
    5. Bromide
  B. Supplemental
    1. Amylase
    2. Calcium and PO_4
    3. Urine porphobilinogen
V. X-rays
  A. Skull
  B. Chest
  C. Abdomen
VI. Lumbar puncture
VII. Electrocardiogram

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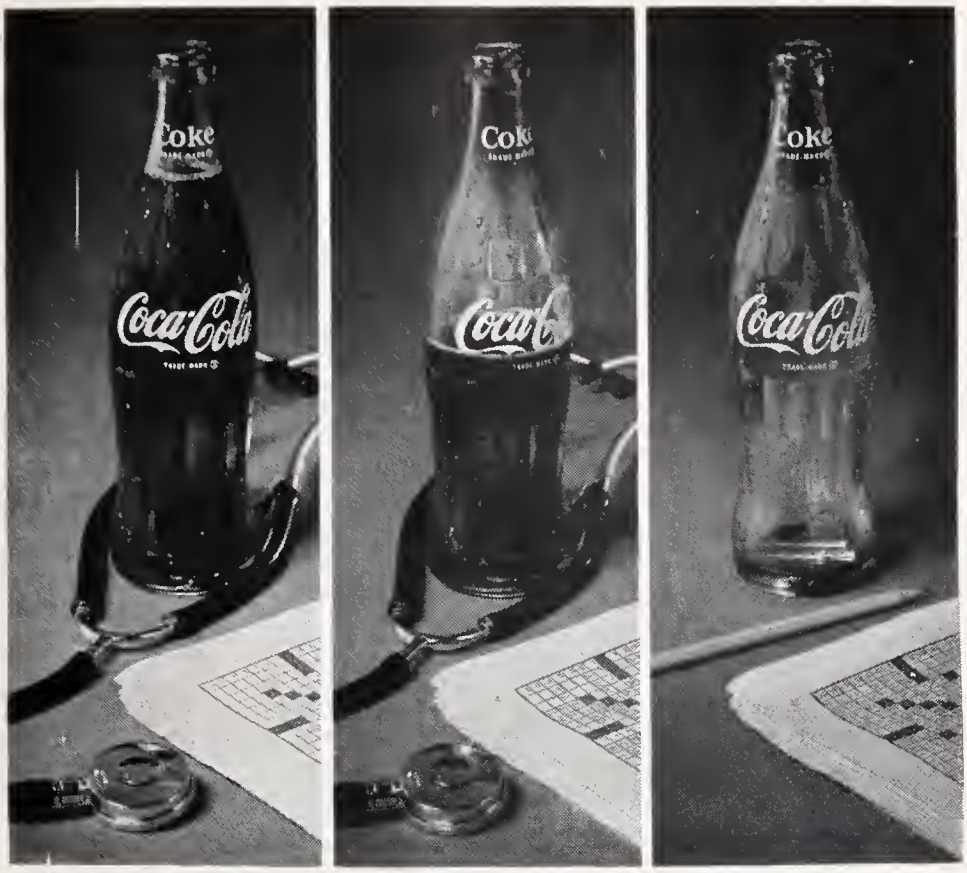

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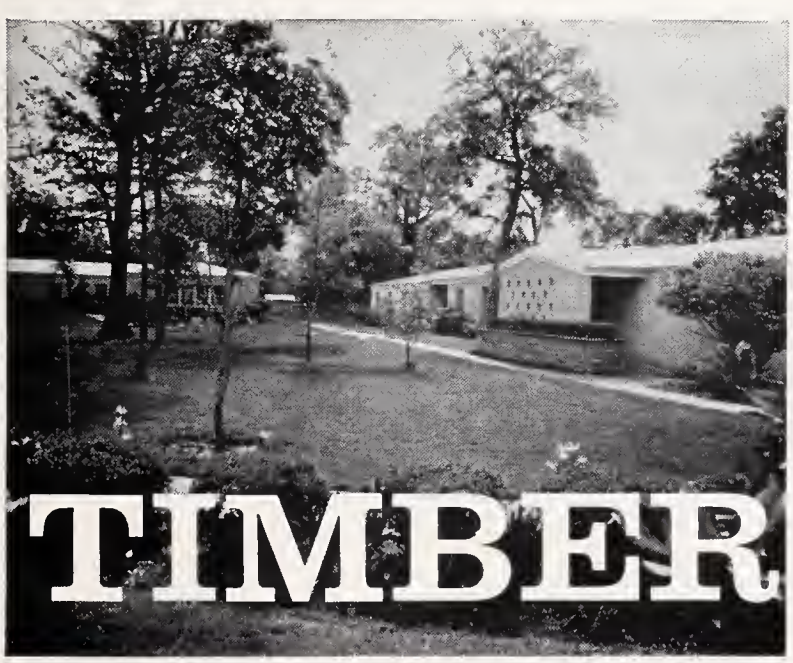

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Pro-Banthine
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Intragastric photography has provided a new and precise method of measuring the effectiveness of anticholinergic drugs. The transition from gastric motor activity to relaxation seen with effective doses of such drugs takes only a few seconds and is easily demonstrated.

The importance of vagal stimulation of gastric hyperacidity and hypermotility makes such measurements particularly important in evaluating the parasympatholytic effect of drugs used in patients with peptic ulcer, gastritis, biliary dyskinesia and other gastrointestinal disorders.

Pro-Banthine has been shown[1] to produce complete gastric motor inactivity with doses of 6 to 8 mg. intravenously. Comparison tests were made with the belladonna fraction, atropine. Measured usual dosage unit versus usual dosage unit, Pro-Banthine was more than four times as effective as the belladonna alkaloid.

See fo

Indications: Peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Oral Dosage: Adequate dosage should be given for optimal results. For most *adult* patients this will be four to six 15-mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily may be required. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications: Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. Pro-Banthine is contraindicated in patients with glaucoma, severe cardiac disease and prostatic hypertrophy.

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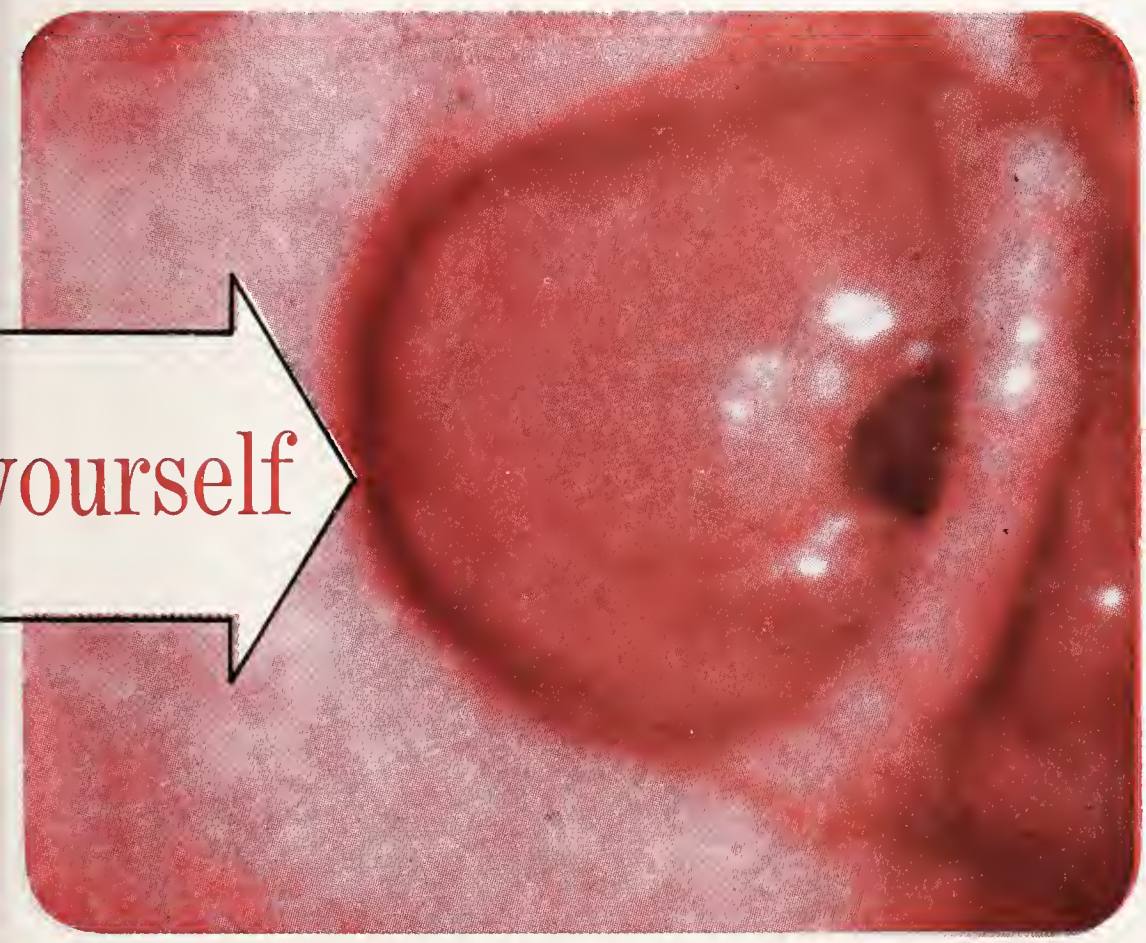

Complete gastric relaxation with Pro-Banthine. As this intragastric photograph demonstrates, gastric relaxation is attained with 6 mg. of Pro-Banthine intravenously; the antrum is relaxed and the pyloric orifice remains open. Full intravenous doses of atropine (4 mg.) produce no measurable effect.

SEARLE

Research in the Service of Medicine

Clinicopathologic Conference

James D. Lehmann, M.D., Steven Schenker, M.D.; Burton Combes, M.D.;
J. P. Sanford, M.D.; D. W. Seldin, M.D.; and Bruce D. Fallis, M.D.

DR. JAMES D. LEHMANN: A 58-year-old white man was admitted to Parkland Memorial Hospital on Aug. 20, 1965, because of jaundice. He died 12 days later.

He had been well until three weeks before admission when he had insidious onset of malaise, weakness, dizziness when standing, anorexia, and distaste for cigarettes. A few days later he noted dark urine, clay-colored stools, and gradually increasing jaundice without pruritus. He said that he had lost 4 lb. in the preceding month, but his admission weight was 135 lb., or 15 lb. less than his normal weight. He had been constipated the week before admission.

He said he had no abdominal pain, vomiting, hematemesis, or melena. There was no history of fatty food intolerance, contact with jaundiced individuals, injections, or contact with animals or lake water. Alcoholic intake was negligible. He had not traveled out of Dallas County. The patient had worked as a painter all his adult life; he used rubber-based paint and some naphtha solvents but had never been overcome by the fumes, and did not use carbon tetrachloride. He had not seen a physician since 1940, when he had a hemorrhoidectomy.

Physical examination on admission revealed blood pressure, 120/75 mm. of mercury; pulse, 68 and regular; respirations, 12; and temperature, 99 F. He was a well developed, well nourished, deeply icteric man. He was alert and well oriented and had a stoic personality. The sclerae were icteric. There was no fetor hepaticus, cervical lymphadenopathy, venous distention, or gynecomastia. The chest was clear to auscultation and percussion. The heart sounds were normal except for a faint apical systolic ejection murmur. The liver was enlarged and extremely tender, and the entire right side of the abdomen was tender to deep palpation without rebound tenderness. The liver contour was questionably irregular. The gallbladder and spleen were not palpable, and there were no other abdominal masses. Bowel sounds were normal, as were the genital organs. A few small, shotty inguinal lymph nodes and one small right axillary lymph node were palpable. There was minimal palmar erythema but no cyanosis, clubbing, or edema. Numerous spider telangiectases were present over both shoulders. Neurological findings were within normal limits, and there was no asterixis.

Initial laboratory studies showed the following values: hemoglobin (Hgb.) 16.3 gm. per 100 ml.; hematocrit (Hct.) 49 percent; white blood cell count (WBC) 11,800, with 77 percent neutrophils, 3 percent bands, and 13 percent lymphocytes; and adequate platelets on smear; urinalysis, normal except for slight albuminuria and bilirubinuria; serum electrolytes, normal; blood urea nitrogen (BUN) 19 mg. per 100 ml.; fasting blood glucose, 115 mg. per 100 ml.; cholesterol, 162 mg. per 100 ml.; serum amylase, 320 Somogyi units; creatinine, 1.6 mg. per 100

This is the first in a new series of clinicopathologic conferences from the University of Texas Southwestern Medical School, Dallas. Guest editor for the series is Dr. Bruce D. Fallis, professor of pathology. Other participants in this conference are Dr. James D. Lehmann, resident in medicine, Parkland Memorial Hospital; Dr. Steven Schenker, assistant professor of internal medicine; Drs. Burton Combes and J. P. Sanford, associate professors of internal medicine, and Dr. D. W. Seldin, professor and chairman, Department of Internal Medicine.

The patient had malaise, weakness, anorexia, weight loss, jaundice, hypoglycemia, slight albuminuria, and high transaminase and serum bilirubin levels. Confusion, asterixis, fetor hepaticus, hypothermia, and coma developed, and the patient died.

ml.; serum albumin, 3.9 gm. per 100 ml. Guaiac test showed no occult blood in the stool. Values from initial liver function studies were: serum bilirubin, 21 mg. per 100 ml. (11 mg. direct); cephalin cholesterol flocculation, 3+ at 24 and 48 hr.; thymol turbidity, 30 units; akaline phosphatase, 11.3 Bodansky units; serum glutamic oxaloacetic transaminase (SGOT), 2,024 units; and prothrombin time, 35 percent of normal. Radiographic studies revealed essentially normal heart and lungs; a liver scan showed poor concentration of Radio-Rose Bengal.

Isolation, bed rest, vitamins, and a high caloric diet were prescribed. Aquamephyton was given without significant response. The patient's appetite was good, and he complained only of mild abdominal discomfort during the first two days. He was given juices hourly, and a bed-time meal because of a tendency toward hypoglycemia when fasting. The third night after admission he awakened with dizziness and sweating, and his blood glucose level was 52 mg. per 100 ml., which was the lowest recorded. He ate well the following day, saying that he had regained his appetite. His rectal temperature was 97.6 F.

On the fifth morning he was confused and manifested asterixis; fetor hepaticus was present. Continuous intravenous administration of 10 percent glucose in water was begun, along with oral neomycin, 6.0 gm. per day. A neomycin enema was given. In the ensuing eight hours the patient became more disoriented and agitated; he had abdominal distention and normal bowel sounds. Vital signs were stable except for persistent hypothermia. A stool guaiac test gave 1+ value. A nasogastric tube was inserted, and a guaiac test of gastric contents gave negative results. Urine output was adequate. Prednisolone, 100 mg. intravenously initially, then 25 mg. every six hours, was begun; antacids were given via the nasogastric tube.

On the sixth day the patient remained unaware of his surroundings; asterixis and fetor hepaticus persisted. The liver decreased somewhat in size. Intravenous fluids were continued. Gastric suction began yielding dark brown material, without bright red blood, which had 4+ guaiac reaction. Laboratory studies showed BUN, 30 mg. per 100 ml.; sodium, 126 mEq. per liter; carbon dioxide content, 27 mEq. per liter; potassium, 5.0 mEq. per liter; blood sugar, 110 mg. per 100 ml.

On the eighth and ninth days the patient's level of consciousness seemed minimally improved. His vital signs were stable; however, he had oliguria, and his abdomen was distended with a demonstrable fluid wave. The liver edge could no longer be palpated. Neomycin was decreased to 3.0 gm. per day because of increasing azotemia. Fluids were restricted to the amount of output plus gastrointestinal losses, and were given as 10 percent dextrose in water and in normal saline.

The last three days, the patient was unresponsive, hypotensive, and his rectal temperature rose from 96 F. to 99-100 F. He had shallow, labored respiration and increasing ascites and peripheral edema. The lungs remained clear. No gross blood was aspirated from the nasogastric tube, although both stool and gastric aspirates had a 4+ guaiac reaction. The prothrombin time fell to less than 10 percent of the control. Blood transfusions were given, and Aramine was begun 36 hours before death to maintain blood pressure. The patient died 12 days after admission. The day before death values from laboratory studies were: Hgb., 12.1 gm. per 100 ml.; Hct., 38 percent; WBC, 18,700; platelets, 125,000; blood urea, 65 mg. per 100 ml.; creatinine, 6.1 mg. per 100 ml.; blood glucose, 142 mg. per 100 ml.; bilirubin, 40 mg. per 100 ml. (direct, 21); cephalin-cholesterol flocculation, 4+ at 24 and 48 hours; thymol turbidity, 19 units; alkaline phosphatase, 6.7 Bodansky units; prothrombin time, less than 5 percent of control.

Clinical Discussion

DR. STEVEN SCHENKER: This man's illness lasted about 30 days, with symptoms of malaise, weakness, anorexia, a 15-lb.

weight loss, and jaundice. Significantly, he denied alcoholic intake, and although he was a painter, there was no history of exposure to organic solvents. He was afebrile and icteric, and had a few spider angiomata and a large, tender liver. Laboratory data gave evidence of parenchymal liver disease and especially liver cell necrosis, as manifested by the high transaminase levels. With no known exposure to toxic agents or ischemic necrosis from shock, transaminase elevation of this degree strongly suggests viral hepatitis. Initially, the patient had slight albuminuria, and this momentarily raised the question of leptospirosis. However, the clinical course is not that of leptospirosis, and albuminuria of this degree is not uncommon in patients with viral hepatitis. I believe we can safely discount leptospirosis, and conclude that the patient had viral hepatitis.

From the beginning, the patient's illness gave evidence of severity. Thus, he had a prolonged prothrombin time, one of the best indices of severe viral hepatitis. In addition, he was hypoglycemic and had a high level of serum bilirubin. A high serum bilirubin level does not necessarily correlate with a severe course of viral hepatitis, though the distinct impression of many clinicians is that it does. The patient's subsequent course certainly bears out the early impressions.

This patient showed the characteristic pattern of confusion, asterixis, fetor hepaticus, and finally coma. He also was hypothermic. In a recent clinicopathologic discussion[1] hypothermia was considered a rare finding in hepatic coma. I don't believe this is the case, and hypothermia should be looked for in every patient with severe liver disease as a possible early clue to the development of hepatic coma. If we look at these patients' charts, I believe hypothermia will be a rather common, though nonspecific, finding.

Asterixis is also an entirely nonspecific sign which may be present in uremia, carbon dioxide narcosis, barbiturate intoxication, or other conditions. It is caused by a lapse in posture of the appropriate muscles, and is associated with periods of electrical silence on the electromyograph; it is best brought out by sustained extension, is absent at rest, and is mitigated by intentional movements.

Fetor hepaticus, by contrast, occurs only in patients with severe liver disease, and is probably related to abnormal metabolism of amino acids, especially methionine. Challenger and Walshe[2] have suggested that the odor is caused by methylmercaptan and dimethyl sulfide. The odor of these compounds is so intense that one part per billion can give a characteristic odor. In some series of patients with severe liver disease, fetor hepaticus has been noted in 40 to 50 percent of cases. When present, this is a helpful diagnostic feature.

Hypoglycemia is rather unusual in a patient with severe liver disease. It may be caused by impaired storage of glycogen in the extensively damaged areas of the liver as well as impaired gluconeogenesis as manifested by the elevated serum levels of various amino acids.

I mention hypoglycemia specifically because it represents a treatable disorder, and although appropriate treatment does not reverse the underlying liver disease, it may salvage the patient and prevent irreversible brain damage during the acute course of his illness.

The management of this patient typifies classical current treatment of hepatic coma and consists essentially of three parts:

1. Removal of the precipitating cause of hepatic coma, such as gastrointestinal hemorrhage, diuretic-induced hypokalemic alkalosis, azotemia, and infection.
2. Reduction of the influx of "toxic" nitrogenous substances into the circulation by removing protein from the diet and "sterilizing" the intestine with antibiotics.
3. Maintenance of adequate caloric intake with attention to electrolyte balance and vitamin supplements.

All these measures were carried out in this patient. In hepatic coma, the best prognosis is for patients who have obvious, potentially reversible, precipitating causes for the coma. The worst prognosis is for patients such as this one, who have primary liver failure without a treatable precipitating factor.

Neomycin is one of the best drugs for reducing intestinal bacterial flora. However, approximately 3 percent of neomycin is absorbed from the intestine, and in the presence of severe renal disease and oliguria this can lead to systemic accumulation of the antibiotic and potential ototoxicity and nephrotoxicity. For this reason, the dose of neomycin was decreased in this patient terminally.

Cortisone was given primarily because little else could be done for him at the time. There is no statistically valid evidence that

cortisone benefits patients with severe viral hepatitis. The only study that suggests benefit is that of Katz and associates,[4] who had essentially a 100 percent mortality rate in a series of patients with fulminant viral hepatitis. Subsequently, when steroids in large doses (comparable to those our patient received) were employed, 9 of 23 consecutive patients survived. Clearly this is not a well-controlled study, but it suggests benefit. Because of this we have been using, until recently, large doses of steroids in patients with severe fulminating viral hepatitis. It is important to bear in mind that large doses of steroids may cause serious side effects. In the study cited,[4] 3 of 23 patients thus treated had major complications probably related to steroid therapy—a perforated peptic ulcer in one and massive gastrointestinal bleeding in two. Furthermore, secondary infections can be masked by steroids, especially when large doses are given. Cook and Sherlock[3] reported the use of steroids in six patients with fulminating viral hepatitis, but none survived. We have recently begun a controlled study of steroid therapy for fulminant viral hepatitis, but have had too few cases to allow valid conclusions.

I believe this patient had subacute hepatic necrosis resulting from viral hepatitis. Because the illness lasted about 30 days, I suspect that the liver, at autopsy, showed areas of both necrosis and nodular regeneration. The patient certainly had hepatic coma, and possibly the brain showed some increase in size and number of protoplasmic astrocytes, even though the coma was brief. The renal lesion probably was secondary to the hepatic lesion, which is the sequence of the hepatorenal syndrome. The renal pathologic findings vary in such cases, with nonspecific changes in the glomeruli or tubules in some instances and no detectable alterations in others. In addition, terminal shock may confuse the renal findings at autopsy. The patient had gastrointestinal hemorrhage, probably from multiple small ulcers of the gastrointestinal tract. Bleeding esophageal varices may develop rapidly in patients with severe viral hepatitis. However, the extent of hemorrhage in this patient would make me suspect that the bleeding was not a major component of his terminal illness. The only other treatment that might have benefited this patient would have been transfusions of fresh blood to try to raise his prothrombin level, and I would have advocated this.

DR. BURTON COMBES: The mortality rate in viral hepatitis is relatively low; most people with hepatitis get well. The striking fact is that despite wide fluctuations in the reported incidence of the disease in this country, the mortality rate has remained relatively stable. This suggests that the lethal form of hepatitis may be caused by an agent that differs from the virus which generally is considered to cause most of the reported cases. Thus, the relatively stable mortality rate may be due to a relatively constant rate of infection by a particularly virulent virus, such as homologous serum hepatitis virus, or to some other as yet unidentified agent. Unfortunately, there is no definitive way to learn which virus is present in a given patient.

In this case, there was no evidence of previous injections or blood transfusions. I wonder whether the patient had received any dental care recently, because improperly sterilized dental instruments may be one important vehicle for transmitting homologous serum hepatitis virus. Unfortunately, we have no definitive information about this. The patient's age, of itself, is associated with a poor prognosis. An interesting and as yet unexplained aspect of hepatitis is that the mortality rate is lowest in persons between the ages of 9 and 14, and rises progressively with increasing age.

As Dr. Schenker pointed out, there is no specific treatment for hepatitis virus infection, and therapy is basically supportive. Outcome largely depends on the extent of hepatic damage that will ensue, and treatment in the severely ill patient centers about protection against hypoglycemia, treatment of hepatic coma, and providing appropriate fluid and electrolytes in the hope that he will have enough hepatic regeneration to survive. I agree with Dr. Schenker's comments about blood loss in this patient. Guaiac-positive stools are fairly common in people with severe viral hepatitis. Extensive gastrointestinal bleeding can cause hypotension and prerenal azotemia, with the blood itself contributing to the azotemia (alimentary azotemia). This patient should have received blood, but I doubt that bleeding was a major factor in the outcome.

As for steroids, the impression as to their effectiveness in the treatment of massive hepatic necrosis due to viral hepatitis is based largely on the report of Katz and associates.[4] Up to 1950, all 16 of their hepatitis patients who developed spontaneous

coma died. Since 1951, 9 of 23 such patients survived. It is of interest that 6 of 12 patients who received cortisone, 1.5 gm. per day, survived. When prednisolone was used instead of cortisone, 2 of 6 survived, and when dexamethasone was used, only 1 of 5 survived. This variation in survival rates may be due to chance, but it is still striking that with cortisone there was a much better recovery rate than with the other steroids.

In our experience over the past five years, eight to ten patients with fulminant hepatitis have received steroids, almost invariably prednisolone, and they all died. Cook and Sherlock[3] noted that all 13 patients with massive hepatic involvement who received hydrocortisone or prednisolone died. We tend to think of steroids as being relatively interchangeable with regard to their anti-inflammatory effect. In view of the uniformly poor results that we and Cook and Sherlock have experienced, and the less favorable outcome noted by Katz and associates with prednisolone and dexamethosone, it seems reasonable to query whether there may be some advantages inherent in the choice of cortisone.

DR. J. P. SANFORD: As Dr. Schenker has pointed out, proteinuria and pyuria occur with infectious hepatitis. Many times these findings raise the question of leptospirosis, which is a good thought. However, in studies during the Korean War, about 30 percent of patients with infectious hepatitis had proteinuria and pyuria, and a lesser percentage had hematuria. Thus, infectious hepatitis, like most other viral diseases, is generalized and causes abnormalities in many organs. Intestinal biopsy reveals changes such as cellular infiltrate and flattening of villi in a high percentage of cases. Therefore, proteinuria and pyuria would be manifestations of the hepatitis.

The problem of hypoglycemia was an important aspect of the patient's management. During his third night in the hospital he awoke with dizziness and sweating. The next day his temperature was 97.6 F. Hypothermia may be a manifestation of hypoglycemia, and this is particularly important in that hypoglycemia may be precipitated iatrogenically: If one administers intravenous glucose to a patient and then stops it when insulin output by the pancreas has been stimulated, severe reactive hypoglycemia may occur. Therefore, one must be quite cautious about the episodic administration of glucose to patients with severe hepatic insufficiency. Unless this patient were maintained on continuous glucose from that point on, I suspect he might have had intermittent hypoglycemia of a fairly severe degree, and this may have accounted for much of his central nervous system deterioration and coma.

In reviewing the course of patients with fulminant hepatitis, almost all appear at first to have ordinary viral hepatitis; initial history and physical findings are not distinguishable from those of patients with benign hepatitis. However, usually within the first ten days their condition begins to deteriorate rapidly. Terminally, ascites and edema often develop, just as in this case.

In regard to the present status of our knowledge of the hepatitis virus, a great deal of effort has gone into the isolation of agents from hepatitis patients and there are numerous reports of the isolation of such agents. However, at present, none of them is more than a so-called candidate agent. Within the past three years there have been numerous isolations of adenoviruses, which for a while were thought possibly to be responsible for hepatitis. However, with more extensive studies this seems to be a chance association, or perhaps the adenovirus plays a supportive role in association with the hepatitis virus. There is an analogy to this in the Rous's sarcoma virus, which is able to infect cells, but cannot produce infectious particles which can subsequently infect second cells unless it is associated with another virus, the so-called Rous-associated virus. The Rous's sarcoma virus is able to replicate nucleic acid but is not able to induce the cell to replicate the capsid protein which is necessary for transmission of the virus to other cells. The Rous-associated virus induces the cell to make the capsid protein, which is a necessary part of the fully infectious virus particle. It is possible that adenovirus agents play a role in symbiosis, if you will, with the specific agent of infectious or serum hepatitis.

DR. D. W. SELDIN: One of the interesting features of this patient's course is a progressive, insidious rise in the serum creatinine level from 1.6 mg. per 100 ml. on admission to 6.1 on the day before death, and to 8.6 on the day of death. There is every reason to believe that the rise in creatinine represents a failure of renal function, probably caused by a collapse of the renal circu-

lation. The failure of renal function in the course of infectious hepatitis can be classified into two broad groups. In one, the failure of renal function can clearly be attributed to identifiable factors which impair renal circulation or injure the kidney directly. For example, it is conceivable that a patient with infectious hepatitis could become profoundly salt-depleted or could bleed massively, and under such circumstances the creatinine would rise. These would be examples of prerenal azotemia. It is also conceivable that the virus might infect the kidney and cause serious renal injury, although to my knowledge this has never been demonstrated.

The second type of renal failure is a peculiar one which seems to have nothing to do with infectious hepatitis or any other specific form of hepatic injury, but may be a feature of any form of profound hepatic failure. It has been studied by Papper, particularly as it occurs in alcoholic cirrhosis.[5] This form of renal failure is characterized by development of oliguria in the presence of high cardiac output and low glomerular filtration rate and renal blood flow. Papper recently demonstrated the presence of high cardiac output and low renal blood flow and glomerular filtration rate in the same patient. He proposed that in advanced hepatic failure, the circulation tends to develop extensive shunts leading to renal vasoconstriction, which in turn leads to renal failure. He has attempted to treat this disorder with norepinephrine, to no avail. In such patients, a collapsing renal circulation and impaired renal function will aggravate hepatic coma, because the blood urea level will rise, and the urea pool will, therefore, expand. An expanded urea pool causes increased ammonia production, and an elevated blood ammonia level is probably a major determinant of hepatic coma.

In this patient, failure of renal function may have contributed to death. For renal failure caused by extensive shunts and renal vasoconstriction there is, as far as I know, no effective treatment. On the other hand, it is possible that the circulatory disturbance was of the first type, which is potentially treatable, and I want to emphasize the question of blood loss. In the first place, the hematocrit was initially 49 percent and terminally 38 percent. This is a sizeable decrement. It could have been caused by modest blood loss, more or less in proportion to the change in the hematocrit readings, and that would not be an overwhelming hemorrhage, assuming that hemodilution was adequate. It is also interesting that the patient was edematous at the time of death. His serum albumin level on admission was normal, but we have no subsequent determinations. The level of serum albumin could have fallen rapidly during his illness as a result of the massive hepatic necrosis. If that is so, shrinkage of plasma volume may well have masked a massive blood loss so that the decrement of hemoglobin and hematocrit may not accurately reflect the extent of hemorrhage. Although it may be true, as others proposed, that blood loss played a minor role in the collapse of the circulation, I think it was a mistake not to have given more blood in the presence of known gastrointestinal bleeding.

Pathologic Findings

DR. BRUCE D. FALLIS: At autopsy, the body was well nourished, deeply icteric, and moderately edematous. The abdomen was protuberant and contained 3,500 ml. of bile-stained ascitic fluid. There was bilateral hydrothorax (right 600 ml., left 200 ml.) with resultant slight compression atelectasis of both lungs.

The liver weighed 1,100 gm. and was small and dark brownish-green; the left lobe was more shrunken than the right. The external surface was coarsely nodular with projecting nodules up to 1 cm. in diameter. The liver cut with increased resistance, and on the cut surface were slightly bulging nodules of greenish-brown liver tissue, varying from 1 to 10 mm. in diameter. Between these nodules were sunken bands, 1 to 4 mm. wide, of firm, gray-white fibrous tissue which was more abundant in the left lobe than in the right. Many of the larger nodules had undergone central infarction and contained a greenish-yellow focus of recent coagulation necrosis, surrounded by a margin of purplish hemorrhage.

Microscopic study of the liver revealed severe chronic hepatitis with early postnecrotic cirrhosis and nodular infarcts. The liver consisted of varying sized masses of freshly necrotic, smudgy, ghost-like liver cells with poorly defined cytoplasmic margins and poorly stained nuclei. Few viable hepatocytes were present. The bands of fibrous tissue contained numerous small bile ducts and ductules and many chronic inflammatory cells, chiefly lymphocytes and macrophages.

Some of the macrophages contained hemofuscin pigment derived from necrotic liver cells. There were a few nodules of regenerating liver cells composed of large cells with bile-stained cytoplasm and large nuclei with prominent nucleoli (Fig. 1).

The infarcted nodules noted grossly were characterized by a central focus of coagulation necrosis with a band-like zone of neutrophilic leukocytes near its margin and a peripheral zone of congestion and hemorrhage.

The hepatic lesions—autolytic necrosis of hepatocytes, attempted regeneration, and inflammatory infiltrate—are typical of viral hepatitis. Some writers have stated that the inflammatory infiltrate of viral hepatitis is almost entirely mononuclear and that neutrophilic leukocytes are rare. However, others have found neutrophilic leukocytes to be frequent in viral hepatitis, and this has been our experience in both needle biopsies and autopsy cases. The extent of fibrosis in this case was unexpected. Fibrosis of slight degree may occur in acute viral hepatitis and is a collapse fibrosis resulting from loss of liver cells and condensation of stroma. However, the degree of fibrosis in this case was too great to be explained as a collapse fibrosis. Evidently new fibrous tissue had been formed (productive fibrosis), indicating a transition from chronic viral hepatitis to early postnecrotic cirrhosis. Regenerating nodules of liver tissue were also present; some were compressed by surrounding fibrous septa, resulting in poor intranodular blood circulation. Nodular infarcts occurred when hypotension further impaired the intranodular circulation.

The transition from hepatitis to postnecrotic cirrhosis in this case indicates that the illness was somewhat longer than the history suggested. Clinically, the patient is said to have been ill for about three weeks prior to hospitalization. In view of the patient's stoic personality and the insidious onset of his illness, the disease could have been present longer. Counting his two weeks in the hospital, the apparent duration of his disease was about five weeks. However, the anatomic changes, especially the considerable degree of productive fibrosis and regenerative hyperplasia, indicate a process of at least six weeks and probably longer. It is not uncommon to discover that viral hepatitis is actually more chronic than the history would suggest. In this patient, the disease probably was smoldering and clinically inapparent for several weeks to a few months and then, for unknown reasons, suddenly worsened with widespread and massive necrosis of liver cells and a fulminant termination.

The extensive fibrosis and nodular regeneration indicate progression to early postnecrotic cirrhosis. Viral hepatitis is now believed to be the most common cause of postnecrotic cirrhosis, although less than 1 percent of cases progress to cirrhosis. The progression of hepatitis to cirrhosis was first clearly shown by Marchand[6] in 1895. Unfortunately, the factors which determine this progression are unknown, although progres-

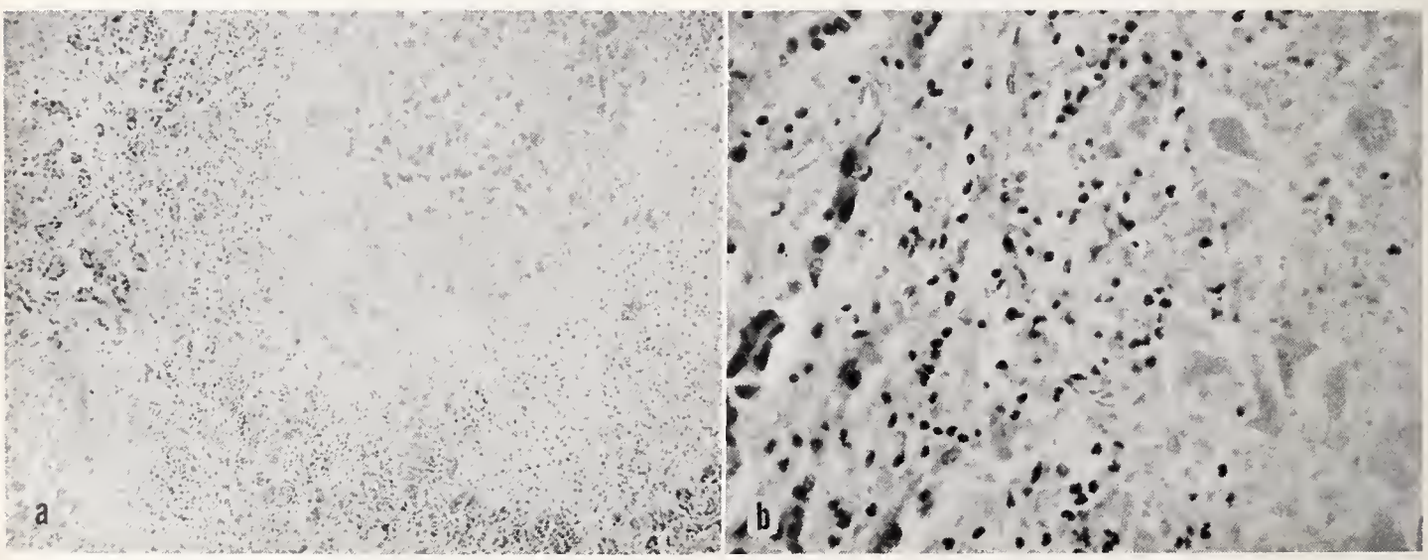

Fig. 1. a. Nodules of necrotic liver cells are present in the upper center and the lower left. Nate the numerous inflammatary cells and the many small bile ducts (upper left). X 82.

b. Necrotic liver cells, some with two or more nuclei, are present in the right half af the field. On the left are bile ducts and inflammatory cells embedded in fibrous tissue. X 352.

sion occurs somewhat more frequently in women than in men.

Each kidney weighed 150 gm. and was soft, swollen, and bile-stained. Microscopically the only changes were severe, cloudy swelling of the tubular epithelium, especially of the proximal convoluted tubules, and bile pigment in the tubular lumina. This nonspecific degeneration, or bile nephrosis, is the usual finding in persons with severe liver disease who develop the hepatorenal syndrome with oliguria and azotemia. Often the anatomic renal changes in such cases are quite unimpressive compared with the clinical picture of rapidly progressive renal failure. Although the renal lesions are nonspecific, they are certainly compatible with failure of intrarenal circulation. The lesions are not caused by any toxic effect of bile, as the somewhat misleading name, bile nephrosis, suggests.

The heart weighed 440 gm. and was dilated. The myocardium was flabby, grayish, and opaque, and microscopic study revealed cloudy swelling. The lungs weighed 1,560 gm. and were the seat of acute pulmonary congestion and edema as well as compression atelectasis due to the bilateral hydrothorax. There was mucopurulent tracheobronchitis but no bronchopneumonia.

There were acute mucosal erosions in the lower third of the esophagus, and microscopic examination revealed surface infection by *Candida albicans*. Such fungous overgrowth is not unexpected in a debilitated patient whose therapy includes adrenal steroids and wide-spectrum antibiotics. The stomach and intestine were congested with many mucosal petechiae and ecchymoses but no ulcers. There was no gross blood in the lumen of the gastrointestinal tract. Presumably, the gastrointestinal hemorrhage noted clinically was the result of diffuse mucosal oozing secondary to a bleeding diathesis complicating the severe liver disease. There were also numerous petechiae in the serous membranes.

The spleen weighed 210 gm. and was acutely congested. The brain weighed 1,260 gm. and was grossly normal. Microscopic study revealed no evidence of hypoglycemic encephalopathy. There was minimal astrocytosis but no hypertrophy of astrocytes. In cases of hepatic coma, microscopic lesions in the central nervous system usually are seen only when the disease is long-standing or recurrent.

In summary, the major autopsy findings were chronic viral hepatitis with early postnecrotic cirrhosis and nodular infarcts, bile nephrosis, anasarca, acute erosive esophagitis, cloudy swelling of the myocardium with cardiac dilatation, acute pulmonary congestion and edema, and petechiae and ecchymoses in the gastrointestinal tract and serous membranes. Death resulted from the severity of hepatitis with resultant hepatic coma.

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Iodine Study Shows Bread Highly Iodized

Persons who must limit their intake of iodine should be aware that bread can be a dietary source of large quantities of iodine, advises Dr. W. T. London and associates from the National Institute of Arthritis and Metabolic Diseases, Bethesda, Md. During a study of iodine intake and excretion, the *Schering Science Bulletin* reports that these investigators were at first puzzled by the many persons eating apparently normal diets who showed signs of high iodine intakes. Subsequently, white bread was found to be the culprit.

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Indications: In depression of any kind—neurotic and psychotic depressive reactions; manic-depressive or involutional psychotic reactions. **Dosage:** Optimal results are obtained at a dosage of two 25 mg. tablets t.i.d. (150 mg./day). **Contraindications and Precautions:** Glaucoma, urethral or ureteral spasm, recent myocardial infarction, severe coronary heart disease and epilepsy. Should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Safety in human pregnancy has not been established. **Adverse Effects:** Side effects, usually mild may include: dry mouth, constipation, dizziness, palpitation, delayed urination, "bad taste," sensory illusion, tinnitus, agitation and stimulation, sweating, drowsiness, headache, orthostatic hypotension, flushing, nausea, cramps, weakness, blurred vision and mydriasis, rash, allergy, transient eosinophilia, granulopenia, altered liver function, ataxia and extrapyramidal signs. **Supplied:** Norpramin (desipramine hydrochloride) tablets of 25 mg., in bottles of 50, 500 and 1000.

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NEWS

June 28 Is Date

Texas Delegates To Nominate Dr. Rouse for AMA Presidency

The Texas delegation to the American Medical Association is proceeding with plans to nominate Dr. Milford O. Rouse of Dallas as President-elect of the AMA for 1966-67. The nomination will be made during the June 26-30 meeting of the AMA in Chicago. An attendance of 45,000, including 15,000 physicians, is expected for the 115th meeting of the AMA, the largest medical meeting in the world.

A diverse scientific program covering virtually every medical specialty will be presented. The AMA's policy-making House of Delegates will meet in the Palmer House hotel, and more than 350 scientific exhibits will be displayed in McCormick Place convention center, where many of the scientific sessions will also be held.

Dr. Rouse, a specialist in gastroenterology, will be nominated on Wednesday, June 28, by Dr. Harvey Renger of Hallettsville, a member of the Texas delegation. Dr. Rouse has served for seven years as a presiding officer of the House of Delegates, four years as vice speaker and now is in his third term as speaker.

Other members of the Texas delegation include Dr. G. W. Cleveland of Austin, Dr. John L. Otto of Galveston, Dr. T. C. Terrell of Fort Worth, Dr. J. C. Terrell of Stephenville, Dr. R. W. Kimbro of Cleburne, Dr. J. L. Cochran of San Antonio, Dr. James H. Sammons of Highlands, Dr. Ridings E. Lee of Dallas, Dr. E. P. Hall, Jr., of Fort Worth, Dr. Denton Kerr of Houston, Dr. R. D. Moreton of Houston, Dr. Walter Walthall of San Antonio, Dr. J. E. Miller of Dallas, Dr. George Turner of El Paso, Dr. N. L. Barker of Paris, and Dr. Haden E. McKay, Jr., of Humble.

An open house honoring Dr. and Mrs. Rouse will be given by the Texas delegation Wednesday evening at the Palmer House.

The Texans also plan to present a resolution on the "Principles of Individual Responsibility" to the AMA House of Delegates.

The resolution stems from action taken at the Texas Medical Association's Annual Session in April, endorsing as ethical the right of the individual physician to participate voluntarily in an individual responsibility program to prevent third party interference in the doctor-patient relationship.

The AMA House of Delegates will convene at 3 p.m. Sunday, June 26, in Arie Crown Theatre at McCormick Place. The scientific program will open at 9 a.m. Monday, June 27, in McCormick Place, and exhibits will open at 10 a.m. Sunday, June 26, in McCormick Place and will be open Monday through Thursday from 8:30 a.m. to 5 p.m.

General registration will be in the lower lobby of McCormick Place, beginning at 10 a.m. Sunday and continuing from 8:30 a.m. to 5 p.m. on following days.

Topics of the six general scientific sessions include emphysema, population expansion, burns, mysterious fevers, community hospitals and coronary care units, and headaches.

Another highlight will be the Sixth Multiple Discipline Research Forum. Topics will include hematology, immunology, and tumor; gastrointestinal problems; metabolism and renal diseases; neurology and pulmonary diseases; and cardiovascular subjects.

The Conference of Presidents and Other Officers of State Medical Societies will open Sunday, June 26, at 9:30 a.m. at the Palmer House, instead of meeting in the afternoon as usual. Speakers will be Dr. Angus Murray, president of the Australian Medical Association, who will relate the Australian doctors' story of defeating the government program to fully socialize the medical profession of Australia; and Mr. Richard M. Nixon, former vice president of the United States.

Meeting simultaneously with the AMA will be the Woman's Auxiliary to the American Medical Association, holding its 43rd Annual Convention at the Drake Hotel, Chicago. Registration opens Sunday, June 26, at 11 a.m., and the convention formally opens at 9 a.m. Monday, June 27.

Also planned to coincide with the AMA and Auxiliary sessions is a five-day and two-evening program for physicians' pre-teens (ages 6-12) and teens. The activities, handled by Gulliver's Trails, Inc., are under the auspices of the Woman's Auxiliary.

Physician's Fee, Hospital Bill Should Be Separate Charges

Three committees of the Texas Medical Association have been assigned to help implement a plan mutually acceptable to physicians, hospitals, and insurance companies whereby the fees of hospital-based physicians can be established, billed, and collected separately rather than as a part of the total hospital bill.

A statement adopted by the TMA House of Delegates in April urges all Association members to strive for legal and ethical arrangements for separation of professional fees and hospital charges. The public needs to be informed regarding such changes. All county medical societies have received copies of the statement on fees for professional services, and have been urged to assist in implementing this statement of policy through appropriate legal, ethical, and communications channels available.

The Committees on Health Insurance and Liaison with Blue Shield of Texas are directed to "actively encourage the insurance industry to make such provisions in their contracts," and a special Committee for Liaison with the Texas Hospital Association is designated "to continue discussions with representatives of the Texas Hospital Association to evolve solutions to the technical problems associated with this change."

President James D. Murphy has appointed the following members of the special committee: Dr. Robert Mayo Tenery, Waxahachie, chairman, and Drs. Robert L. Daily, Wichita Falls; Mylie E. Durham, Jr., Houston; James A. Hallmark, Fort Worth; E. A. Maxwell, San Antonio; Harvey Renger, Hallettsville; and R. W. Kimbro, Cleburne.

The House of Delegates stipulated that the standing Committee on Liaison with Blue Shield of Texas be reactivated, and appointees are Dr. Renger, chairman, and Drs. Durham, Kimbro, Olin B. Gober, Temple; Dewey W. Johnston, Fort Worth; Denton Kerr, Houston; Jack D. Ramsey, Abilene; and Alvin O. Severance, San Antonio.

Members of the Committee on Health Insurance are Dr. Ernest A. Maxwell, San Antonio, chairman, and Drs. Jack L. Eidson, Weatherford; Norman West, Hillsboro; Mario E. Ramirez, Roma; George L. Walmsley, Baytown; John Douglas, Austin; Billy B. Markette, San Antonio; John Q. Thompson, Temple; and Porter K. Mason, Dallas.

The corporate practice of medicine is illegal in Texas, and the policy statement specifies that no member of TMA should engage in practices which dispose of his professional attainments or services to any hospital, corporation, or lay body by whatever name called under conditions which permit the sale of his services by such agency for a fee. It also expresses official disapproval of any arrangement under which any member of the Association merges his professional fee with hospital costs into a single charge to the patient.

San Antonio Television Shows Medics at Work

A television series, "Doctors at Work," sponsored by the Bexar County Medical Society, is being shown weekly on Channel 12 in San Antonio.

The half-hour programs, telecast each Sunday at 2:30 p.m., include a film presentation of doctors at work and a panel discussion by local physicians.

The series which began on May 1 will continue through Aug. 7.

Topics scheduled include leukemia, June 19; accidents, June 26; medical examination of the eyes, July 3; heart attack, July 10; cancer of the colon, July 17; alcoholism, July 24; bursitis, July 31; and convulsions, Aug. 7.

Molecular Medicine Complex To Be Built

Plans for a multimillion dollar research, treatment, and teaching complex to be known as the Wadley Institutes of Molecular Medicine have been announced by the Wadley Research Institute and Blood Bank of Dallas.

The new complex will be located in Leland Fikes Research Center on Harry Hines Boulevard near Dallas Love Field. Construction on the first three buildings, hospital, computer institute, and educational building, will begin this summer, at a cost exceeding 10 million dollars.

The center, believed to be the first of its kind in the world, will make possible a broad scale new attack against cancer and related diseases through the approach of molecular medicine, the study and treatment of the body's molecules.

In addition to the hospital, computer science institute, and educational center for which funds or a portion of needed funds have been donated, the new Wadley Center also will include in its initial phase a medical science center composed of research laboratories and related facilities and an institute of hemotherapy devoted to research on blood and blood products.

Other units will be added as the need arises and funds are acquired, according to Dr. Joseph M. Hill, director of Wadley. Eventually the new center will replace entirely the present Wadley Research Institute in Dallas. For the present, Wadley Blood Bank and its blood bank laboratories will remain at their old location.

Texas 65 Insurance Waives June Premium

The 64 member companies of the Texas 65 Health Insurance Association last month voted to waive the June premium of the 32,000 current enrollees in the program, while continuing their coverage until July 1.

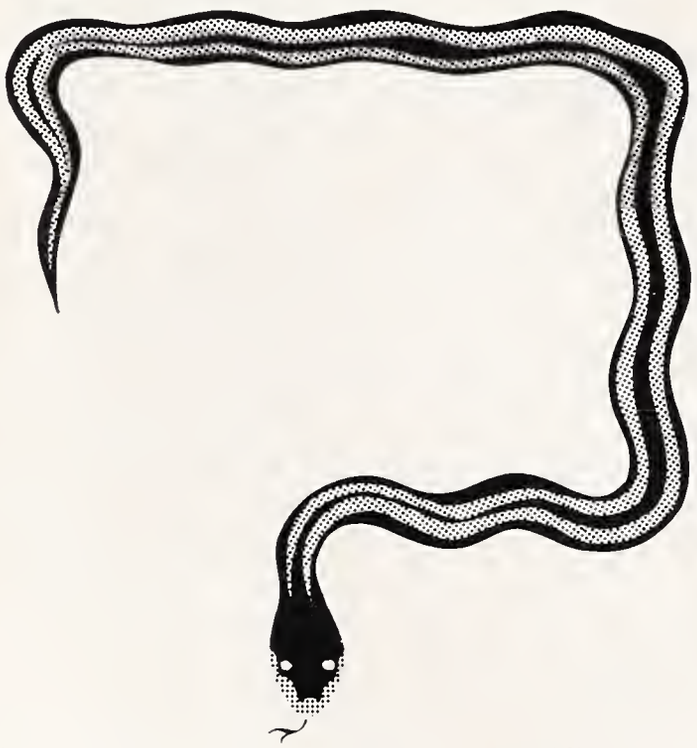
The Association announced that it had collected its last premium. Authorized by the 1963 Texas Legislature to offer health insurance coverage to all Texans past the age of 65, the Association will be replaced by Medicare on July 1.

Participants in the program were notified in January that all coverage would cease on July 1, but that Texas 65 would remain in operation past that date to process claims incurred before Medicare became effective.

Though Texas 65 was established to offer health insurance to Texans over the age of 65, it has insured the under-age-65 husbands and wives of those enrolled. So that these Texans will not be without health insurance protection until they become eligible for Medicare, the Southland Life Insurance Company of Dallas, one of the 64 member companies of Texas 65, has agreed to offer them identical coverage to Texas 65, at the same price they are now paying. There are about 1,000 enrollees, aged 45-65, in this group.

"In 40 of 44 cases of irritable or spastic colon, Cantil [mepenzolate bromide] or Cantil with Phenobarbital reduced or abolished abdominal pain, diarrhea and distention and promoted restoration of normal bowel function . . . Cantil [mepenzolate bromide] proved to be singularly free of anticholinergic side-effects . . . Urinary retention, noted in two cases was eliminated in one by reducing dosage."[1]

CHARMS THE HYPERACTIVE COLON



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helps restore normal motility and tone

IN BRIEF:

One or two tablets three times a day and one or two at bedtime usually provide prompt relief. Cantil with Phenobarbital may be prescribed if sedation is required.

Dryness of the mouth or blurring of vision may occur but it is usually mild and transitory. Urinary retention is rare. Caution should be observed in prostatic hypertrophy—withhold in glaucoma. Cantil with Phenobarbital is contraindicated in patients sensitive to phenobarbital.

Supplied: CANTIL (mepenzolate bromide) —25 mg. per scored tablet. Bottles of 100 and 250. CANTIL with PHENOBARBITAL —containing in each scored tablet 16 mg. phenobarbital (warning: may be habit forming) and 25 mg. mepenzolate bromide. Bottles of 100 and 250.

1. Riese, J.A.: Amer. J. Gastroent. 28:541 (Nov.) 1957

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Alcohol Studies Institute To Convene in Austin

Some 300 participants are expected to attend the Ninth Annual Institute on Alcohol Studies at the University of Texas in Austin this summer, July 24-29.

Experts will lecture on medical, social, psychological, governmental, and clerical aspects of the drinking problem. The latest data on coping with the problem will be aired. Special interest forums will focus on social welfare, industry, education, law enforcement, and other fields.

The Institute is sponsored by the University of Texas Division of Extension and the Texas Commission on Alcoholism. Cooperating are the Texas Education Agency and the Coordinating Board of the Texas College and University System, according to Macon W. Freeman, executive director of the Texas Commission on Alcoholism.

Among the leaders at the Institute will be Miss Mary Ann Faulkner, Louisiana authority who works as clinic administrator and supervisor of social work in the state's alcoholism clinic in New Orleans; Dr. Milton Maxwell, executive director of the Rutgers University Summer School of Alcohol Studies; and Dr. Shervert Frazier, newly appointed commissioner of the Texas Board of Mental Health and Mental Retardation.

Registration information for the Institute may be obtained from the 22 local Councils on Alcoholism throughout Texas, or from the Texas Commission on Alcoholism, 808 Sam Houston State Office Building, Austin 78701.

Information may also be obtained on how to apply for scholarships to defray the cost of tuition.

Two Texans in Nicaragua With S.S. Hope Mission

Two Texas physicians, Dr. Robert R. Shaw of Dallas and Dr. Lewis M. Helfer of San Antonio, have begun a tour of voluntary service aboard the S.S. Hope, the famed white hospital ship now on a mission to Nicaragua.

Dr. Shaw, professor of thoracic surgery and chairman of the Division of Thoracic Surgery at the University of Texas Southwestern Medical School, was the recipient of the Bruce Douglas Award presented by the Michigan Thoracic Society in 1963 and the Marchman Award presented by the Dallas Southern Clinical Society in 1964.

Dr. Helfer is an associate clinical professor of neurosurgery in the University of Texas Postgraduate School of Medicine.

The two Texans are among 27 volunteers participating in the current teaching-treatment tour. The Hope will remain in Nicaragua until November. Since arriving in mid-January, Hope doctors, nurses, and technologists have been working with their Nicaraguan counterparts, sharing their knowledge of modern medical techniques.

Founded in 1958 by Dr. William B. Walsh, Project Hope is the principal activity of The People-to-People Health Foundation, Inc., Washington, D.C. The Project maintains land programs on the three continents visited by the ship, continuing Hope's work after the vessel has departed.

Astronauts Slated For Panel Talks

A panel discussion on "Cardiopulmonary Aspects of Space Travel" will highlight the 32nd Annual Meeting of the American College of Chest Physicians in Chicago June 23-27.

The panel discussion, to be held on June 25, will be moderated by Dr. Charles A. Berry, chief of Center Medical Programs, NASA Manned Spacecraft Center, Houston.

Featured on the panel will be astronauts Col. Frank Borman, USAF, and Capt. James Lovell, USN. Also participating on the panel will be Lt. Col. John Ord, USAF, director of Crew Test and Evaluation, Headquarters AMD, Brooks Air Force Base, Texas, and Dr. Stuart Bondurant, associate professor of medicine, Indiana University Medical Center, Indianapolis.

The five-day meeting, which precedes the American Medical Association's Annual Session in Chicago, will include other panel discussions, seminars, round table discussions, scientific motion pictures and formal papers.

The AMA's Section on Diseases of the Chest and the American College of Chest Physicians will hold a combined meeting at McCormick Place on Monday, June 27. The Chest Physicians' "Fireside Conferences" will be held that evening at the Sheraton-Chicago Hotel as part of the combined meeting.

Information on both sessions may be obtained from Mr. Murray Kornfeld, Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago, Ill. 60611.

Cancer Conference Set In Denver on July 15-16

The 20th annual Rocky Mountain Cancer Conference will be held July 15-16 at the Brown Palace Hotel in Denver, Colo., featuring some of the nation's most distinguished speakers on the subject of cancer.

Morning sessions for the two-day conference will deal with "Malignancies and Allied Diseases of the Chest" and "Diagnostic Methods and the Frequency of Their Utilization for All Forms of Cancer in the General Practice of Medicine and Surgery."

The afternoon session of the first day will be devoted to scientific papers by guest speakers, and the second afternoon will feature an "Information Please" session.

The conference is cosponsored by the Colorado Division of the American Cancer Society and the Colorado Medical Society.

Further information may be obtained from the Rocky Mountain Cancer Conference, 1809 East 18th Avenue, Denver, Colo. 80218.

Texas Youth Wins Science Fair Award

A Texas boy won one of the ten Award of Merit certificates presented by the American Medical Association at the 17th International Science Fair in Dallas last month.

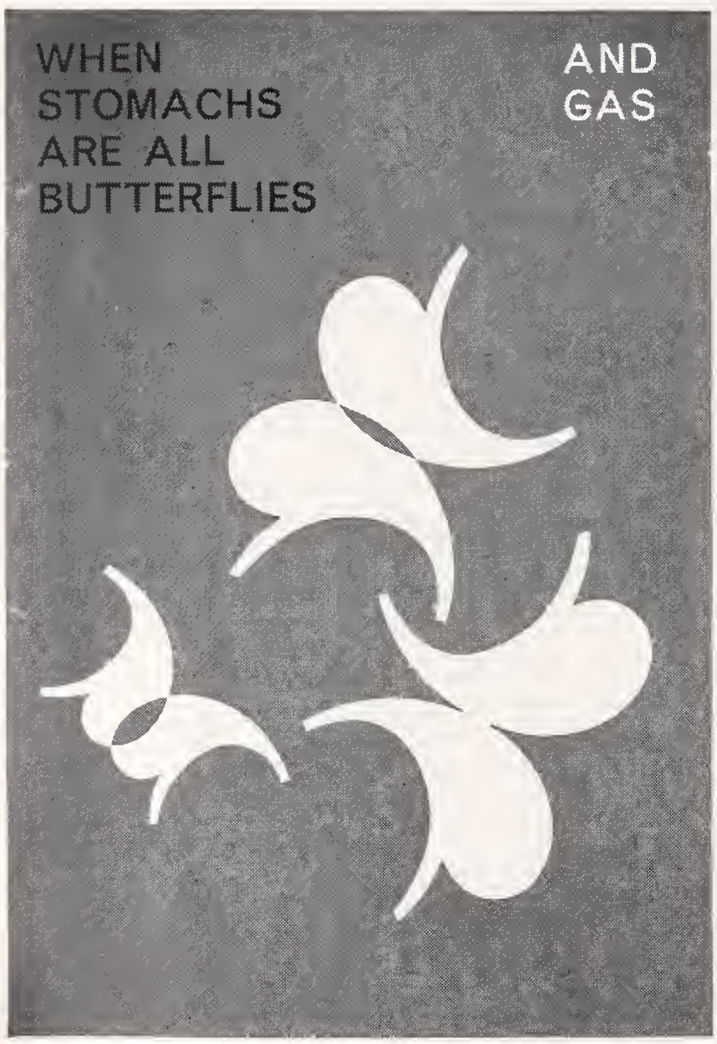
Edwin Ronnie Collier, 15, of Mesquite, received the award for his study on "The Immunological Study of the Thymus Gland and Its Role in Transplanting."

A committee from the AMA Council on Postgraduate Programs selected the winners from 419 finalists, and Dr. James Z. Appel, AMA President, made the presentation at the health awards program.

DACTILASE®

Each tablet contains:
Dactil® (piperidolate hydrochloride), 50 mg.;
Standardized cellulolytic* enzyme, 2 mg.;
Standardized amylolytic enzyme, 15 mg.;
Standardized proteolytic enzyme, 10 mg.;
Pancreatin 3X** (source of lipolytic activity), 100 mg.; Taurocholic acid, 15 mg.

*Need in human nutrition not established.
**As acid resistant granules equivalent in activity to 300 mg. Pancreatin N.F.



In chronic or acute indigestion, fluttery, gassy stomachs obtain prompt, gratifying relief through the antispasmodic, surface anesthetic and enzymatic activity of Dactilase. Dactilase decreases hypermotility and pain and reduces the production of gas. Dactilase does not induce stasis, but helps restore normal tone. It has little or no effect on enzyme secretions, but *adds* enzymes, thus contributing to the digestive efficiency of the patient.

Side Effects and Contraindications: Dactilase is almost entirely free of side effects. However, it should be withheld in glaucoma and in jaundice due to complete biliary obstruction.

Administration and Dosage: One tablet with, or immediately following, each meal. Tablets should be swallowed whole.

Supplied: Bottles of 60 and 250.

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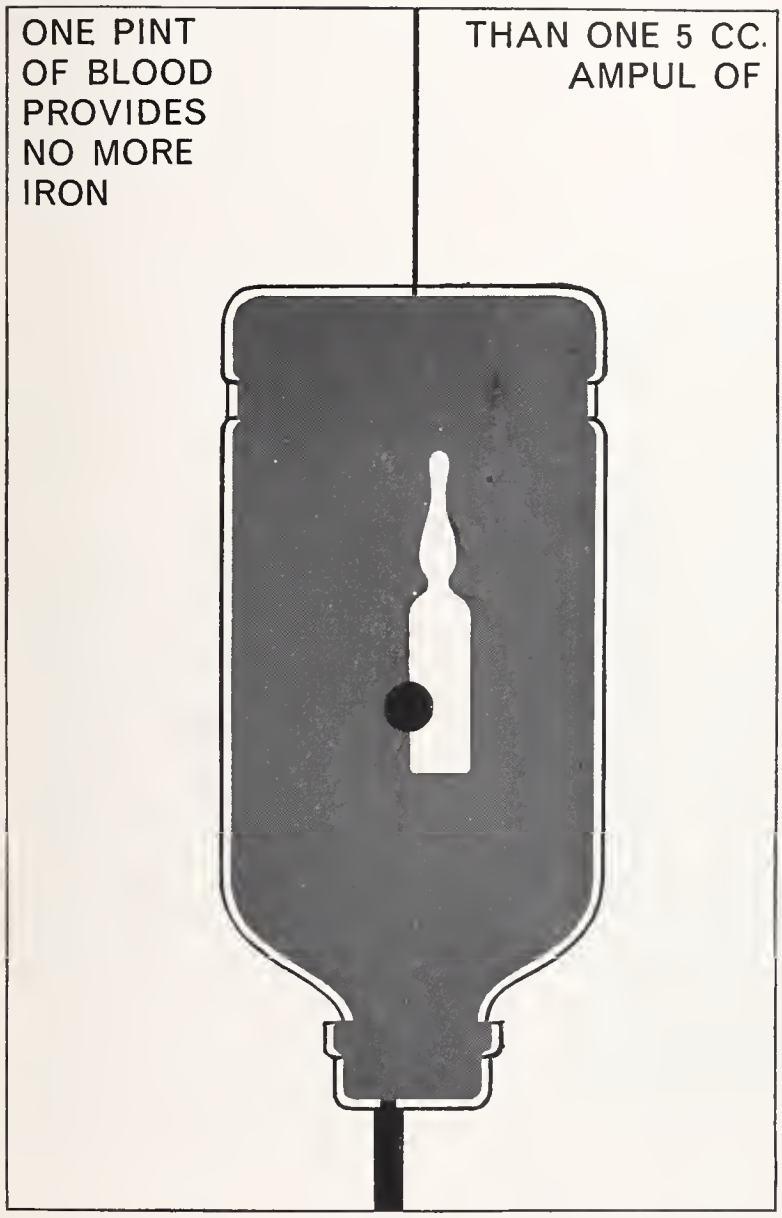
Obstetrical or post—surgical patients requiring a dependable increase in hemoglobin will receive as much iron (250 mg. in a 5 cc. ampul) as in one pint of blood. Imferon (iron dextran injection) is less expensive and it avoids the well-recognized hazards of whole blood transfusion. When patients cannot—or cannot be relied upon to—take oral iron, Imferon (iron dextran injection) will rapidly supply needed iron for reserve stores.



IMFERON®
(iron dextran injection)

IN BRIEF: ACTION AND USES: A single dose of Imferon (iron dextran injection) will measurably begin to raise hemoglobin and a complete course of therapy will effectively rebuild iron reserves. The drug is indicated only for specifically-diagnosed cases of iron deficiency anemia and then only when oral administration of iron is ineffective or impractical. Such iron deficiency may include: patients in the last trimester of pregnancy; patients with gastrointestinal disease or those recovering from gastrointestinal surgery; patients with chronic bleeding with continual and extensive iron losses not rapidly replenishable with oral iron; patients intolerant of blood transfusion *as a source of iron;* infants with hypochromic anemia; patients who cannot be relied upon to take oral iron.

COMPOSITION: Imferon (iron dextran injection) is a well-tolerated solution of iron dextran complex providing an equivalent of 50 mg. in each cc. The solution contains 0.9% sodium chloride and has a pH of 5.2-6.0. The 10 cc. vial contains 0.5% phenol as a preservative.

ADMINISTRATION AND DOSAGE: Dosage, based upon body weight and Gm. Hb./100 cc. of blood, ranges from 0.5 cc. in infants to 5.0 cc. in adults, daily, every other day, or weekly. The total iron requirement for the individual patient is readily obtainable from the dosage chart in the package insert. Deep intramuscular injection in the upper outer quadrant of the buttock, using a Z-track technique, (with displacement of the skin laterally prior to injection), insures absorption and will help avoid staining of the skin. A 2-inch needle is recommended for the adult of average size.

SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylactoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported. Initial test doses of 0.5 cc. are advisable.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

CONTRAINDICATIONS: Imferon (iron dextran injection) is contraindicated in patients sensitive to iron dextran complex. Since its use is intended for the treatment of iron deficiency anemia only it is contraindicated in other anemias.

CARCINOGENICITY POTENTIAL: Using relatively massive doses, Imferon (iron dextran injection) has been shown to produce sarcoma in rats, mice and rabbits and possibly in hamsters, but not in guinea pigs. The risk of carcinogenesis, if any in man, following recommended therapy with Imferon (iron dextran injection) appears to be extremely small.

SUPPLIED: 2 cc. ampuls, boxes of 10; 5 cc. ampuls, boxes of 4; 10 cc. multiple dose vials.

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In colicky infants Pediatric Piptal with Phenobarbital slows down spasm, diminishes pain and crying and improves feeding patterns. It permits sleep and rest for patient and family. The less than hypnotic amount of phenobarbital in the recommended dose affords a mild, calming action and enhances the antispasmodic action of Piptal (pipenzolate bromide). The latter drug, as reported in the medical literature, has a favorable ratio of effectiveness to side-effects which is unusual in anticholinergics and thus is particularly appropriate to pediatric use.

PEDIATRIC PIPTAL® WITH PHENOBARBITAL

each cc. contains 6 mg. phenobarbital (warning: may be habit forming); 4 mg. Piptal® (pipenzolate bromide), and 20% alcohol.

Pleasant-tasting Pediatric Piptal with Phenobarbital is miscible in milk, formulas and fruit juices, and may also be given by dropper directly on the infant's tongue. Dosage is 0.5 cc. 15 minutes before feeding; in severe cases, 1.0 cc. four times daily. High doses may occasionally cause constipation with tenesmus and, rarely, flushing without fever. It is contraindicated in bowel obstruction or sensitivity to phenobarbital or anticholinergics. Available in 30 cc. dropper bottles, droppers calibrated to deliver 0.5 cc.

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A New Tool for Interprofessional Cooperation

OUR "Code for Physicians and Attorneys of Texas," in existence for many years, was amended by the Texas Medical Association House of Delegates and approved by the Board of Directors of the State Bar of Texas on April 15. A replacement of the original section on "Medical Reports," this amendment was the result of the joint effort of the Association's Committee on Liaison with the State Bar of Texas and a similar committee from the State Bar.

Entitled "Medical Reports," this newly adopted section reads as follows:

> It is recognized that the physician-patient relationship is a confidential one and that a physician should not give medical information concerning a patient except on proper authority. Those medical reports already in the physician's office are to be furnished at a reasonable charge for clerical service to the requesting party, and any requests which require additional examinations or tests should be paid for by the requesting party.
>
> Nothing in this section shall be construed as requiring the patient's consent for the physician to render medical reports, as required by law, concerning a patient treated or examined to the party paying for such examination or treatment. The patient or his representative shall be furnished a copy of the report upon written request.

The Attorney

Requests to the physician for medical records should be made in writing and be made at such a time as to give the physician reasonable and adequate notice of the need of such reports. If a report is requested on a physician's patient, the attorney should provide the physician with written authorization from the patient. The attorney should be specific in his requests for records and should specify the specific conditions about which he seeks information and should likewise indicate whether he is asking for a prognosis or not.

Suggested Procedure

When the attorney for the defendant arrives at that stage of the case where he requires the records of the patient's doctor he should jointly notify the doctor and the patient's lawyer. At this point, the plaintiff's lawyer, knowing full well that the doctor is subject to subpoena, should do two things. First, notify the patient that the patient's records must go to the defendant's lawyer, explaining to the patient that all hospital and doctor records are available by subpoena. Secondly, obtain his client's written consent for release of the records and deliver this release to the doctor. Explain to the patient that such a release gives the doctor more time to prepare his records and present them properly when not under strain of a subpoena.

The Physician

The physician should have the patient's written authorization, or legal process, before releasing any report or test concerning a patient. Properly authorized written requests should be honored promptly. Undue delays in providing medical reports or bills bearing on a patient's legal rights may prejudice his case. The physician must keep records adequate to supply a patient's attorney all pertinent information regarding the patient-client's medical history.

Recognition of two important factors led to the revision of the Medical Reports section.

1. The physician-patient relationship is private and confidential and should not be breached in any manner by the physician without proper written consent or authorization from the patient.

2. Any medical record or report may be "discovered" by legal process if the report is even vaguely germane to a pending lawsuit, though the legal process for such discovery may be lengthy and expensive.

The new section on "Medical Reports" recognizes these important and vital aspects of the law. This new section, in our opinion, sets forth the procedure which will simplify the obtaining of those records which can be legally secured anyway and, at the same time, protects the delicate and all-important physician-patient relationship.

A careful reading of the "Suggested Procedure" portion under "The Attorney" indicates that the main burden for securing patient records rests, as it should, upon the attorney representing the claimant. However, the suggested procedure may not always be followed exactly and it is important for the physician to realize that his main concern should be whether or not his patient has, either through his attorney or directly, authorized, in writing, the physician to release medical records or reports. Once the physician receives such written authorization, he should forthwith provide the records requested, insuring at the same time that he retains original records for his use. Without written authorization from the patient, the physician should refuse to permit another individual to view the medical records of a patient.

As suggested in the first paragraph of the section, office records "are to be furnished at a reasonable charge for clerical service to the requesting party." The amount of this "reasonable charge" must reflect a *good-faith* determination by the doctor himself. It is no secret that in the past a few physicians have attempted to charge fees for this service which were unconscionably high and could not in any way be described as a "reasonable charge" for the work performed or time consumed.

As the second paragraph indicates, the amended code doesn't seek to make any change in the present procedure under the Workmen's Compensation Law, which

specifically provides for medical examination of a claimant by a doctor selected by the insurance carrier. The law holds that when a person files a claim under this Act, he consents, by implication, to the furnishing of such reports.

If this voluntary method for disclosure of records is not followed and a subpoena for certain records is properly served upon the physician, then the recipient of that subpoena has no choice but to appear in court and formally ask the presiding judge to "quash" the subpoena or to produce those records set out in the subpoena. If the physician merely disregards the subpoena in the mistaken belief that the medical records constitute a "privileged communication" between patient and physician, he is misreading the law in Texas and is subjecting himself to possible penalties for contempt of court.

Thus, this amended section on medical reports provides a safe and simple method for the disclosure of records which may otherwise be discovered by a legal process—which costs the patient money and the physician time. As in the case of any voluntary and mutually agreed upon procedure, the Code is no magic wand which will eliminate all problems between doctors and lawyers. It is, as the Code says, "designed to achieve a better understanding between the medical and legal professions." It can only achieve this goal when both physicians and attorneys seek, in good faith, to utilize its provisions for the overall benefit of their patients and clients.

—Philip R. Overton, LL.B., and Sam V. Stone, Jr., LL.B., Austin, Legal Counsel, Texas Medical Association.

"We're having trouble packaging that new dermatologic cure-all—it keeps dissolving the bottle."

Physical Therapy Group Sets July Conference

The American Physical Therapy Association will hold its 43rd Annual Conference July 10-15 at the Biltmore Hotel in Los Angeles, Calif.

Physicians, nurses, occupational therapists, speech therapists, prosthetists and orthotists, and members of other allied health professions are invited to attend the program sessions and exhibits. Non-member registration fee is $25.

Additional information may be obtained from Helen J. Hislop, Ph.D., Director of Conference Services, American Physical Therapy Association, 1790 Broadway, New York City 10019.

Berry Plan Deadline Aug. 1 for '66 Graduates

The deadline for applying for participation in the armed forces' Physicians' Appointment and Residency Consideration Program (Berry Plan) has been set for Aug. 1, according to the *AMA News*.

Medical students who will graduate this spring must apply by that date to the US Department of Defense to take part in the Berry Plan, which makes it possible for physicians who are liable for active military duty to apply for a Reserve commission. With the Reserve commission, the new physicians may be called to duty at a time mutually acceptable to both the doctor and the branch of service for which he has volunteered.

The program also permits the deferment of a selected number of interns who will be permitted to complete residency training before being called to active duty.

Under the Berry Plan, 1966 medical school graduates may choose active duty immediately upon completion of internship; active duty as late as one year following completion of internship; or deferment for residency training in specialties required by the armed forces.

A "Statement of Preference," which must be filed with the Assistant Secretary of Defense, Health and Medical, in Washington before Aug. 1 is included in a booklet describing the program, which has been mailed to all draft-eligible 1966 medical graduates.

Drug Production Seminar Scheduled for July 17-22

The second seminar on Control Procedures in Drug Production will be held July 17-22 at the Hershey Hotel in Hershey, Pa., stressing "current good manufacturing practices" and bringing together the best information available to university, government, and business on quality controls in drug production.

Sponsors of the seminar are the University of Wisconsin's School of Pharmacy and the Extension Services in Pharmacy of the University Extension Division, in cooperation with the Pharmaceutical Manufacturers Association and the US Food and Drug Administration of the Department of Health, Education, and Welfare.

Participants will be limited to 60 representatives from PMA member firms and 40 others, chosen from quality control, production, and engineering personnel. Meetings will be closed, but proceedings will be published.

Application forms can be obtained from Dr. William L. Blockstein, The University of Wisconsin, Extension Services in Pharmacy, 190 Pharmacy Building, Madison, Wis. 53706.

Birth Defects Center Now in Second Year

The Birth Defects Center of the Department of Pediatrics, the University of Texas Medical Branch, Galveston, is now in its second year of operation, through the financial assistance of the Texas Chapter, The National Foundation—March of Dimes.

Any child under 15 years of age who is a resident of Texas and has a birth defect may be referred to the Center for evaluation.

Financial arrangements at the Center are handled several ways. Patients able to meet medical expenses may be accepted on a sliding fee basis. Many children qualify for financial aid from state and local agencies. Limited and prior financial arrangement for patients not meeting these two requirements may be made by special agreement with the patient's local chapter of the National Foundation—March of Dimes.

Patients may be referred from

licensed physicians and authorized agencies. Referrals should include a summary of prior medical care received by the patient. In emergency situations direct telephone referrals are accepted.

To initiate referrals, physicians should contact the Medical Coordinator, Birth Defects Program, Department of Pediatrics, The University of Texas Medical Branch, Galveston 77551.

Effective Rubella Vaccine In Experimental Stage

The first effective experimental vaccine against rubella (German measles) has been developed by two scientists of the US Public Health Service's National Institutes of Health.

Developers of the vaccine, Dr. Harry M. Meyer, Jr., and Dr. Paul D. Parkman, both of the Division of Biologics Standards, reported their findings at a recent meeting of the American Pediatric Society in Atlantic City.

Surgeon General William H. Stewart of the USPHS predicted the control of rubella and its associated birth defects in the "not too distant future," as a result of the newly developed experimental vaccine.

"Until now, experimental live rubella preparations have caused typical rubella, and the infection was communicable to uninoculated contacts," Dr. Stewart stated.

In experiments with the latest vaccine, inoculated children developed immunity with no accompanying fever or rash, and the control group was not infected by contact with the inoculated group.

To test the virus, the two pediatricians-virologists worked with the Department of Pediatrics of the University of Arkansas Medical School and the Arkansas Children's Colony.

Austin Doctor To Serve Clinic in Guatemala

Dr. Alfred J. Kelly, Austin general practitioner, will take his family to Guatemala in July for a month's service at the Maryknoll Mission Clinic in San Pedro Necta.

The clinic, which has a new 28-bed hospital, needs doctors for similar service for the months of June and August, according to Dr. Kelly. Calling the clinic duty "a real opportunity to serve God and country in medical care and goodwill where it is badly needed," Dr. Kelly pointed out that a house is provided for the volunteering doctor and his family.

San Pedro Necta, in Huehuetenango Province, is the first town across the border from Mexico on the main highway. Dr. Kelly said the trip could be made by automobile in four or five days or by air in three or four hours.

Two registered nurses, he said, have just arrived from the United States. The doctor's typical daily schedule includes seeing 35 to 50 patients with the help of five nurses, the sisters, and other assistants.

Additional information may be obtained from the Catholic Medical Mission Board, Inc., 10 West 17th Street, New York, N.Y. 10011, or from the Rev. Joseph Rickert, M.M., San Pedro Necta, Huehuetenango, Guatemala. Dr. Kelly can be reached by telephone in Austin, at GReenwood 2-1315.

Interns, Residents To Receive VA Aid

Aid for military veterans holding approved internships and residencies is now official, according to the *AMA News*.

Final Veterans Administration regulations now state that veterans can receive the new GI Bill of Rights benefits as interns and residents as well as in medical school.

Each month of active duty at any time since Jan. 31, 1955, qualifies a student for an equal number of monthly benefits ($100) for a maximum of 36 months.

Youth Opportunity Summer Jobs Noted

The National Aeronautics and Space Administration has announced that it will hire this summer several hundred young men and women over its normal summer employment levels, as part of the President's Youth Opportunity Program.

At the same time, NASA called upon private business and industrial employers to follow NASA's lead in hiring young people this summer, to make the program fully effective.

Through the youth program last summer, at the President's request, one million young people found jobs during the months they were out of school. The situation for youth ages 16 through 21 seeking summer jobs is as serious this year as it was last.

"Getting those jobs may be the difference between being able to go back to school or not going back," President Johnson pointed out.

Under the program, employers hire for the summer one extra trainee for every 100 employees on the present payrolls. The President emphasized that the program would be "worthless or worse" if it replaced regular employment opportunities.

Employers and organizations willing to cooperate in the program should advise the nearest Federal-State Employment Office. All boys and girls, 16 through 21, who want to work this summer and who don't have jobs may contact the nearest employment service office.

USPHS Offers Student Aid, School Improvement Grants

Educational improvement grants and scholarship grants are now available to schools of medicine, dentistry, optometry, osteopathy, and podiatry as a result of the Health Professions Educational Assistance Amendments of 1965. Scholarship grants are also available to schools of pharmacy, Surgeon General William H. Stewart of the US Public Health Service has announced.

Basic educational improvement grants are intended to be used by schools to increase the quality of their educational programs. To be eligible for a grant, the school must assure the USPHS that it will increase its enrollment. For the current fiscal year, each recipient school is allowed $12,500, plus $250 for each full-time student enrolled.

Scholarship grants will be used by schools to provide assistance to students from low-income families who would not be able to attend a health professions school without financial support. Scholarship students, usually full-time first-year students entering this fall, may receive up to $2,500.

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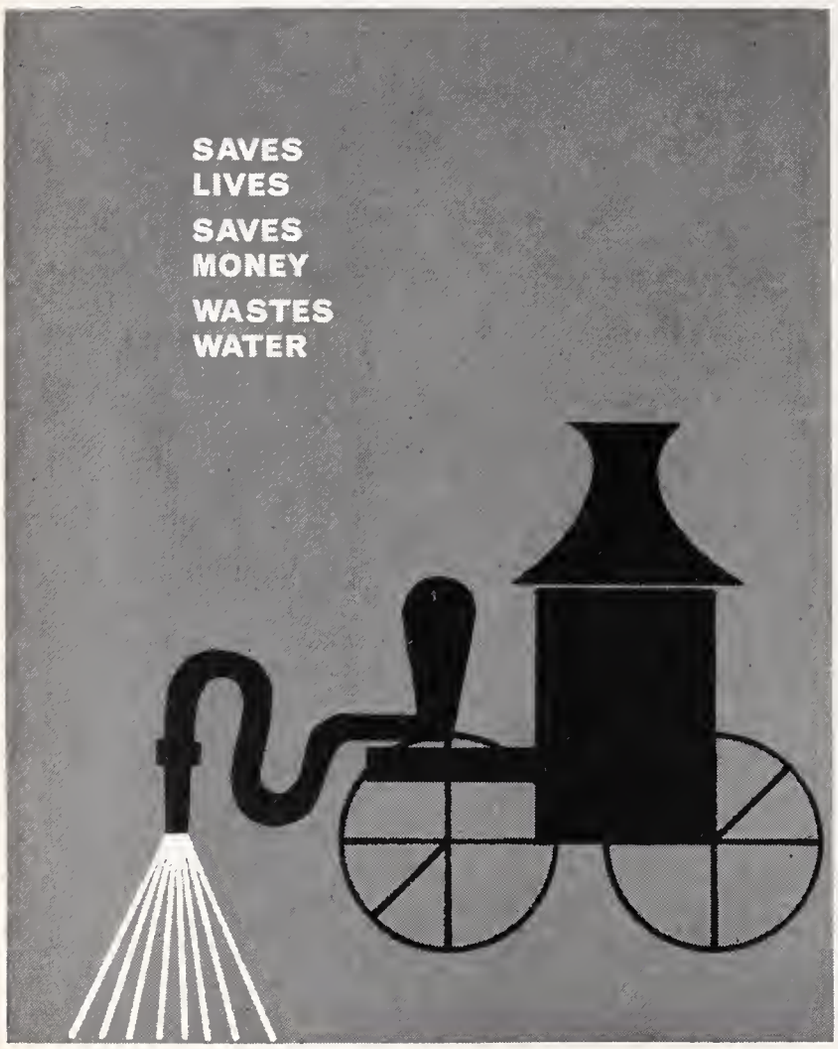

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Side Effects: Nausea, flushing, constipation, skin rash, muscle cramps and gastric discomfort have occasionally been noted; rarely thrombocytopenia and bone marrow depression, photosensitivity, cholestatic jaundice, pancreatitis, perimacular edema, gout and diabetes have been caused by the administration of thiazides.

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July Postgraduate Assembly To Be Held in Houston

The Postgraduate Medical Assembly of South Texas will be held July 18-20 at the Shamrock Hilton Hotel in Houston, featuring programs of interest to general practitioners and specialists presented by outstanding medical and surgical specialists in both lecture and panel form.

Attorney General Waggoner Carr will be guest speaker at the assembly's luncheon on Monday, July 18. Tuesday's luncheon speaker will be Dr. Charles J. Frankel, associate professor of orthopedic surgery at the University of Virginia and editor-in-chief of the Lawyers' Medical Cyclopedia. He will discuss "The Doctor and the Law." On Wednesday "The REAL Difference Between Freedom and Communism" will be discussed at the luncheon by Mr. Val Gould, representative of the Quaker Oats Company.

Among the Texas speakers at the assembly will be Dr. Norman M. Kaplan, assistant professor of medicine and chief of the Endocrine Clinic, University of Texas Southwestern Medical School, Dallas, who will speak on "Primary Aldosteronism and Essential Hypertension," "Problems of the Steroid Treated Patient," and "Obesity—Can the Curse be Cured?"

Also Dr. Marcel Patterson, professor of medicine, University of Texas Medical Branch, Galveston, speaking on "The Problem of Chronic Diarrhea," "Functional Gastrointestinal Disease," and "Gastrointestinal Ulceration."

Also Dr. Lucius Waites, medical staff, Scottish Rite Hospital, and clinical associate professor of pediatrics, University of Texas Southwestern Medical School, Dallas, who will discuss "Medical Aspects of School Failures," "Reading and Writing Disabilities—Diagnosis and Training," and "The Role of Perceptual Disorders in Academic Failure."

Expected also are many other outstanding speakers from over the country and one speaker from Barcelona, Spain, Dr. Pascual Gonzalez-Marin, Director of Surgery of the Orbit and Lacrimal Passages, Barraques Institute.

In addition to lecture sessions, covering presentations in internal medicine, psychiatry, pediatrics, urology, orthopedics, dermatology, obstetrics and gynecology, ophthalmology, and otolaryngology, four panel discussions, "Pearl Sessions," will be held. These will cover "Menopause and Hormones. Why? What Kind? How Long? How Much?," "Gastrointestinal Ulceration," "Problems of the Steroid Treated Patient," and "The Present and Future of Organ Transplantation."

Advance registrations and requests for further information should be directed to the Postgraduate Medical Assembly of South Texas, 209 Jesse H. Jones Library Building, Houston 77025. The program is acceptable for 20 accredited hours by the American Academy of General Practice.

Postgraduate Courses

Neurology for the Internist, Winston-Salem, N.C., June 15-18.—The American College of Physicians Postgraduate Course No. 19 will be offered at the Bowman Gray School of Medicine of Wake Forest College.

The course is designed for internists and family physicians to assist with diagnosis and treatment of neurological problems. Particular attention will be directed at advances in diagnosis and management of cerebral vascular diseases.

One part of the course will be concentrated on disorders of consciousness and mentation, while another will include neurologic disorders related to metabolic abnormalities or other systemic diseases. The significance of headache, convulsions, and vertigo will be discussed, as will new diagnostic techniques.

In addition to the Bowman Gray faculty, guest faculty members will include Dr. Robert B. Aird, University of California School of Medicine; Dr. Malvin Cole, New Jersey College of Medicine and Dentistry; Dr. Thomas W. Farmer, University of North Carolina School of Medicine; Dr. Joseph M. Foley, Western Reserve University School of Medicine; Dr. Albert Heyman, Duke University School of Medicine; Dr. Aneel Patel, Western Reserve University School of Medicine and Cleveland Metropolitan General Hospital; Dr. Fred Plum, Cornell University Medical College; and Dr. E. Peirson Richardson, Jr., Harvard Medical School and Massachusetts General Hospital.

General Practice Review, Denver, July 18-23.—The 12th annual postgraduate course designed especially for the general practitioner will be held at the University of Colorado School of Medicine.

Physicians may register for the entire course or for any selected days, topics for which include: Monday, medicine; Tuesday, pediatrics; Wednesday, surgery; Thursday, trauma; Friday, obstetrics and gynecology; and Saturday, dermatology.

Other postgraduate courses of interest to the general practitioner slated by the school are pediatrics, Aug. 1-5, Estes Park, Colo.; internal medicine, Aug. 8-12, Estes Park; The Hospital Medical Staff Conference, Oct. 3-7, Estes Park; family planning and genetic counseling, Oct. 26-28, Denver; management of fractures, Nov. 2-4, Denver. These dates are subject to change.

Two other courses, premature infant care, and management and care of respiratory insufficiency, will be scheduled for one week, three times yearly.

For further information and detailed programs, write to The Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver, Colo. 80220.

Pamphlet Now Available On Premedical Education

"Premedical Education in Texas . . . A Need," a pamphlet compiled by the Texas Medical Association's Committee on Liaison with Medical Schools, recently was mailed to 1,500 high school superintendents and premedical advisers at junior colleges and colleges.

The pamphlet is designed as a guide to educators, premedical advisers, and physicians in giving the most effective advice to prospective medical students at the high school level.

Chairman of the committee which prepared the booklet is Dr. Russell L. Deter of El Paso.

Additional copies are available on request from the Texas Medical Association, 1801 North Lamar Blvd., Austin, Texas 78701.

Pamphlets

"Today's Facts About Arthritis," a newly revised pamphlet published by the Arthritis Foundation, is available free to anyone requesting a copy. It may be obtained from local chapters of the foundation or from the organization's headquarters, The Arthritis Foundation, Box 2525, New York, N.Y. 10001.

The pamphlet provides clear and precise basic information patients need to know about the many rheumatic diseases and answers a number of specific questions. It also emphasizes the great need for the patient to act intelligently with a physician in a well-regulated, individualized program of care and treatment.

"Hardening of the Arteries—Cause of Heart Attacks," a new, illustrated National Heart Institute leaflet, has been issued by the US Public Health Service.

The publication describes what an artery is, how arteries harden, trouble spots resulting from hardening of the arteries, and what a heart attack is and how it happens.

Single free copies of the leaflet (PHS Publication #1392) are available upon request from the Public Health Service, Department of Health, Education and Welfare, Washington, D.C. 20201. Multiple copies at $6.75 per 100 may be purchased from the Superintendent of Documents, Government Printing Office, Washington, D.C. 20402.

A 40-page booklet, entitled "Residencies for Physicians, Public Health Service Bureau of Medical Services," USPHS Publication #1408, describes opportunities for physicians to receive residency training in Public Health Service hospitals.

The hospitals, which have expanded their training programs, now have about 230 residency positions and further expansion is planned. Physicians may specialize in anesthesiology, dermatology, general practice, internal medicine, obstetrics and gynecology, ophthalmology, orthopedic surgery, pathology, pediatrics, preventive medicine, radiology, surgery, or urology.

Single copies may be obtained free from the Public Health Service, Department of Health, Education, and Welfare, Washington, D.C. 20201.

New Books In TMA Library

Ackerknecht, E. H.: *History and Geography of the Most Important Diseases*, New York, Hafner Publishing Co., 1965.

Dale, Sir Henry Hallett: *Adventures in Physiology*, London, England, Wellcome Trust, 1965.

Davis Memorial Lecture Series, University of Illinois College of Medicine: *Essays in the History of Medicine in Honor of David J. Davis, M.D., Ph.D.*, Chicago, University of Illinois Press, 1965.

Dewhurst, Kenneth: *John Locke, Physician and Philosopher*, London, England, Wellcome Historical Medical Library, 1963.

Dubois, E. L.: *Lupus Erythematosus*, New York, McGraw-Hill Book Co., 1966.

Hambling, J., and Hopkins, P.: *Psychosomatic Disorders in Adolescents and Young Adults*, London, England, Pergamon Press, Inc., 1965.

Heinz, H. J., Co.: *The Heinz Handbook of Nutrition*, ed. 2, New York, Blakiston Division, McGraw-Hill Book Co., 1965.

International Congress of Gerontology: *Age With a Future*, 6th Congress, Copenhagen, 1963, Philadelphia, F. A. Davis Co., 1964.

Kramer, C.: *Medical Malpractice*, rev. ed., New York, Practising Law Institute, 1965.

McLennan, C. E.: *Synopsis of Obstetrics*, ed. 7, St. Louis, C. V. Mosby Co., 1966.

Moss, B. B.: *Caring for the Aged*, New York, Doubleday & Co., Inc., 1966.

Robinson, S. J., et al.: *Congenital Heart Disease*, ed. 2, New York, Blakiston Division, McGraw-Hill Book Co., 1965.

Smith, C. H.: *Blood Diseases of Infancy and Childhood*, ed. 2, St. Louis, C. V. Mosby Co., 1966.

University of Texas M.D. Anderson Hospital: *Tumors of Bone and Soft Tissue*, Chicago, Year Book Medical Publishers, 1965.

Wilson, W. P.: *Applications of Electroencephalography in Psychiatry*, North Carolina, Duke University Press, 1965.

Wright-St. Clair, R. E.: *Doctors Monro*, London, England, Wellcome Historical Medical Library, 1964.

Book Notes

Radiologic Diagnosis in Infants and Children. Armand E. Brodeur. 503 pp. St. Louis, C. V. Mosby Co., 1965. $26.50.

IT IS SELDOM that a busy physician can truthfully say that he enjoys reading a medical textbook from cover to cover. This is such a book. Authored by the chief radiologist at the Cardinal Glennon Memorial Hospital for Children, St. Louis, it is written in a clear, refreshing style directed toward the general radiologist, the pediatrician, and the resident in these fields.

Important basic concepts are emphasized, with much attention given to differential diagnosis and pitfalls in diagnosis. Most of the lesions in the pediatric age group that would be encountered in the general hospital are described accurately and succinctly. Rare lesions are left to the standard textbooks on pediatric radiology.

The material is well organized according to anatomic regions, introduced by brief comments on pertinent normal anatomy and physiology. Innumerable superb reproductions of radiographs are included; in fact, this book approaches the completeness of an atlas of pediatric radiology applicable to the general radiology department.

The reviewer has already found this book to be an invaluable asset in his daily film-reading sessions.

—JOSEPH SELMAN, M.D., Tyler

The Burn Wound. Capt. Stanley E. Order and Col. John A. Moncrief. 72 pp., illus., Springfield, Ill., Charles C Thomas Publisher, 1965. $7.75.

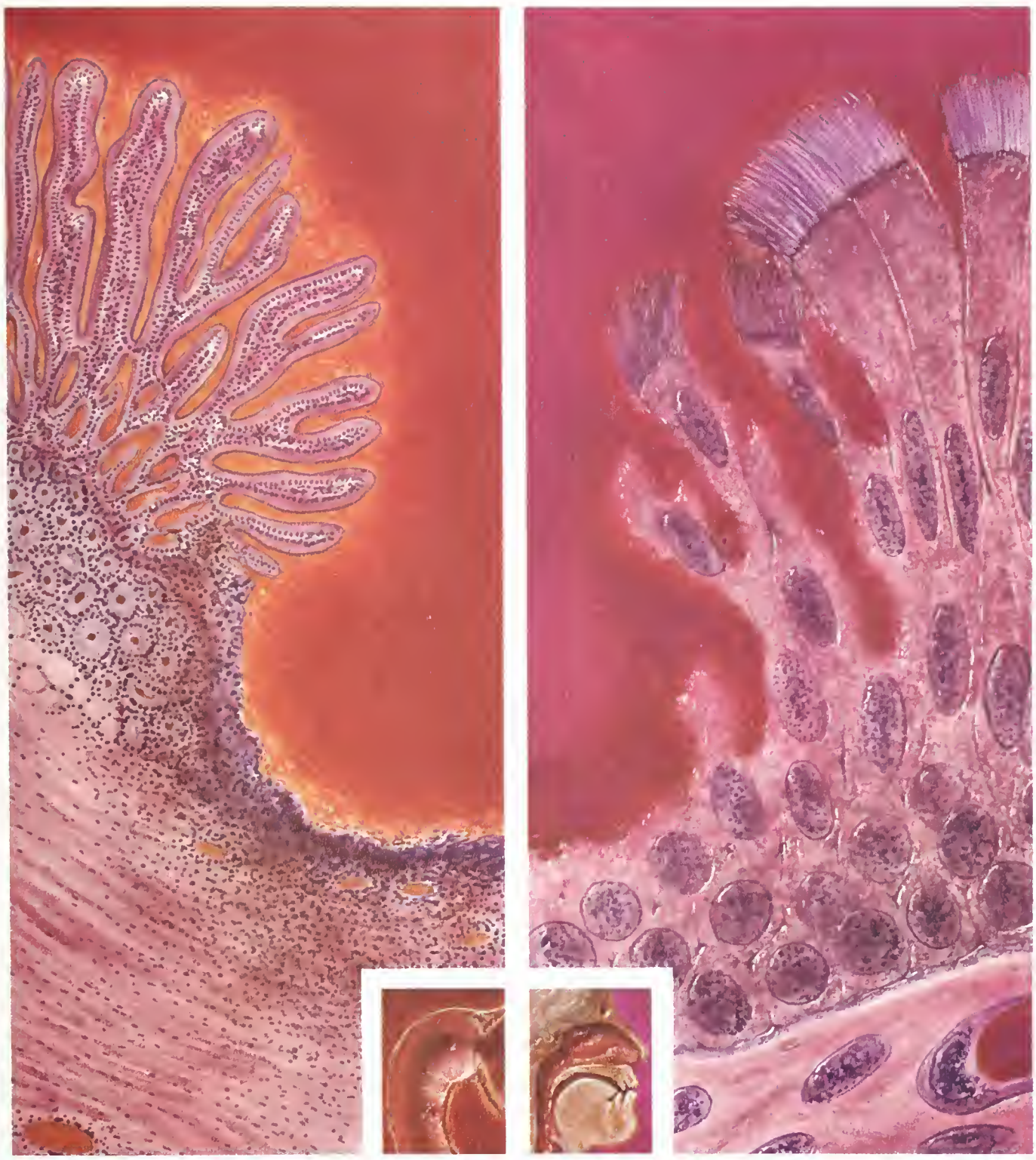
THIS ESSAY is an attractive book with 72 oversize pages, 60 references, 135 clear medical photographs, arteriographs, and micrographs which increase the understanding of the text. The excellent clinical photographs show arteriographs, homografts, autographs, and different stages of healing. Arteriographs of upper and lower extremities and of other locations demonstrate the depth of arterial avascularity and devitalization of tissue.

Clinical and experimental investigation show that the mortality rate depends on the areas involved, depth, and sepsis. Pseudomonas are the principle cause of infection

spring 1966

DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M.D., Director of Medicine.



Duodenal mucosa with ulcer crater—Approx. 80× Magnification.

Loosening epithelial cells of nasal mucosa during early stage of cold—Approx. 1800× Magnification.

this issue: partners in misery

Partners in misery: common cold and duodenal ulcer*

A century or two ago our forefathers believed that the common cold was caused by an excess of evil humors. Today we regard these baleful humors as a result of the cold rather than its cause. Only in the last ten years have some of the offending viruses been cultivated, and we now know many others can be transmitted to human volunteers although they cannot yet be grown in the laboratory. Various body defenses react to infection by a virus, and in the respiratory tract the lung is protected by the phagocytic properties of the pulmonary alveolar macrophage.[1]

Because of the brief history and large proportion of unidentified viruses, effective prophylactic vaccines have not yet appeared. A universal antiviral agent seems even more remote. Meanwhile, these viruses continue to produce epidemics of upper respiratory infection at seasonal intervals, particularly in spring, fall and winter, during which they propagate and distribute progeny indiscriminately. As a result, they are responsible for significant work loss. Since the immunity afforded is temporary, recurrent infections are common.

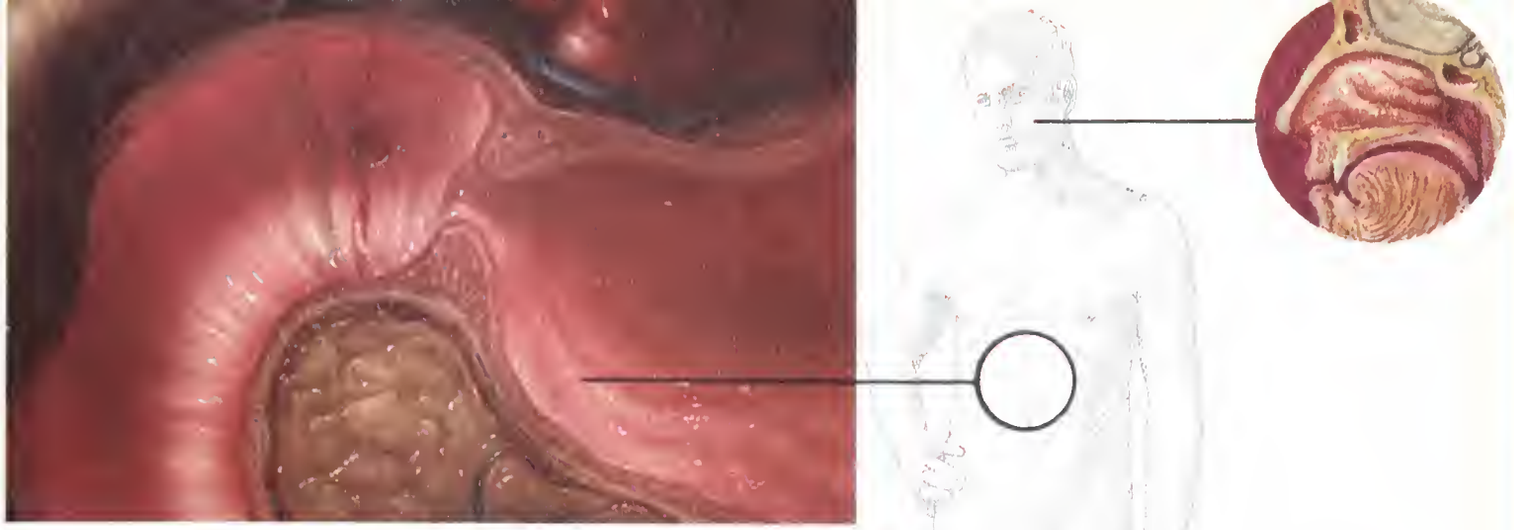
This recurrent morbidity in spring and fall is shared by duodenal ulcer, itself a cause of significant loss in wages and medical expense. Thirty years ago Emery and Monroe reported 1,279 recurrences of peptic ulcer of which 13 per cent could be attributed to upper respiratory infection, thus confirming a long-held clinical impression.[2] While the inflammation, congestion and secretion of the mucosa of the nasopharynx in the common cold, and the hyperemia and hypersecretion of the gastric mucosa during acute exacerbation of a duodenal ulcer may not be directly linked, it is probable that the miseries of the infection are reflected in the pain of the ulcer. The following case history illustrates this point.

Case report A 48 year old white male executive, suffering from hematemesis and melena for 12 hours was admitted to hospital on April 8, 1965. He gave a history of duodenal ulcer proved by x-ray studies 23 years previously. In the intervening years he suffered from typical epigastric pain occurring two hours postprandially, usually for two or three weeks each spring and fall. A hospital admission two years earlier had followed hematemesis of moderate amount.

The present admission was preceded by a recurrence of pain during a period of unusual business pressure. In addition he caught a cold one week before his entry to hospital and his already irregular eating habits were made more so by loss of taste and appetite, blocked nose and general malaise. During the final 36 hours he used aspirin to relieve his cold, taking 2 or 3 tablets with a glass of water on 6 or 7 occasions. Before retiring he drank a glass of hot toddy.

He awakened about 1 a.m. and vomited dark red blood and recently ingested food mixed with whisky. Melena followed and he was transferred to hospital. On admission he was pale, cold and sweating. Apart from epigastric tenderness and black stool on the examining finger after rectal examination, his general physical examination was not remarkable. Temperature was normal, pulse was thin and thready with a rate of 114 beats per minute, and blood

The commonly found "clover-leaf" roentgenographic abnormality of chronic duodenal ulcer due to spastic contractions and scarring.



Typical site of duodenal ulcer showing anatomical relationship to gastroduodenal artery and common bile duct.

pressure measurement was 104/60mm. of mercury. Hemoglobin estimation was 8.6 grams per cent, but white blood count, chest x-ray and urinalysis were within normal limits. No further bleeding occurred. After blood transfusions and a suitable medical regime he was dismissed two weeks later and advised to return later for consideration of surgery.

The care of the ulcer patient suffering from a viral infection of the upper respiratory tract is additionally handicapped by the local and constitutional symptoms so produced. Indifference to food often makes it difficult to "feed a cold" even in a patient without an ulcer. In the ulcer patient this becomes more than a homily, for malaise and apathy towards irksome routine produce a lack of interest in adhering to an ulcer program and result in its eventual failure if these symptoms are not relieved. The absence of curative treatment for these viral infections has spawned many a strange therapeutic offspring and some of the more generally accepted remedies contain a special risk for the ulcer patient.

To the gastric physiologist, the shot heard around the world was not fired at Concord but was discharged through the stomach of Alexis St. Martin. The worthy voyageur had a more than modest thirst and on several occasions William Beaumont observed through the resulting fistula the changes of gastritis which followed an immoderate ingestion of alcohol.[3] It is now well known that alcohol stimulates the production of gastric juice and the patient with an ulcer, even in remission, should not drink alcohol without first taking food, milk or antacid. The use of various alcoholic remedies for the common cold is to be strongly discouraged in this type of patient. Factors already present can produce an acute exacerbation of ulcer symptoms without the added stimulus of alcohol.

Headache and facial pain, sore throat and generalized aching are subjective discomforts of viral infections of the upper respiratory tract or their complications. Relief from them is usually sought in the family medicine cabinet, and of all the medications habitually stored there, salicylates are the most frequently used. Oral ingestion of salicylates has produced

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(Advertisement)

substernal pyrosis and sometimes epigastric pain in susceptible individuals. Bleeding from erosions of the gastric mucosa has followed their use and has been severe enough in some instances to cause hematemesis and melena.[4] It has been suggested that interference with the protective mucous layer of the stomach allows this to happen. There is no evidence that hypersecretion occurs, but the taking of aspirin has been followed by exacerbation of ulcer symptoms and occasionally by gastrointestinal bleeding.[5] The ulcer patient with a cold should take salicylates with an antacid, or preferably other means of symptomatic relief should be found.

Caffeine has two actions on gastric secretion. One directly stimulates production of acid and the other potentiates the out-pouring of gastric juice as a response to other stimuli.[6] In high doses to animals, it has produced erosive gastritis and peptic ulceration. Caffeine-containing beverages are discouraged for the ulcer subject and forbidden during an acute exacerbation.[7] Many cold remedies contain caffeine.

To sufferers from allergic rhinitis springtime brings symptoms of nasal obstruction, loss of taste and smell and malaise similar to those caused by viral infection but due instead to allergens which at that time of year are principally tree pollens. Relief from these symptoms can be obtained, though not wisely, by the use of steroid medication. Unfortunately, chronic sufferers from these allergies have used this approach with varying degrees of success. Steroid hormones increase gastric secretion and delay the healing of experimental ulcers.[8,9] Clinically their administration has been associated with reactivation of healed duodenal ulcers, and with bleeding and perforation which were not always preceded by typical ulcer distress. Because of these harmful effects, their use in the ulcer patient is best avoided for other than serious medical problems and then only with adequate antacid coverage.

Summing up It is immaterial by which pathways the malaise and lassitude, depression and irritability activate an ulcer, but it is the physician's responsibility to ensure that the medications he selects to relieve the symptoms of a cold do not further aggravate the ulcer.

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and fatal sepsis. The authors have proven the effectiveness of 10 percent Sulfamylon cream, which they have perfected. A review of the dynamics and management of second and third degree burns is given.

In 1964 all burn patients at the Army Research Unit were treated with 10 percent topical Sulfamylon cream, occlusive dressing, permanent and temporary skin grafts. There was a considerable retardation in the morbidity rate. The remarkable ability of this cream to combat infection has been a factor in reducing morbidity and saving areas of dermal tissue which would have been destroyed by infection.

With abundant clinical and experimental material at Brooke Army Medical Center at Fort Sam Houston, a tremendous investigative research has been carried out. The most intensive, exacting, and comprehensive studies have been made by the authors.

Since publication of the book, more than 1,915 burn patients have been treated at this unit. The use of Sulfamylon cream has greatly decreased the need for permanent skin grafts. Many patients from Vietnam and elsewhere were in critical condition, due to sepsis. Such cases had dramatic improvement.

—E. L. GRAHAM, M.D., Cisco

The Cell, an Atlas of Fine Structure. Don W. Fawcett. 448 pp., illus., W. B. Saunders Co., 1966. $11.

IT HAS BEEN said many times that exploration of, and insight into, the microcosm is as important as the development of a knowledge of our macrocosm, a concept to which I wholeheartedly subscribe. This field already has made great contributions to histologic anatomy, physiology, and pathology, both in botany and zoology (including people). Ultrascopy is one of the bridges which will more firmly unite gross and histologic anatomy, physiology, pathology, and biochemistry.

Although it is clear that the main advances in histology and histopathology will be made through ultrascopy, it still remains impractical to provide more than a few students with laboratory experience in the methodology. Yet, if a student is to read critically the literature which results from ultrascopy, he must gain experience in interpretation of electron micrographs.

The accomplished purpose of Dr. Fawcett's volume is to provide students with a laboratory manual which, though statedly not a textbook or reference work, will provide insight into ultrascopy for any biologically trained person, whether working in a laboratory or not. It is organized into three sections know respectively as "The Cell Organelles," "The Cell Inclusions," and "The Cell Surface." All the subsections are preceded by brief general discussion. Excellent reproductions of microphotographs occupy the full right page and each is discussed by a short, pertinent caption on the page to the left. This organization resulted, for me, in a pleasant, "uncluttered" learning experience.

Further, an excellent bibliography is provided and the list of credits is impressive.

For beginning students there could be more lower magnification micrographs. These lower magnifications are important for general orientation, since beginning students are not accustomed to thinking in terms of angstroms and must be led gradually into such conceptualization. Likewise, three-dimensional diagrammatic representations could help students acquire good insight more rapidly into such structures as organelles and secretory products.

Most definitely *The Cell* is not a textbook, but an atlas. It is, however, an atlas from which every physician readily can obtain sound insight into a methodology which will profoundly influence his profession.

—D. J. SIBLEY, JR., M.D., Austin

A Savage Place. Frank G. Slaughter, Garden City, N. Y., Doubleday & Co., Inc., 1964.

THIS BOOK is well written, rapidly paced, and the story dramatically entangled with bitter social and personal conflicts. It narrates the story of the conversion of a small town hospital into a voluntary one by a newly appointed, board certified staff surgeon. The board thought that when hiring the surgeon he would provide them with the accreditation they needed, but instead ended up with a new director. The transformation of the hospital and people involved makes for pleasant reading.

Statistically Speaking. W. K. Garlington and H. E. Shimota. 106 pp., Springfield, Ill., Charles C Thomas, Publishers, 1964. $5.50.

DURING THE PAST several years, a greatly increased interest in statistical methods has become apparent. This is particularly true of those involved in research. Indeed, without the use of statistical techniques, it is difficult for the research worker to comprehend the results he has obtained during an experiment. It is not surprising, therefore, to find statistics is a required course for science majors in a growing number of universities throughout the country.

In these courses, the emphasis is on teaching the future research worker how to design experiments and analyze the results so that he will obtain the most information from his data. Current textbooks in the field have been written with this in mind. Ignored is the much larger group of individuals who do not need to know the details in depth. They do feel a need, however, to understand the underlying principles, limitations, and shortcomings of the various statistical tests. This group includes practicing physicians, social workers, educators, and law enforcement officers.

Statistically Speaking has been written to fill this void. It does not have sufficient mathematical analysis to satisfy the serious research worker (although as a prelude to his more involved study of the field, it should be invaluable), but it does give enough information so that technical reports of research results can be understood.

The book is divided into four parts, dealing with the research report, the fields of descriptive and inferential statistics, and statistical fallacies and pitfalls.

All too seldom does one discover in the scientific field, a volume which is informative and authoritative and at the same time readable. The authors have successfully walked the tightrope between meaningless jargon and highly involved mathematics. They have made their primary point that statistics prove nothing, but show only the mathematical probability of one event in a relationship to another.

This book is recommended reading for all who are interested in the biomedical sciences.

—ALFORD R. HAZZARD, M.D., Austin

The Functional Pathology of Disease. Arthur Grollman, 2nd ed. New York, McGraw-Hill Book Co., 1963.

DR. GROLLMAN has brought the entire volume up to date. New sections on the nervous system, diabetes, the joints, and autoimmune diseases have been added, while retaining the general approach of the first edition. Each chapter is concluded by a good list of appropriate references. An excellent index is provided.

Principles of Public Health Administration. John J. Hanlon, 4th ed., St. Louis, C. V. Mosby Co., 1964.

DR. HANLON in this fourth edition has attempted to update his material to accommodate it to the great change that has taken place since the publication of the first edition. To quote, "about 90 percent of remedies prescribed and of the food and other items purchased by the housewife were not available when the first edition of this book was written." He has tried to make data and concepts current by stressing "public" in "public health" since he feels that they are involved in the shift from one historical era to another. In this shift the public health worker will need new "analytic teaching and learning" with a better understanding of human nature.

Fitness for the Whole Family. Paul Dudley White and Curtis Mitchell, Garden City, N. Y., Doubleday & Company, Inc., 1964.

ALTHOUGH this book is written mainly for the young families, there is no age limit in which a person can acquire a sound mind and a healthy body. Nineteen contributors tell how everyone can be physically and mentally fit. Authorities in medicine, nutrition, and physical education explain that with proper diet and adequate exercise we can help ourselves to a better way of life.

Surgical Clinicopathological Conference of the Massachusetts General Hospital. Edited by Benjamin Castlemen and John F. Burke, Boston, Little, Brown and Co., 1964.

SELECTED surgical cases discussed in this book are from records from the past 25 years. Dr. W. B. Cannon found that the discussion of real case histories could be used as a teaching exercise for medical students. Some of the clinical cases discussed in this book concern abdominal pain, cervical and mediastinal mass, chronic phlebitis, jaundice, septicemia, and shock, just to name a few. The table of contents, list of discussants, and a complete index enable the reader to find a special case quickly.

Disaster Handbook. Solomon and Evelyn Garb, New York, Springer Publishing Company, Inc., 1964.

DISASTER HANDBOOK is for all people concerned about prevention of casualties in disasters and capable of disaster management. It is essential to those who find themselves in key positions when a disaster occurs—doctors and nurses, primarily.

The book provides the information, concepts as well as practical rules, that permit the reader to cope with disaster as a "phenomenon in its own right." Detailed descriptions of a number of disasters are included to give substance, dimension, and reality to the presentation.

The major types of disaster from airplane crashes, epidemics, and fires to panic crushes and tornadoes are taken up in 22 separate chapters, each in turn presenting the specific aspects of disaster management, casualty prevention, and optimum care for the largest possible number of victims. The tasks confronting doctors, nurses, and other leaders after a nuclear explosion are defined and described in the final section.

Cellular Injury. Ciba Foundation Symposium. Edited by A. V. S. de Reuck, and Julie Knight, Boston, Little, Brown and Co., 1964.

THE CIBA Foundation is an international center where scientists are encouraged to meet informally to exchange facts and ideas, mainly at international symposiums, each lasting one to four days, and of which there are five or six each year. The proceedings of these conferences are issued with a minimum of editing in order to pass on to a wider audience the information and lively speculations in the chosen field of outstanding workers from many countries.

Correlating with another symposium published recently on lysosomes, "Cellular Injury" begins with a discussion and review of the lysosome concept in relation to cell damage. The ensuing discussion illuminates tested theories on injury by exogenous, endogenous and humoral agents, and on the mechanisms of protection by drugs, on organogenesis and necrosis, and on the fine structure of damaged cells and the lesions produced by viruses.

Recent Deaths

DR. J. DURSO

Dr. Joseph Durso, 38, Port Arthur obstetrician, died April 15, 1966, in Port Arthur. Cause of death was carcinoma of the stomach.

Dr. Durso, a native of Port Arthur, had practiced medicine in his hometown for more than 10

DR. DURSO

years. He was born Aug. 28, 1927, to Sam and Josephine Arisco Durso, and attended school in Port Arthur, where he was graduated from St. Mary's High School.

He received a bachelor of arts degree from the University of Texas and his M.D. degree from the University of Texas Medical Branch in Galveston in 1952. He interned at Robert B. Green Memorial Hospital in San Antonio and completed his residency at John Sealy Hospital in Galveston.

At the time of his death he was president-elect of the medical staff of St. Mary's Hospital and president-elect of the North Port Arthur Rotary Club. Besides holding membership in the Jefferson County Medical Society, Texas Medical Association, and American Medical Association, Dr. Durso was a diplomate of the American Board of Obstetrics and Gynecology.

Dr. Durso also belonged to the Knights of Columbus and St. Catherine Catholic Church. He was active in scouting and was vice president of the Parents Club of St. Catherine School. He also was a member of Nu Sigma Nu medical fraternity.

His wife, the former Kathleen Connelly of Austin, survives, as do a son, Samuel Christopher Durso; four daughters, Mary Josephine, Elizabeth Anne, Susan Kathleen, and Barbara Louise Durso; his parents, Mr. and Mrs. Sam Durso; a brother, Sam M. Durso; a sister, Mrs. Claude E. Jones, and his grandfather, Joe Arisco, all of Port Arthur.

DR. W. M. PHILLIPS

Dr. Warren Martin Phillips of San Antonio, 82, died on April 20, 1966, of coronary heart disease and hypertension.

A psychiatrist with the Veterans Administration since 1926, Dr. Phillips had been a resident of San Antonio for 40 years.

Born Jan. 19, 1884, in Newton, Ill., to George and Rhoda Abbott Phillips, he attended Illinois public schools and did premedical work at the Northern Indiana Normal School in Valparaiso, Ind. He received his M.D. degree in April, 1911, from the College of Physicians and Surgeons, now part of the University of Arkansas School of Medicine in Little Rock.

Dr. Phillips, who served in the US Army from 1917 to 1919, was a member of the Bexar County Medical Society, Texas Medical Association, and American Medical Association. He also was a 32nd degree Scottish Rite Mason and a member of the Christian church and the Alzafar Shrine.

Survivors are his wife, Mrs. Emma Jane Phillips of San Antonio; a daughter, Mrs. Dorothy Thomas, and a son, Dr. Claude M. Phillips, both of San Antonio; three sisters, Mrs. Pearl Wilson of Bogota, Ill., Mrs. Cloe Deckard of Robinson, Ill., and Mrs. Lucille Caldwell of Newton, Ill.; three grandchildren, and two great-grandchildren.

DR. L. O. HAYES

Dr. Lindley Onslow Hayes, 65, formerly director of the College Health Service at North Texas State University in Denton, died in Denton on March 28, 1966.

A native of Denton County, Dr. Hayes was born Feb. 19, 1901, to the late Dr. E. N. and Gertrude Hill Hayes. He attended public schools in Denton and received a bachelor of science degree from the University of Texas. He received his M.D. degree from Tulane University School of Medicine in New Orleans in 1925 and served an internship at Parkland Hospital in Dallas in 1925-1926.

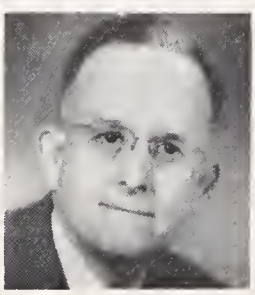
After maintaining a general practice in Denton and Midland, he joined North Texas State College as director of its health service in 1930, where he served until his retirement in June, 1965.

Dr. Hayes took a number of postgraduate courses at Tulane University School of Medicine in New Orleans, Harvard Medical School in Boston, and the University of Texas Southwestern Medical School in Dallas.

A member of the Denton County Medical Society, Texas Medical Association, and American Medical Association, Dr. Hayes was president of his county society in 1933. He also belonged to the First Baptist Church in Denton, the Masonic Lodge, and Phi Beta Pi medical fraternity.

During World War II he served as a major in the US Medical Corps.

Dr. Hayes' wife, who survives him, is the former Jo Anna Lolley of Denton.



DR. HAYES

DR. A. H. JOPLING

Dr. Anna Hammond Jopling, wife of Dr. J. L. Jopling, died April 27, 1966, in Paris, Tex., after an extended illness.

She and her husband had practiced in Paris for 20 years, being associated with the L. P. McCuistion Community Hospital where she specialized in internal medicine and diagnosis.

Daughter of Dr. Job L. and Arra Abels Hammond, Dr. Jopling was born in Paris on Nov. 9, 1905. After attending schools in Paris and what is now Texas Woman's University in Denton, she was graduated from the University of Texas and received her M.D. degree from the University of Texas Medical Branch in Galveston in 1932.

She and Dr. Julian L. Jopling were married while both were medical students. They practiced at Taylor before moving to Paris.

Dr. Jopling was a member of the Lamar-Delta Counties Medical Society, Texas Medical Association, and American Medical Association. She was an active member of the Episcopal Altar Guild and sponsor of a junior altar guild, and also belonged to the Tejas Garden Club.

Survivors include her husband; three daughters, Mrs. Grady Barrett, Jr., Hudson, Ohio, Miss Jan Jopling, Austin, and Mrs. Stephen F. Weger, who returned recently from Europe; six sisters and brothers, Mrs. Leonard Short and Dr. D. S. (Scott) Hammond, Paris, Mrs. Ed Tolbert, Sherman, Mrs. J. M. Douglas, Portland, Ore., Mrs. Jack Pridgen, Waco, and Job Hammond, Fort Worth; and three grandchildren.

DR. JOPLING

DR. J. G. YOUNG

Dr. John Garnett Young, 66, died in Dallas on May 4, 1966. Dr. Young, a past president of the Dallas County Medical Society, had practiced pediatrics in Dallas from 1927 until his retirement in 1964.

The son of F. L. and Martha Higgins Young, he was born in Greenville, Tex., on Sept. 4, 1899. He attended Bowdoin College in Brunswick, Maine, and received his M.D. degree in 1924 from Harvard Medical School, Boston. After an internship at Fifth Avenue Hospital of New York Medical College's Metropolitan Hospital Center, Dr. Young held a residency in pediatrics at Massachusetts General Hospital in Boston.

Dr. Young was former president of the Dallas Child Guidance Clinic, Dallas Southern Clinical Society, Dallas County Medical Society, Dallas Pediatric Society and the Texas Pediatric Society. He held honorary memberships in Dallas County Medical Society, Texas Medical Association, and American Medical Association. He was a clinical professor of pediatrics at the University of Texas Southwestern Medical School. He had been chief of staff at Children's Hospital.

He was a fellow of the American College of Physicians and the American Academy of Pediatrics, and was a member of the Trudeau Society and the American Diabetes Association. He was an elder in the Church of Christ and a member of Phi Chi medical fraternity, and served on the Dallas City Health Advisory Board.

Dr. Young also served as a member of the board of the Harding College in Searcy, Ark., a member of the board of the St. Mark's School of Texas in Dallas, and president of the Board of Trustees of Southwestern Christian College in Terrell, Texas.

Survivors include his wife, the former Isabel Stults; a daughter, Mrs. John H. Van Amburgh of Dallas; two sons, Dr. Robert C. Young of Dallas and Dr. David L. Young of New Haven, Conn.; four brothers, Eldon Young of Houston, Frank L. Young of Greenville, Paul C. Young of Fort Worth, and Harold H. Young of Odessa; five sisters, Mrs. A. H. Dunlap of Austin, Mrs. J. P. Maddox of Oklahoma City, Okla., Mrs. Landon Wythe of Granbury, Mrs. Guy Vanderpool of Lubbock, and Miss Beth Young of Palo Alto, Calif.; and twelve grandchildren.

DR. YOUNG

DR. A. W. HARRIS

Dr. Alfred William Harris of Dallas, 53, was accidentally drowned on April 28, 1966, when flash floods in Dallas washed his car off a road near his home.

A prominent cardiologist and clinical professor of medicine at the University of Texas Southwestern Medical School, Dr. Harris had practiced medicine in Dallas since 1940 as a specialist in cardiology.

Born May 17, 1912, in St. Louis, Mo., the son of Joseph and Rebecca Schrieber Harris, Dr. Harris received his premedical education at Washington University in St. Louis. In 1935, he received his M.D. degree from Washington University School of Medicine.

Dr. Harris served an externship at Barnes Hospital in St. Louis from 1934 to 1935, and an internship at St. Paul Hospital in Dallas from 1935 to 1936. He held residencies in internal medicine at St. Paul Hospital, Dallas, and at Peter Bent Brigham Hospital in Boston, and a fellowship at Harvard Medical School in Boston.

Dr. Harris was a member of the Dallas County Medical Society, Texas Medical Association, American Medical Association, Dallas Southern Clinical Society, American Heart Association, Southern Medical Association, and was a fellow of American College of Cardiology, and a diplomate of the American Board of Internal Medicine in the sub-specialty of cardiovascular diseases. He was also a fellow of the American College of Physicians, serving as Governor for Northern Texas.

He was a past president of the Dallas Heart Association, Dallas Internist Club, Texas Rheumatism Association, Texas Heart Association and the Southern Section of the American Federation for Clinical Research. He was a charter member of the Southwestern Society of Nuclear Medicine.

DR. HARRIS

Dr. Harris also was a past president of Washington University Alumni Association of Dallas and a member of Temple Emanu-El.

He was an assistant in medicine at Harvard Medical School and instructor in clinical medicine at Baylor University College of Medicine in Dallas before beginning his private practice. His hospital appointments included chief of medical service at Gaston Episcopal Hospital, attending physician at Parkland Hospital, and consultant physician at Baylor University Medical Center, St. Paul Hospital, Methodist Hospital, and Shannon Memorial Hospital in San Angelo. He was assistant chief of medical service at Baylor Medical Center from 1950 to 1964, and at the time of his death was a consultant in cardiology at Veterans Administration Hospital and Children's Medical Center in Dallas.

Dr. Harris received three silver medallions for his service from the Texas Heart Association and one from the Dallas Heart Association.

Survivors, all of Dallas, include his wife, the former Claire Marie Schlenter; a daughter, Miss Kathryn Ruth Harris; a son, Alfred W. Harris, Jr.; his mother, Mrs. Joseph Harris; a sister, Miss Shirley Jean Harris; and a brother, Harry Harris.

DR. L. W. JACKSON

Dr. Luke Walford Jackson, 55, a prominent San Antonio physician for more than a quarter of a century, died in San Antonio April 26, 1966, of a cerebral hemorrhage, thrombocytopenia, and acute myelogenous leukemia.

A native of San Antonio, he was the son of Dr. L. B. and Lizzie Rose Walford Jackson.

A graduate of Breckenridge High School in San Antonio, he

DR. JACKSON

DR. MARKEWICH

DR. TREADAWAY

received a bachelor of arts degree from the University of Texas in Austin and his M.D. degree in 1934 from the University of Pennsylvania School of Medicine in Philadelphia. He held internships at Wilkes-Barre General Hospital, Wilkes-Barre, Pa., and Fifth Avenue Hospital in New York, and a residency at the Jersey City Medical Center, Jersey City, N.J., before beginning his practice in San Antonio in 1937.

Dr. Jackson was a member of the Bexar County Medical Society, Texas Medical Association, American Medical Association, the Texas and American Academies of General Practice, and the Christian Medical Society. He also belonged to the Rebel Printmakers Club and was a former president of the San Antonio Photographic Society. He was a deacon of the First Baptist Church in San Antonio and a Scottish Rite Mason.

He served as a lieutenant colonel with the US Army Medical Corps from 1953 to 1956.

Survivors include his wife, the former Margaret Jacqueline Adams; two sons, Philip W. Jackson of Kingsville and Bennett E. Jackson of San Antonio; and two sisters, Mrs. R. G. Bagwell of Kilgore and Miss Helena Jackson of Canyon Lake.

DR. J. MARKEWICH

Dr. Jake Markewich, 53, a Houston internist, died April 6, 1966, in Houston.

Born Aug. 22, 1912, in Poland, he was the son of Joseph and Gertrude Sherman Markewich.

After attending Lamar Junior College in Beaumont and the University of Texas in Austin, Dr. Markewich received his M.D. degree from the University of Texas Medical Branch in Galveston in 1938. He held an internship at Jewish Hospital of St. Louis, Mo., and a residency at the Hotel Dieu Hospital in Beaumont before opening a general practice in Beaumont in 1940.

Following service as a captain in the US Army Medical Corps from 1941 to 1946, Dr. Markewich opened a practice in Houston in internal medicine, where he practiced until his death.

He was a member of the Harris County Medical Society, Texas Medical Association, American Medical Association, and the Houston Academy of Internal Medicine. He also belonged to Congregation Beth Yesurun, AF&AM, Scottish Rite, and the Doctors Club of Houston.

His wife, the former Florence T. Herskovitz, survives, as do two daughters, Marilyn Sue and Maurine, both of Houston; a son, Gary Steven Markewich, who attends the University of Texas Medical Branch; three sisters, Mrs. Isaac Neuman of Bay City, Mrs. Louis Weintraub of Houston, and Mrs. Haskell Farb of Galveston; and a brother, Louis Markewich of Bay City.

DR. T. L. TREADAWAY

Dr. Thomas Lester Treadaway, 61, a Brownfield resident since 1913, was killed in a head-on collision near Barstow, Tex., April 2, 1966.

Dr. Treadaway, a retired physician and surgeon, was born July 30, 1904, at Miles, Tex., to the late Dr. Thomas Lafayette and Elsie Neese Treadaway. He attended Brownfield public schools and received his premedical education at Simmons College in Abilene, now Hardin-Simmons University, where he participated in intercollegiate football, basketball, and baseball.

He was graduated with honors from the University of Texas Medical Branch in Galveston in 1927, and served internships at John Sealy Hospital in Galveston and Robert B. Green Memorial Hospital in San Antonio. During his 30 years of active practice, he took additional training at Cook County Graduate School of Medicine, Chicago, and attended several postgraduate training courses.

Dr. Treadaway entered practice with his father in Brownfield in 1928, later moving to Lamesa for three years. He returned to Brownfield in 1932 to open the first hospital in his hometown. He was joined by his partner, Dr. A. H. Daniell, in 1934, and continued in the operation of the Treadaway-Daniell Hospital, now the Brownfield General Hospital, until his retirement from active practice in 1958.

Dr. Treadaway assisted in the formation, as a charter member, of the Dawson-Lynn-Terry-Gaines-Yoakum Counties Medical Society, which was organized in 1929. He served continuously for six years as secretary of the society, later becoming its president.

He was a member of the Texas Medical Association, American Medical Association, Nu Sigma Nu medical fraternity, and Alpha Omega Alpha honorary medical fraternity. He was an active member of the First Methodist Church in Brownfield, the Brownfield Chamber of Commerce, and was a past member of the Rotary Club.

He served for many years as joint county health officer and in 1958 received an award for 20 years' service as medical advisor to the local Selective Service Board.

When Dr. Treadaway retired from active practice in 1958, he became engaged in stock-farming, with particular interest in livestock breeding.

His survivors include his wife, Doris Marchbanks Treadaway, whom he married in 1947; a daughter, Doris Jane Treadaway, a student at St. Stephens' Episcopal School, Austin; two sons, Roland Turner Treadaway, Grove, and Thalmann Lester (Roscoe) Treadaway, Seminole; and five grandchildren. He is also survived by two sisters, Mrs. Mozelle Fitzgerald, Brownfield, and Mrs. Mattie Lee Long, Dallas; and one brother, S. J. Treadaway, Austin.

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COMING MEETINGS

American Medical Association, Annual Meeting, Chicago, June 26-30, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Austin, Public Relations Conference, Oct. 1, 1966; Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

JUNE

American Proctologic Society, Cleveland, Ohio, June 20-23, 1966. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

JULY

American Medical Women's Association, Rochester, N. Y., July 7-9, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

Postgraduate Medical Assembly of South Texas, Houston, July 18-20, 1966. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966, Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 8-10, 1966. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill., Sec.

American Association for Thoracic Surgery. Dr. H. T. Bahnson, Presbyterian University Hospital, Pittsburgh, Pa., Sec.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. G. Whittlesey, 219 E. 42nd St., New York, Sec.

American College of Allergists. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Gastroenterology, Philadelphia, Oct. 23-26, 1966. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, Dr. Robert A. Peterman, 3 Craig Court, Totowa Borough, N. J., Sec.

American College of Obstetricians and Gynecologists, Houston, Oct. 24-26, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, February, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American College of Surgeons, Annual meeting, San Francisco, Oct. 10-14, 1966. Dr. Samuel P. Harbison, 230 Lothrop St., Pittsburgh, Sec.

American Congress of Physical Medicine and Rehabilitation, San Francisco, Aug. 28-Sept. 2, 1966. Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., Sec.

American Gynecological Society. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, New York City, Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Hospital Association, Chicago, Aug. 29-Sept. 1, 1966. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Neurological Association. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Philadelphia, Oct. 1-5, 1966. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Washington, D.C., Sept. 16-22, 1966. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis, Ind., Sec.

American Thoracic Society. F. W. Webster, 1790 Broadway, New York 19, Exec. Sec.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Chicago, Nov. 4, 1966. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104 Sec.

Gastrointestinal Research Group, Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Private Clinics and Hospitals, Dallas, Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La., Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Mrs. R. F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston, Pres.

Southwestern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland Ave., Birmingham, Ala., Exec. Dir.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec., 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore, Md.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of General Practice, Houston, Sept. 12-14, 1966. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 11, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Division, American Cancer Society. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Heart Association, Houston, Sept. 16-18, 1966. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Fort Worth, Sept. 29-Oct. 1, 1966. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk. Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Surgical Society, Houston. Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Spring, 1966. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

Blackford Memorial Lectures, Denison, Sept., 1966, Dr. Fred W. Shelton, 600 N. Highland Ave., Sherman, Chm.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Southwest Regional Cancer Conference, Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

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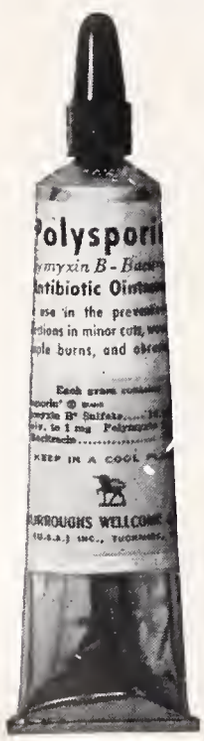

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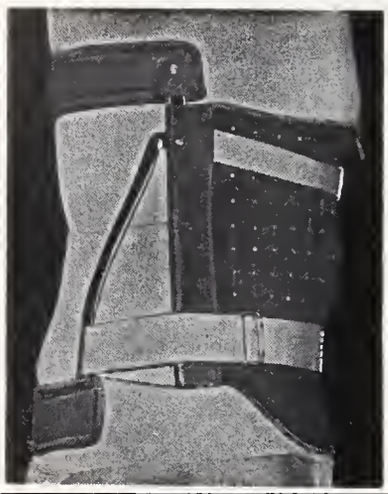

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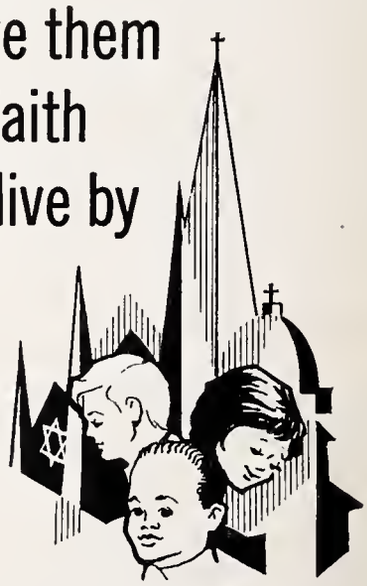

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(1) Portnoy, J.; Brewer, J. and Harris, A.: PUBLIC HEALTH REPORTS. *77:*645-652, August 1962. (2) Joseph, J. M. and Warner, G. S.: A WORKSHOP MANUAL. Md. State Dept. Health, Bureau of Lab., Balto., Md., September 1962. (3) Wollenweber, H. L.: OFF. PATH., *2*, February 5, 1963. (4) Portnoy, J.: MILIT. MED., *128:*414-417, May 1963. (5) Portnoy J.: THE AMER. JOUR. OF CLIN. PATH., *40:*473-479, November 1963. (6) Buck, A. A. and Mayer, H.: THE AMER. JOUR. OF HYG., *80:*85-90, July 1964. (7) Brown, W. J.; Donohue, J. F. and Price, E. V.: PUBLIC HEALTH REPORTS, *79:*496-500, June 1964. (8) Clayton, J. L.; Lindhardt, E. M. and Fraser, R. S.: PUBLIC HEALTH LAB., *22:*206-207, November 1964. (9) Lucatorto, F. M.; Katz, B. D. and Toto, P. D.: THE J.A.D.A., *69:*697-699, December 1964. (10) Portnoy, J.: PUBLIC HEALTH LAB., *23:*43, March 1965. (11) Reed, E. L.: PUBLIC HEALTH LAB., *23:*96-103, May 1965.

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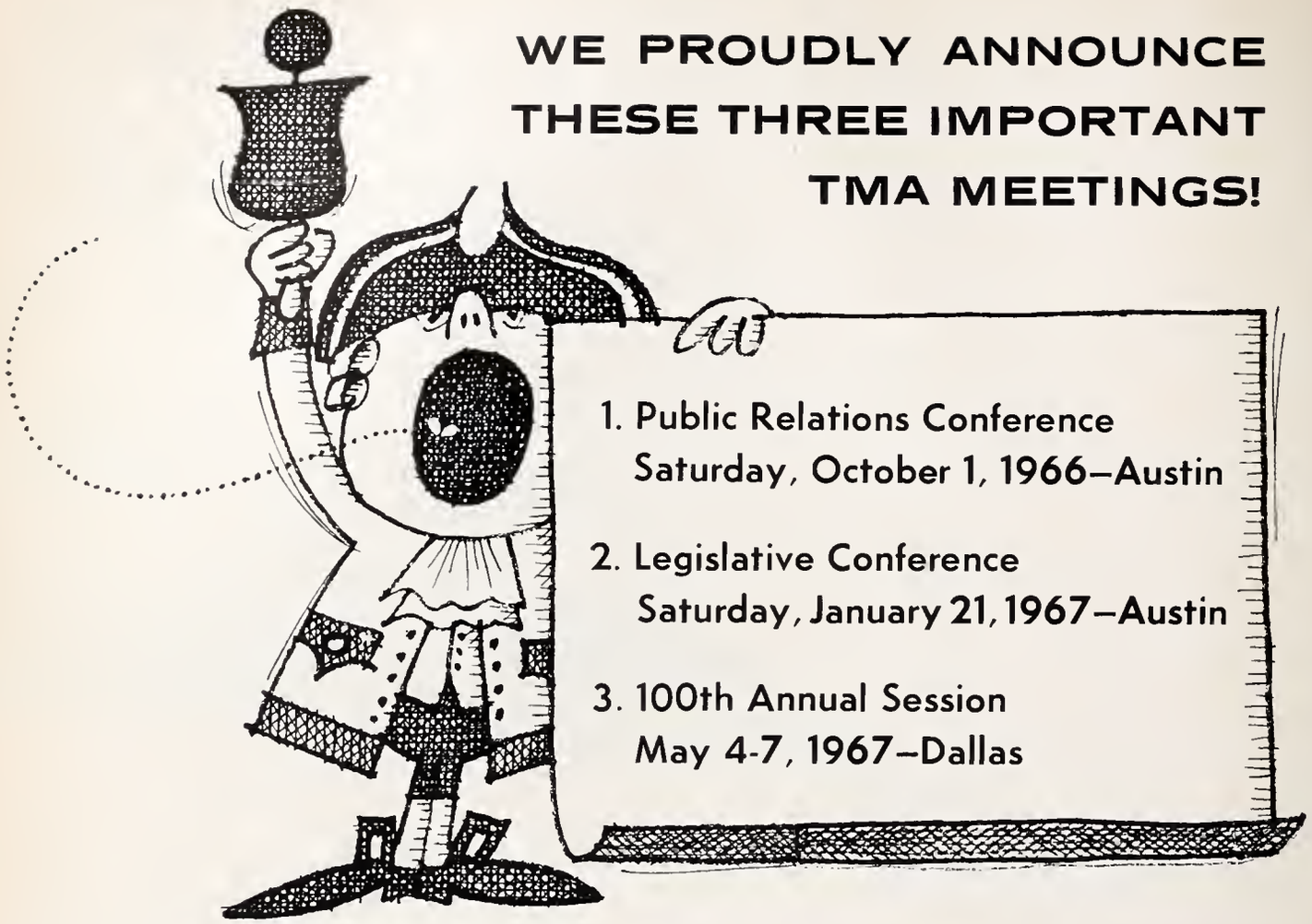

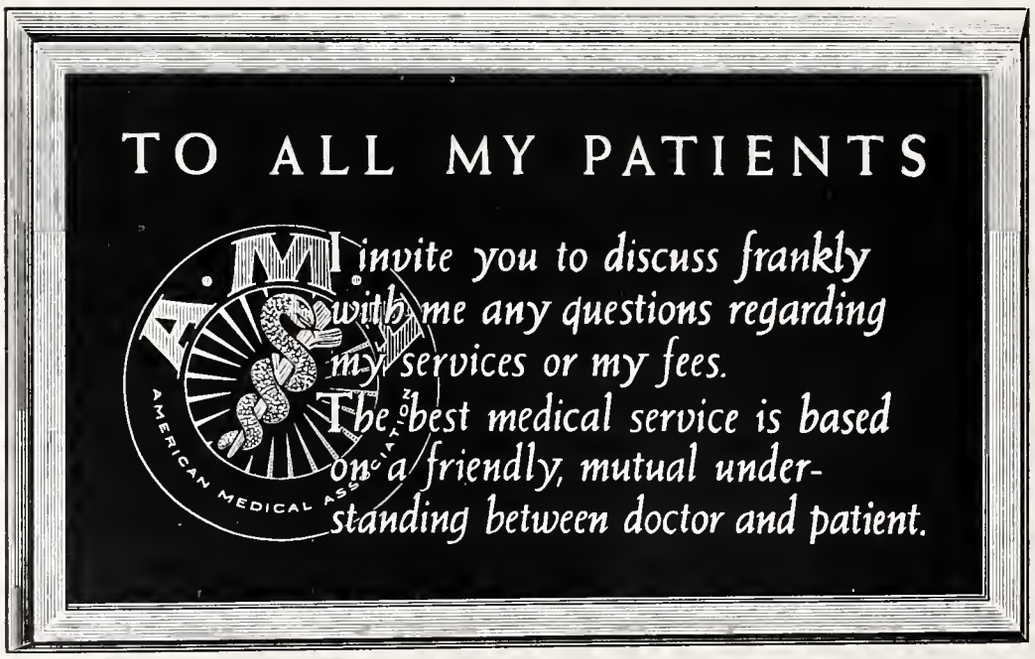
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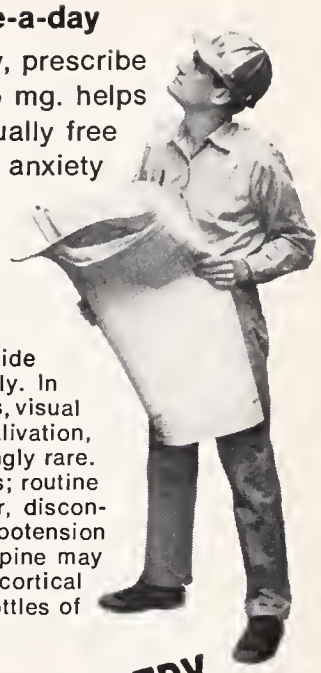

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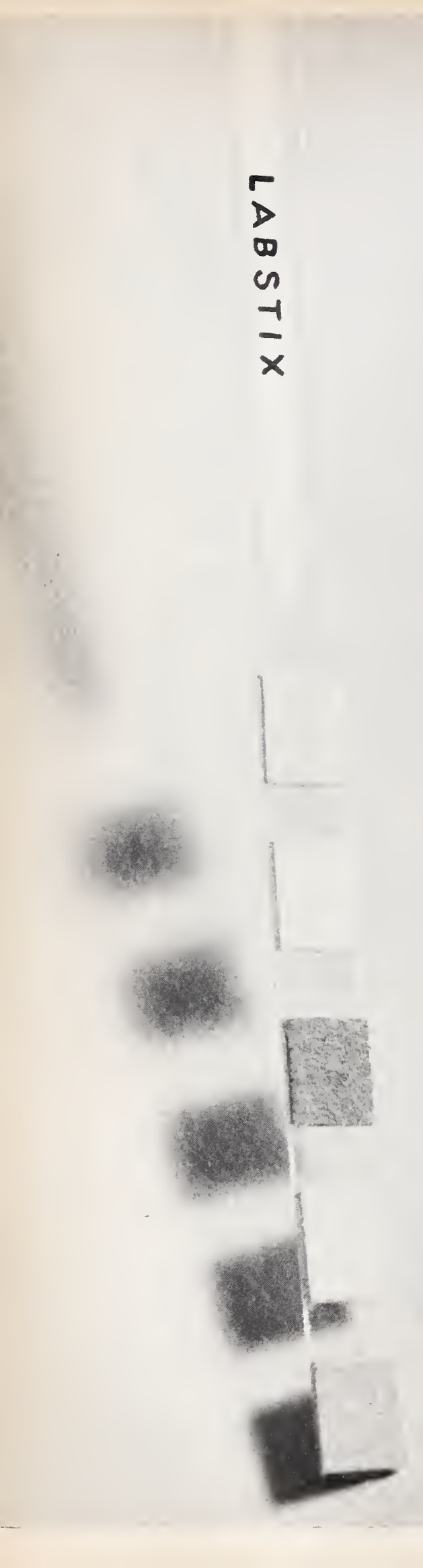

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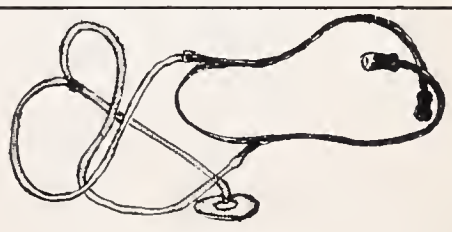

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MODERN MEDICAL-DENTAL CENTER located only three blocks from Northwest Memorial Hospital being constructed has space available for ENT, Eye, Dermatology, Psychiatry. Houston's best practice opportunity and location. Personal interview required of all interested. Write Box 804, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED: GENERAL PRACTITIONERS for generalist type work in partnership with three other physicians, Texas Hill Country, salary one year and partnership after one year. Town of 5,000 with trade area of 30,000. Reply to Box 957, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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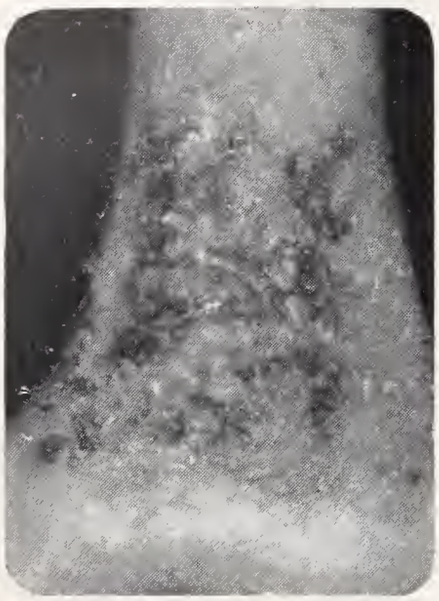
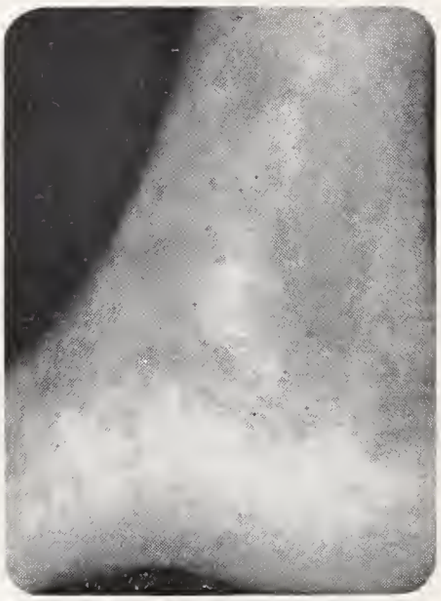


R. R., Age 11—Before treatment—atopic eczema of long standing



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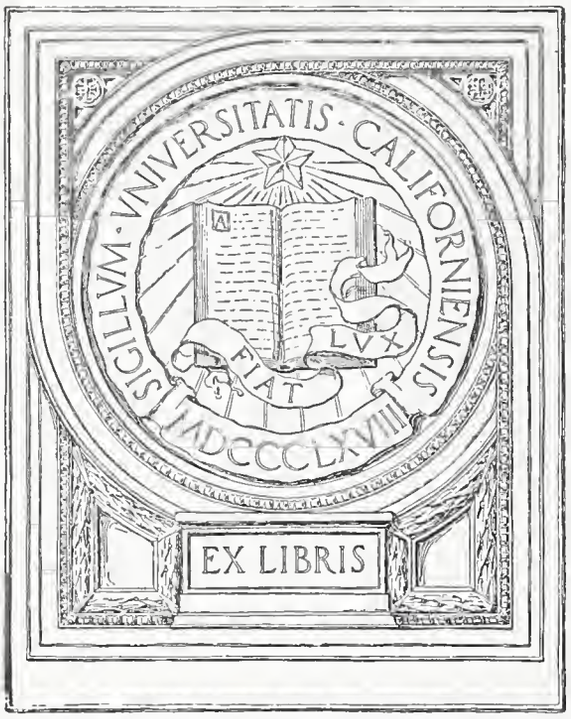

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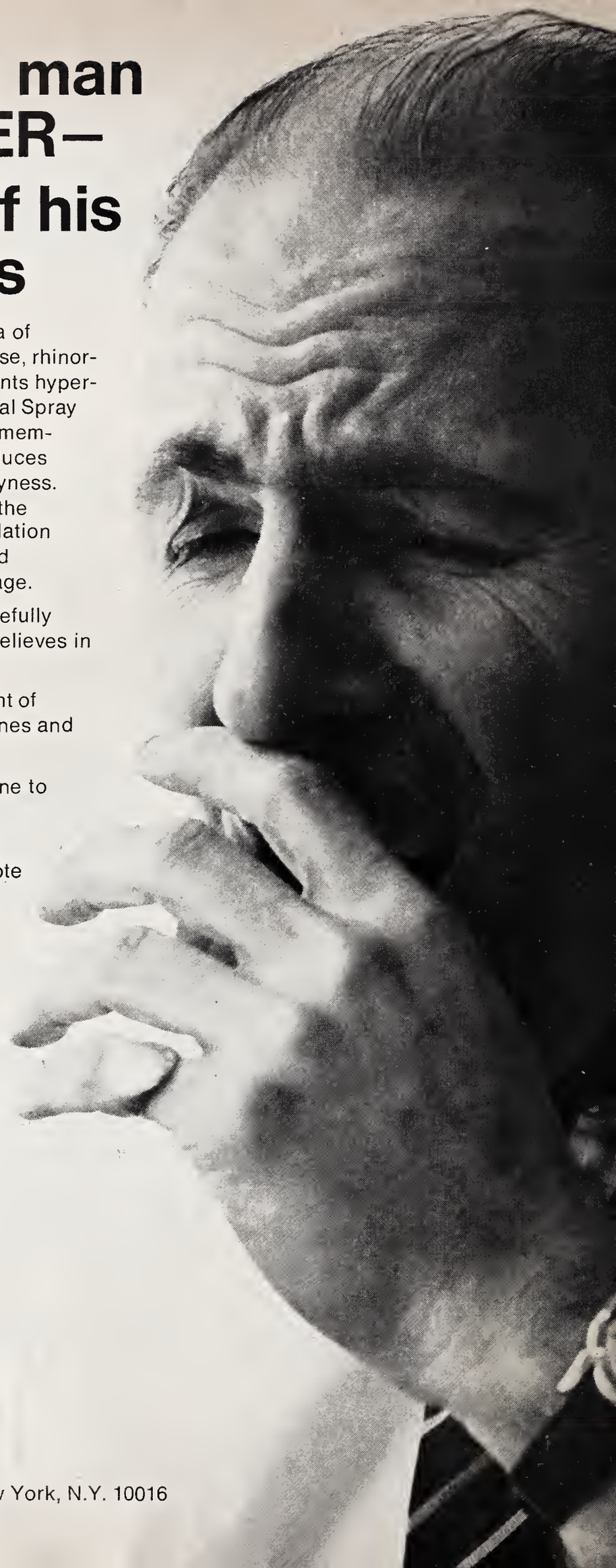

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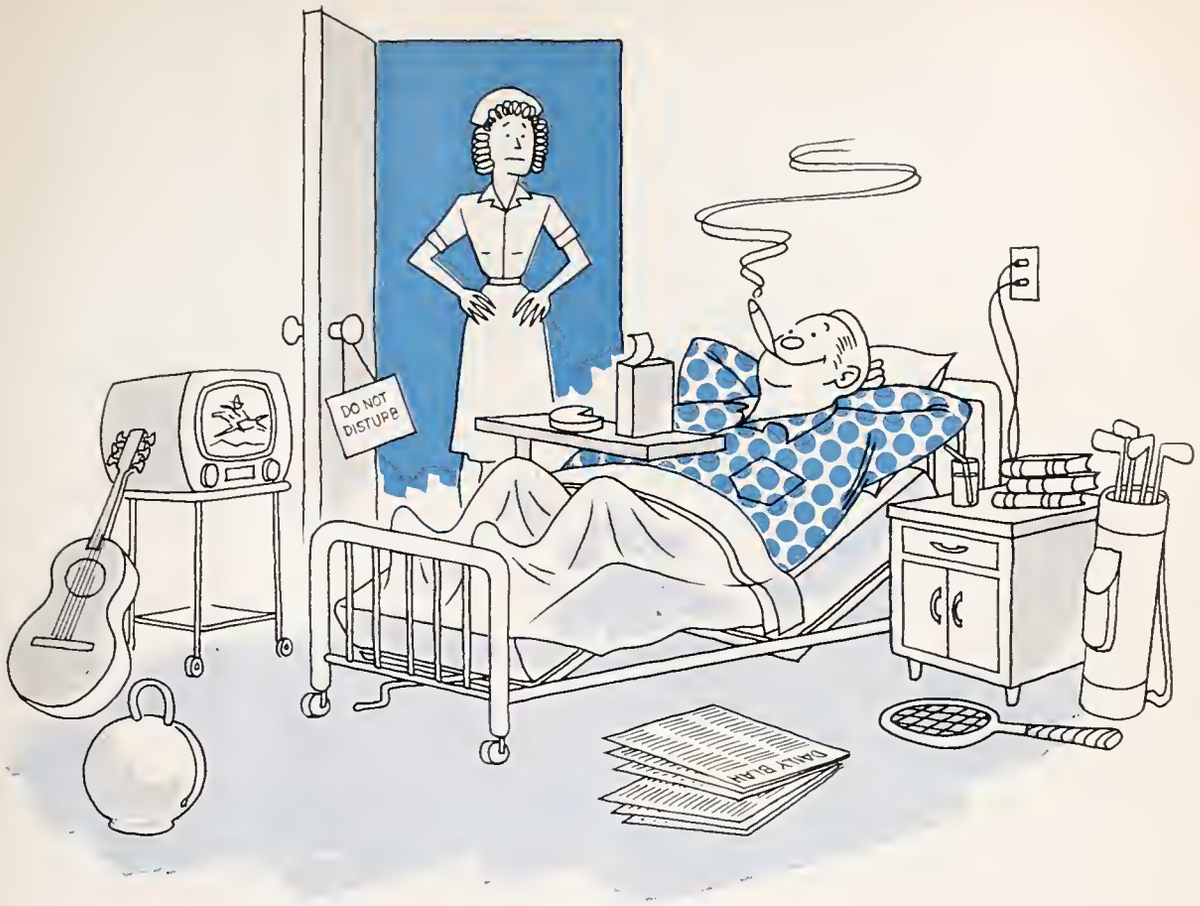

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Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

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*"*Sexual impotence treatment with methyl testosterone - thyroid (ANDROID) a double blind study" - Montesano, Evangelista: Clinical Medicine, April 1966.*

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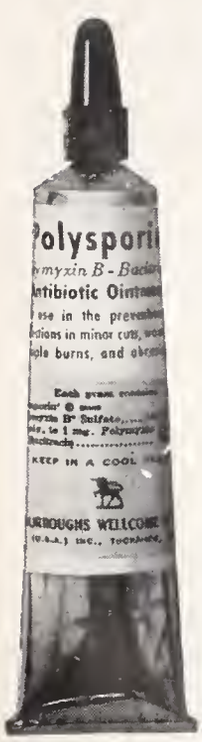

USE 'POLYSPORIN'® brand POLYMYXIN B-BACITRACIN ANTIBIOTIC OINTMENT

broad-spectrum antibiotic therapy with minimum risk of sensitization

Caution: As with other antibiotic products, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs. **Contraindication:** This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.
Supplied: In ½ oz. and 1 oz. tubes
Complete literature available on request from Professional Services Dept. PML.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.

This space
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for
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to the
TUBEX
system

Only TUBEX offers so complete a line of closed-system injectables, and it's still growing

—no other system is safer

—no other system is more efficient

—no other system offers more convenience

—no other system assures greater patient comfort

TUBEX sterile cartridge-needle units are prefilled with measured doses. Each unit is used once, and discarded. This eliminates danger of cross contamination, as well as problems of sterilization, storage, and measurement. Ultra-sharp, siliconized needles keep patient discomfort to a minimum. No other injectable system is easier to use, easier to store, easier to carry.

JUST ARRIVED!

4 New TUBEX® Sterile
Cartridge-Needle Units
Vitamin B Complex with B_{12}
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Hydroxocobalamin, 100 mcg./cc.
Hydroxocobalamin, 1000 mcg./cc.

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Wyeth® SERVICE TO MEDICINE

Closed Injection System
Sterile Cartridge-Needle Unit
Hypodermic Syringe
Wyeth Laboratories • Philadelphia, Pa.

Blood-glucose screening for all your patients?

...because "Abnormalities of glucose metabolism are among the [most common] encountered in clinical practice...."* Simple, quick, economical blood-glucose screening with DEXTROSTIX® Reagent Strips is practicable in every regular physical examination, emergency situation, and whenever hypo- or hyperglycemia may be of clinical significance—for "The precision and accuracy of DEXTROSTIX ...meet the need for an always available simple screening method...."* All that is required for screening with DEXTROSTIX is 60 seconds and a globular drop of capillary or venous blood. Abnormal readings will be a valuable aid to diagnosis; normals will help you establish an important baseline for future reference.

Marks, V., and Dawson, A.: Brit. M. J. *1*:293, 1965.

DEXTROSTIX—
provides a clinically useful determination when performed according to directions†

DEXTROSTIX is not intended to replace the more precise analytical laboratory methods.

Yes—all your patients

AMES COMPANY, INC.
Elkhart, Indiana

AMES

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Night Leg Cramps...
Frequent Bedfellow in Diabetes, Arthritis, and Peripheral Vascular Disorders*

*"Nocturnal cramps occurring in the calf muscles and small muscles of the feet have been encountered in a significant number of diabetic patients."[1]

"... nocturnal cramps may be the presenting symptoms of patients with arteriosclerosis obliterans, deep thrombophlebitis, varicose veins, osteoarthritis..."[2]

now...specific therapy for night leg cramps

QUINAMM™

Consistently effective, QUINAMM provided complete relief in 94% of 200 patients studied, many of whom were severe cases refractory to other medication.[3] Your prescription for one tablet at bedtime often controls painful night cramps with the initial dose... helps restore restful sleep.

QUINAMM Composition: Each white, beveled, compressed tablet contains: Quinine Sulfate 4 grains (250 mg.), Aminophylline 3 grains (200 mg.). **Precautions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastraintestinal disturbance. Discantinue use if ringing in the ears, deafness, skin rash, or visual disturbances occur. **Contraindication:** QUINAMM is contraindicated in pregnancy because of its quinine content. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets. **Caution:** Federal law prohibits dispensing without prescription. **References:** 1. Shuman, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuck, E., et al.: Angiology, 12:102, 1961. 3. Rawls, W. B., et al.: Med. Times, 87:818, 1959.

WALKER *Laboratories* Division of Richardson-Merrell Inc. Mt. Vernon, New York 10551

when anxiety is part of the clinical picture

Librium®
(chlordiazepoxide HCl)

In prescribing: Dosage—Adults: Mild to moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d.
Side Effects: Side effects, usually dose-related, include drowsiness, ataxia, minor skin rashes, edema, menstrual irregularities, nausea and constipation. When treatment is protracted, blood counts and liver function tests are advisable. Paradoxical reactions may occasionally occur in psychiatric patients. Individual maintenance dosages should be determined.
Precautions: Advise patients against possibly hazardous procedures until maintenance dosage is established. Though compatible with most drugs, use care in combining with other psychotropics, particularly MAO inhibitors or phenothiazines; warn patients of possible combined effects with alcohol. Observe usual precautions in impaired renal or hepatic function, in long-term treatment and in presence of depression or suicidal tendencies. Exercise caution in administering drug to addiction-prone patients or those who might increase dosage; withdrawal symptoms, similar to those seen with barbiturates or meprobamate, can occur upon abrupt cessation after prolonged overdosage. Caution should be exercised in prescribing any therapeutic agent for pregnant patients.
Supplied: Capsules, 5 mg, 10 mg and 25 mg, bottles of 50.

Roche Laboratories • Division of Hoffmann - La Roche Inc • Nutley, N.J. 07110

"All Interns are Alike"

It stands to reason. They all go through the same training; they all have to pass the same tests; they all have to measure up to the same standards; they all are underpaid, too. Therefore, all interns are alike.

That's utter nonsense, of course. But it's no more nonsensical than what some people say about aspirin. Namely: since all aspirin is at least supposed to come up to certain required standards, then all aspirin tablets must be alike.

Bayer's standards are far more demanding. In fact, there are at least *nine specific differences* involving purity, potency and speed of tablet disintegration. These Bayer® standards result in significant product benefits including gentleness to the stomach, and product stability that enables Bayer tablets to *stay* strong and gentle until they are taken.

So next time you hear someone say that all aspirin tablets are alike, you can say, with confidence, that it just isn't so.

You might also say that all interns aren't alike, either.

when readings indicate hypertension

Time for Naturetin®

SQUIBB BENDROFLUMETHIAZIDE

to reduce blood pressure

In the management of your hypertensive patients, Naturetin is good therapy to start with, good therapy to stay with.

In mild hypertension, Naturetin lowers blood pressure gradually toward normotensive levels. In long-term therapy, Naturetin may keep blood pressure low—for months, sometimes years. When used in combination with other antihypertensive agents, blood pressure often falls further—and lower doses of both drugs are usually possible.

Clinical trials have proven Naturetin effective—without serious side effects.[1,2] And, when used to treat patients with cardiac edema and hypertension, "in no instance did the concentration of serum potassium fall below 3.1 mEq. per liter."[3] (Normal range for serum potassium: 3.5-5.0 mEq./liter).[4]

When readings indicate hypertension, start with Naturetin, stay with Naturetin.

Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available—Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: 1. Telfeyan, S. A.: Clin. Med. *70*:1668, 1963. 2. Shepard, H. L.: J. Am. Geriatrics Soc. *11*:363, 1963. 3. Cummings, D. E.; Goodman, R. M., and Steigmann, F.: J. Am. Geriatrics Soc. *12*:161, 1964. 4. Castleman, B., ed.: New England J. Med. *268*:1462, 1963.

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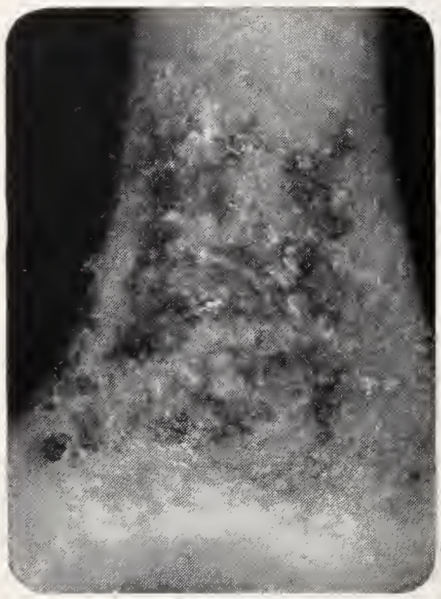
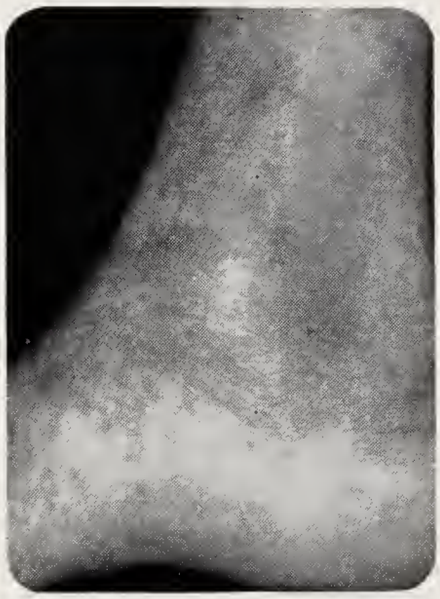
Squibb Quality
—the Priceless Ingredient

eczema: scourge of childhood



R. R., Age 11—Before treatment—atopic eczema of long standing



After treatment—with ARISTOCORT Topical Ointment 0.1% for two weeks

ARISTOCORT® Triamcinolone Acetonide Topicals have proved exceptionally effective in the control of various forms of childhood eczema: allergic, atopic, nummular, psoriatic, and mycotic.

In most cases responsive to topical ARISTOCORT, the 0.1% concentration is sufficiently potent. The 0.5% concentration provides enhanced topical activity for patients requiring additional potency for proper relief.

Administration and Dosage: Apply sparingly to the affected area 3 or 4 times daily. Some cases of psoriasis may be more effectively treated if the 0.1% Cream or Ointment is applied under an occlusive dressing.

Contraindications: Tuberculosis of the skin, herpes simplex, chicken pox and vaccinia.

Precautions and Side Effects: Do not use in the eyes or in the ear (if drum is perforated). A few individuals react unfavorably under certain conditions. If side effects are encountered, the drug should be discontinued and appropriate measures taken. Use on infected areas should be attended with caution and observation, bearing in mind the potential spreading of infection and the advisability of discontinuing therapy and/or initiating antibacterial measures. Generalized dermatological conditions may require systemic corticosteroid therapy. Steroid therapy, although responsible for remissions of dermatoses, especially of allergic origin cannot be expected to prevent recurrence. The use over extensive body areas, with or without occlusive nonpermeable dressings, may result in systemic absorption. Appropriate precautions should be taken. When occlusive nonpermeable dressings are used, miliaria, folliculitis and pyodermas will sometimes develop. Localized atrophy and striae have been reported with the use of steroids by the occlusive technique. When occlusive nonpermeable dressings are used, the physician should be aware of the hazards of suffocation and flammability. The safety of use on pregnant patients has not been firmly established. Thus, do not use in large amounts or for long periods of time on pregnant patients.

Packages: Tubes of 5 Gm. and 15 Gm.; ½ lb. jar.

PHOTOGRAPHS COURTESY OF M. M. NIERMAN, M.D.

Aristocort® Topical Ointment 0.1% and Cream 0.1%, 0.5%
Triamcinolone Acetonide

Also available in foam form and with neomycin.

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618-6

the sedentary life is often the seat of low back pain

The human spine is not engineered for prolonged sitting at desks, pianos, typewriters and drafting boards. The stresses set up by the heavy, forward-tilted head and trunk, balanced precariously on an insufficient base, result in strain of the dorsal musculature, particularly at the low lumbar level.

The unusual muscle-relaxant and analgesic properties of 'Soma' make it especially useful in the treatment of low back sprains and strains. 'Soma' is widely prescribed ☐ **to relieve pain** ☐ **to relax muscles** ☐ **to restore mobility.**

Indications: 'Soma' is useful for management of muscle spasm, pain, and stiffness in a variety of inflammatory, traumatic, and degenerative musculoskeletal conditions. It also may act to normalize motor activity in certain neurologic disturbances.

Contraindications: Allergic or idiosyncratic reactions to carisoprodol.

Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

One instance each of pancytopenia and leukopenia, occurring when carisoprodol was administered with other drugs, has been reported, as has an instance of fixed drug eruption with carisoprodol and subsequent cross reaction to meprobamate. Rare allergic reactions, usually mild, have included one case each of anaphylactoid reaction with mild shock and angioneurotic edema with respiratory difficulty, both reversed with appropriate therapy. In cases of allergic or hypersensitivity reactions, carisoprodol should be discontinued and appropriate therapy initiated. Suicidal attempts may produce coma and/or mild shock and respiratory depression.

Dosage: Usual adult dose is one 350 mg. tablet three times daily and at bedtime.

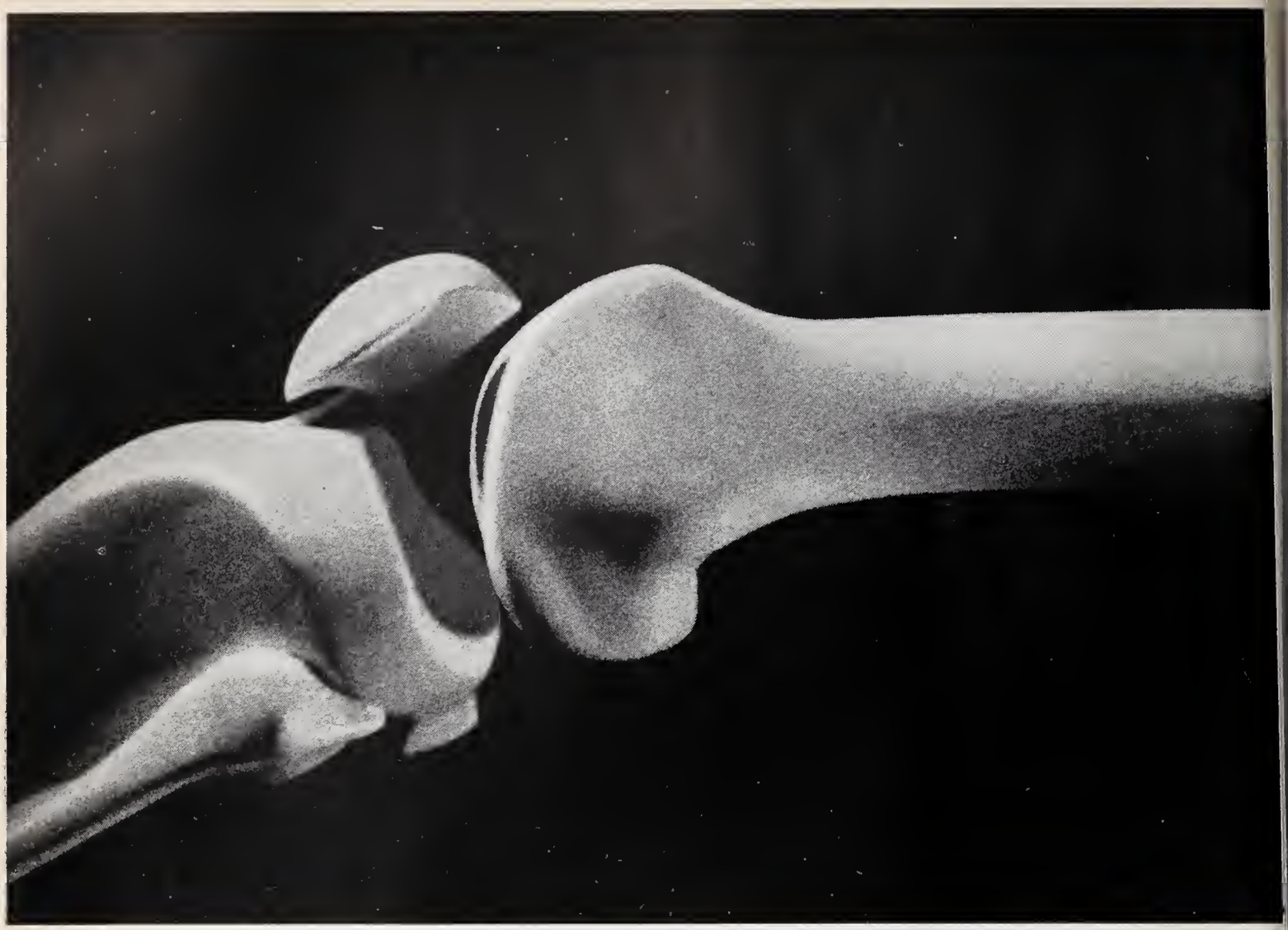
Supplied: Two Strengths: 350 mg. white tablets and 250 mg. orange, two-piece capsules.

Before prescribing, consult package circular.

for the relief of low back sprains and strains

SOMA®
(CARISOPRODOL)

Wallace Laboratories, Cranbury, N.J.
26501J



Butazolidin® alka

phenylbutazone	100 mg.
dried aluminum hydroxide gel	100 mg.
magnesium trisilicate	150 mg.
homatropine methylbromide	1.25 mg.

Usually works within 3 to 4 days in osteoarthritis

The trial period need not exceed 1 week. In contrast, the recommended trial period for indomethacin is *at least 1 month*.

That's why it's logical to start therapy with Butazolidin alka—you'll know quickly whether or not it works. And usually, it will.

A large number of investigators have reported major improvement in about 75% of cases. Some patients have gone into remission. Relief of stiffness and pain may be followed quickly by improved function and resolution of other signs of inflammation. And Butazolidin alka is well tolerated, especially since it contains antacids and an antispasmodic to minimize gastric upset.

Contraindications
Edema, danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile, or when other potent drugs are given concurrently. Large doses are contraindicated in patients with glaucoma.

Precautions
Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should be closely supervised and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Make regular blood counts. Use greater care in the elderly.

Warning
If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy.

Adverse Reactions
The most common are nausea, edema and drug rash. Hemodilution may cause moderate fall in red cell count. The drug may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, generalized allergic reaction, stomatitis, salivary gland enlargement, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hepatitis, jaundice, and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently.

Dosage
The initial daily dosage in adults is 300-600 mg. daily in divided doses. In most instances, 400 mg. daily is sufficient. When improvement occurs, dosage should be decreased to the minimum effective level: this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

Also available: Butazolidin®,
brand of phenylbutazone
Tablets of 100 mg.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York

BU-3804 P

Geigy

makes sleep irresistible

nidar®

EACH TABLET CONTAINS:

Pentobarbital Sodium	25 mg.
Secobarbital Sodium	25 mg.
Butabarbital Sodium	7.5 mg.
Phenobarbital	7.5 mg.

(WARNING: MAY BE HABIT FORMING)

ARMOUR PHARMACEUTICAL COMPANY
Chicago, Illinois, U.S.A.

nidar®

Sleep comes easy...lingers...departs naturally

Gentle doses of 4 barbiturates assure uninterrupted sleep

2 barbiturates act fast...in 20 to 30 minutes

2 long-range barbiturates come into play to sustain sleep for up to 8 hours

Tiny amounts of individual barbiturates means Nidar is well tolerated

Patients enjoy a refreshing, clear-headed wake-up

makes sleep irresistible

IN BRIEF:

EACH TABLET CONTAINS:

Pentobarbital Sodium	25 mg.
Secobarbital Sodium	25 mg.
Butabarbital Sodium	7.5 mg.
Phenobarbital	7.5 mg.

(WARNING: MAY BE HABIT FORMING)

Dosage: One or two tablets, one-half hour before bedtime.

Indications: For night-time sedation and refreshing sleep up to eight hours.

Contraindications: Patients sensitive to barbiturates. Use with caution in the presence of moderate to severe hepatic disease.

Supplied: Bottles of 100 tablets.

CAUTION: Federal law prohibits dispensing without a prescription.

ARMOUR PHARMACEUTICAL COMPANY
Chicago, Illinois, U.S.A.

BSP® DISPOSABLE UNIT

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A RELIABLE, PROVEN PRODUCT . . .

The BSP test, introduced in 1925, remains one of the most sensitive laboratory procedures available for determining hepatic function.

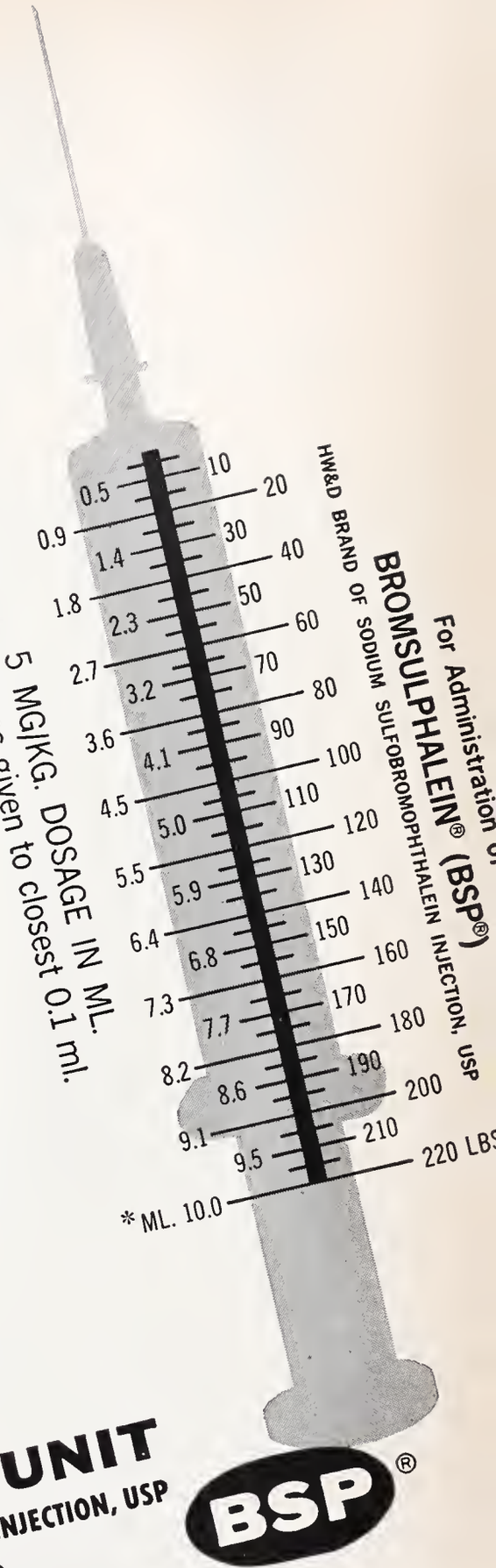
A STERILE, CONVENIENT PUT-UP . . .

Each BSP Disposable Unit contains: a sterile, glass, disposable syringe with the 5 mg./kg. BSP dosage schedule imprinted on the barrel, a sterile needle, alcohol swab and a 7.5 ml. or 10 ml. size ampule of Bromsulphalein.

The BSP unit is designed for maximum patient protection. The precalibrated syringe makes weight calculations unnecessary and provides for proper dosage regardless of patient-weight.

ECONOMICALLY PACKAGED . . .

Prepared for economic unit dispensing, this completely disposable put-up affords a savings in time and labor—the most costly commodities.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*dosages given to closest 0.1 ml.
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10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210 220 LBS
BSP®

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(50 mg. per ml.)

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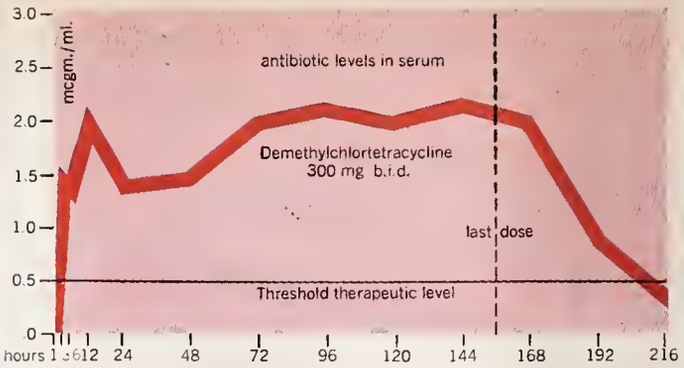
HW&D

greater potency

lower mg intake per day

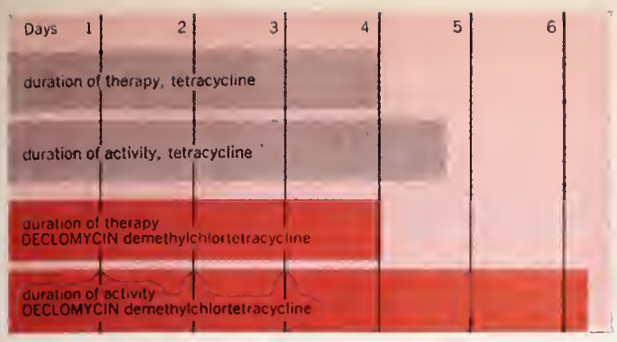
600 mg versus 1,000 mg



higher activity

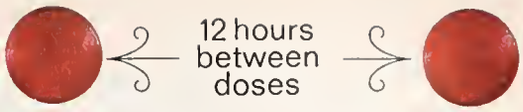
levels than ordinary tetracyclines

From Sweeney, W. M.; Dornbush, A. C., and Hardy, S. M.;
Amer. J. Med. Sci. 243:296 (Mar.) 1962



1-2 days' "extra" activity

after the last dose to protect against relapse



one 300 mg Tablet mid-morning

one 300 mg Tablet mid-evening

It's made for b.i.d.

in G.U. infections broad-spectrum performance above and beyond the activity of ordinary tetracyclines

DECLOMYCIN®
DEMETHYLCHLORTETRACYCLINE

Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

Contraindication—History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of discomfort.

Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

656-6-3762

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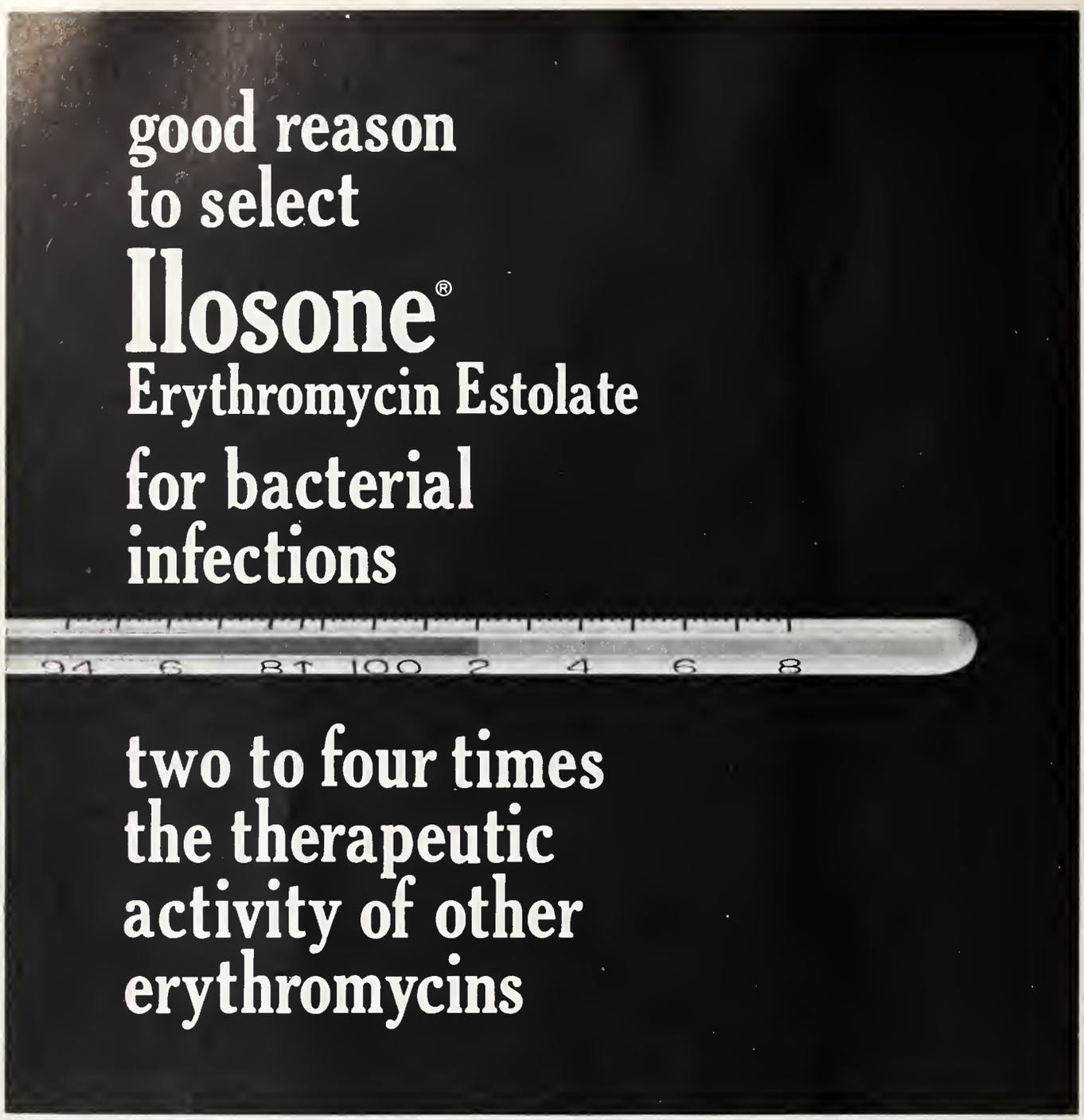

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Sucking Instinct and Mothering

ONE OF THE first responses of a newborn is to suck, and in the early weeks and months of life most species of animals, however primitive or sophisticated, indulge a baby's ability in this regard. It is simple, readily provoked by lip stimulation, and a natural way to help acquire food. Indeed, sucking is automatic in the newborn and young infant. It often persists after a feeding and is greatly decreased, or increased and exaggerated, in babies with neurologic dysfunction. In this context sucking is a neurologic act and reflects a fundamental medullary reflex pattern not primarily pleasurable nor psychologically gratifying.

But infants do not react and mature just by instinct and reflex; in accordance with distinctive inheritable and developmental roots they grow and learn through the human processes of mothering and of social stimulation and teaching. This is not to imply a dichotomy between the neurologic and environmental aspects of development but rather to emphasize their integration and to show that early infantile experiences even affect one another.

In this connection it is well known that the development of infants is profoundly influenced by the quality of mothering, an intimate affectional relationship—not restricted to mothers—of caring, talking, loving, fondling, playing, feeding, rocking, soothing, and comforting that enriches an infant's natural capacities to grow and mature to successively higher levels of complex biosocial and psychological abilities.

In our culture, however, is the long-held theory that babies possess an instinctual need—a libidinal oral need—to suck. The theory presupposes that babies derive pleasurable feelings from sucking. Once upon a time infants supposedly derived from sucking a bit of gratification devilishly akin to infantile sexual orgasms—and if it is incomplete, and the need thereby unmet and unfulfilled in infancy,

then satisfaction will be secured through such inordinate oral activities as thumb, finger, toe, pacifier, and blanket sucking and such nonoral activities as twisting hair and cuddling a stuffed animal, rag doll, or pillow.

Thus far no evidence has verified these oversimplified aspects of sucking or the claim that inadequate sucking itself is the primary cause of habitual or fixed oral behavior or a stunting of emotional and psychological development. In fact most normal infants and young children, as well as young primates, show in varying intensity and for variable periods of time a natural attachment to a thumb, finger, or blanket. In some instances, however, because nurses, physicians, and friendly neighbors think babies should have a certain quantum of total sucking time, sucking activities become a learned or partially learned pattern. In addition the ability of human newborns to be adapted successfully to nonsucking feeds indicates the dispensability of the isolated point of view that sucking is an instinctual need demanding gratification.

Eighteen years ago Fredeen[2] reported his ten-year study of feeding several hundred babies, including prematurely born babies, at birth from a cup. The infants experienced no sucking of breast, bottle, and pacifier. The study demonstrated that cup feeding, a major nonsucking experience, meets all nutritional and biological needs for normal growth and development and, in addition, is not associated with increased rates of thumbsucking, extraoral activities, and psychological difficulties in infancy and childhood.

Recently Davis and Ruiz[1] reported their psychological examinations of 20 young adults fed by the cup in Fredeen's earlier group and compared them to the test data of 60 young adults fed in infancy by the bottle, breast, or both. The personality test data indicate no correlation between nonsucking and sucking and personality, physical development, and academic achievement in young adulthood. Nor were early sucking experiences related to later psychosexual development. But of particular interest, Davis and Ruiz discovered that the "cup-fed subjects (specifically girls) . . . [were] better adjusted psychologically than the breast-fed subjects . . . [a] finding [that] should be cross-validated before any inferences may be drawn. Whether breast-fed girls in this socioeconomic group generally have prolonged or shortened nursing, have cold or warm mother, etc., may be important factors correlated with this feeding experience in this particular group. . . ."

Although the foregoing study may be regarded as inadequate—for one thing the quality of mothering in infancy is unknown—it emphasizes the need for long-term observations, especially life-long studies from one generation to the next. This is difficult in human beings, yet must be done to overcome the treachery, and often the meaninglessness, of retrospective analysis and interpretation. In this connection one is reminded of the developmental studies of Harlow's rhesus monkeys isolated at birth and raised on dummy mothers. The monkeys thrived normally but developed a deep personal attachment for diaper pads "reminiscent of the human infant's attachment to its blankets, pillows, rag dolls or cuddly teddy bear."[3] Nor did the infant monkeys grow up to become balanced adolescent and adult monkeys. Not only were they socially aberrant but after being mated their behavior toward their own young indicated they also were uniformly poor parents. Indeed they practiced a poor brand of monkey "mothering"; they neglected and rejected their offspring and punished them unduly when they cried.[4]

The significance of sucking in infancy is not its early automaticity nor a categorical instinct requiring satisfaction by sucking or feeding but its evolvement as a developmental complex increasingly affected by many factors. Chief among the latter are the attitudes of the mother and the amount of maternal handling, the process of mothering as practiced not only by the mother, father, siblings, and nonfamily persons but also as influenced by relatives, physicians, and nurses.

A neurologically handicapped infant may suck excessively, even compulsively, regardless of the quality of mothering, or the mother's attitude toward him or toward thumb sucking and mouthing a pacifier. But a baby deprived of a "psychological base of operations," reared with inadequate handling by parents and siblings, regarded in part or in large measure not as a human being but as a human object, and overstuffed with pacifiers and bottles, may become virtually a non-nutritive compulsive sucker and feeder. And, too, such an infant is apt to become stunted in the developmental process of growing up not because of a surplus of pacification but because of a deficiency of mothering. Indeed in such instances preoccupation with sucking often is a natural substitute for failure to attain mothering, the need

being not increased sucking or synthetic pacifying, nor in meeting a sucking need, but increased quality of mothering. Thus, it appears to be increasingly clear that the decisive influence in early infancy is not the amount of sucking, or total sucking time, but the quality of total mothering.

—Fred M. Taylor, M.D., Houston.

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Intrauterine Transfusions—A Pediatrician's View

FOUR MAIN problems occur in hemolytic disease of the newborn: late anemia, kernicterus, hydrops fetalis, and stillbirth. In late anemia the incompatibility of mother's antibodies for fetal red cell antigen may be mild; the infant may have only a positive Coombs' test and little jaundice. However, his hemoglobin slowly drops each week, and at six weeks he may have a hemoglobin ratio of 4 or 5 gm. per 100 ml. and incipient heart failure. Obviously, this can be prevented by checking the hemoglobin and hematocrit weekly on all infants with incompatibility factors (Rh, ABO, and other) for the first eight weeks after birth and transfusing them with 30-50 ml. of packed red blood cells if the hemoglobin falls below 7 gm. per 100 ml.

Kernicterus, also called bilirubin encephalopathy, is the yellow staining of the basal nuclei of the brain by unconjugated bilirubin. On approximately the third or fourth day a jaundiced infant may become lethargic with poor muscle tone and poor sucking reflex. This is followed by a high-pitched cry, increased muscle tone, extensor rigidity, athetoid movements, and usually death on the sixth day. This condition can be prevented by multiple exchange transfusions to keep the indirect bilirubin level below 18-20 mg. per 100 ml. in hemolytic disease of the newborn.

In 140 exchange transfusions at the University of Texas Medical Branch during a five-year period, excluding infants with severe hydrops, two died as a result of the exchange transfusion; one had severe hemorrhage due to thrombocytopenia following a second exchange and another had cardiac arrest. The latter might have been prevented, for we have gained experience in recognizing early signs of distress during exchanges. Only one infant developed kernicterus. Ironically, this was a premature infant with hyaline membrane disease, anoxia, multiple congenital anomalies with a single umbilical artery, an indirect bilirubin of 18.8 and a direct bilirubin of 1.3 mg. per 100 ml.

Finally, there is the problem of stillbirth or the newborn with hydrops fetalis. As the fetal red blood cells are hemolyzed by the mother's antibodies, anemia and high output cardiac failure result, and when complicated by loss of protein ascites, generalized edema and pericardial effusion follow. Treatment of hydrops at birth consists of resuscitation if the child is not breathing, abdominal and pericardial paracenteses, and immediate exchange transfusion establishing a deficit of 40-60 ml. to reduce the enormous venous pressure. At the University of Texas Medical Branch during the last five years, there have been five infants born with severe hydrops. Only two have survived which is true of other studies. We had no cases of hydrops due to ABO disease in our series, and I know of only one case report in the literature.[1]

The only satisfactory treatment for hydrops or stillbirth is prevention. An increased incidence of stillbirth has been demonstrated when there is a history of increased severity in previously affected infants and rising antibody titer in the mother. The low-risk mother, one with no previously severely affected infant, would not have a stillborn or hydropic infant if delivered at 37-38 weeks, but in Boggs' series, 9 of 81 low-risk pregnancies resulted in stillbirth or hydrops fetalis when allowed to proceed to term.[2] In the group of intermediate-risk mothers, those who previously have had a severely jaundiced infant due to hemolytic disease of the newborn, 44 percent of the pregnancies delivered at term and only 2 percent of those delivered at 36 weeks resulted in stillbirth or hydrops fetalis. Finally, of 17 high-risk pregnancies, those in which the mothers had high antibody titer and history of a previous stillbirth, 88 percent of the infants were lost at term, including 12 stillbirths and three deaths attributed to hydrops, but only six cases of hydrops occurred in the 20 babies delivered at 34-35 weeks (30 percent). Similar findings

have been reported from Finland. An investigator there recommended interrupting low-risk pregnancies at 38 weeks, intermediate-risk pregnancies at 36-37 weeks, and high-risk pregnancies at 34-35 weeks. The neonatal mortality rate of infants delivered before 34 weeks was too great for delivery to be recommended.

Even in the case of a previous stillbirth there is always the disconcerting fact that the present fetus may be Rh negative and if delivered early may develop hyaline membrane disease. Fortunately, when amniocentesis is performed, an Rh negative or unaffected infant can be predicted with a normal curve. The dangers of amniocentesis are said to be few, but they include the serious hazard of sensitization of the mother to the fetal red blood cells. Amniocentesis is contraindicated if the mother does not have circulating antibodies present.[3] Since delivery before 33 weeks results in a prohibitive loss of infants, intrauterine transfusions have been recommended for the severely affected fetus to prevent intrauterine death.[4]

Reports on the success rate of intrauterine transfusion average approximately 30 percent and are open to question statistically. However, when a fetus seems doomed because of a maternal history of previous stillbirth, high maternal antibody titer, and elevated delta optical density of the amniotic fluid before 32 weeks' gestation, an intrauterine transfusion seems to be its only chance for life. Hydrops will develop quickly and the opportunity to save the child later by intrauterine transfusion will be lost.

Dr. L. C. Powell, professor of obstetrics at the Medical Branch, has performed six intrauterine transfusions in the past year—two transfusions each on three fetuses. One died in utero, and another developed hydrops at 32 weeks and died 10 minutes after delivery. The third infant survived intrauterine transfusions at 30 and 32 weeks. He was delivered at 34 weeks, received four exchange transfusions, and is now a healthy infant.

An editor of *Obstetrical and Gynecological Survey* points out that ". . . these severely sensitized women are so desperate for a child that they will submit to any procedure which promises some hope; and even if intrauterine transfusions prove unsuccessful, they gain a certain psychological satisfaction in the knowledge that everything possible has been done. But it is not difficult to picture obstetricians some years hence . . . looking back on these efforts with amused indulgence, because it is to be hoped that they will then have at their disposal the only logical solution to the problem: an immunological method which will inactivate Rh antibodies."[5]

As he has predicted, these studies already are being reported.

—MYRON M. NICHOLS, M.D., Galveston.

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If Given Enough Rope...

AMERICA'S physicians have become resigned to the fact that Medicare is a law of the land and that soon they will be practicing medicine under its provisions—whether they like it or not.

When Title XIX and other basic and optional aspects are fully implemented, the American people are likely to be stunned by the cost of the Medicare program. Even the most naive citizen cannot believe that the current increase in the social security rate will be sufficient to cover the cost of Medicare. In May, 1966, Social Security Commissioner Robert Ball said that the Administration may seek a further hike in the social security tax or a hike in the wage base to give social security recipients greater benefits. He mentioned the possibility of raising the wage base from the current $6,600 to $15,000 per year—and that even this increase might be inadequate.

Most physicians have given reduced rates to elderly patients who have low incomes; and, often, these indigent elderly persons have been treated without charge. Most hospitals have been equally generous. Methods of charging for professional and hospital care under Medicare have been outlined by the Social Security Administration. It has been suggested that the physician will be expected to charge Medicare patients on the same basis as other patients, and that he

will be able to raise his fees for Medicare patients in the same order that he raises his fees for non-Medicare patients. This sounds fair. It is expected, however, that physicians will reap greater net incomes.

Physicians are human beings with the same frailties as other businessmen. The temptation to charge all that the traffic will bear exists despite some safeguards in the law. Inevitably, some physicians will follow this profitable path as they did before severe restrictions were placed on the program for dependents of the armed services personnel. Obviously, excessive charging can be accomplished in many ways other than by raising fees for an office visit or an operation—such as by unnecessary visits or operations.

At best, the Medicare program will be extremely costly. Unless all concerned use prudence and hold expenses down, the medical profession will be assailed as the villain. If charging of Medicare patients is widely abused, the medical profession can expect the noose to tighten quickly around its neck. Not only can physicians expect total coverage of all medical problems by Medicare, but nearly total bureaucratic control from Washington with the inevitable rapid deterioration of medical care.

—Everett R. Veirs, M.D., Temple.

Viruses and Cancer

Recent progress in the study of viral transformation of cells in vitro and in vivo offers new avenues in the search for the viral etiology of human cancer.

Avian and murine leukemias induced by RNA-containing viruses serve as one model for the study of leukemia in man. There are several advantages in using this model:

1. Infectious virus of distinct structure can be readily identified in vivo throughout the life span of the diseased animal, either circulating in the blood or in the malignant cells themselves.

2. Virus can also be detected throughout the life span of cultures that have undergone malignant transformation in vitro.

3. Furthermore, virus can be detected by its ability to interfere with the multiplication of related viruses in susceptible cells.

In recent studies on human leukemia, findings have been similar to those observed in virus-induced leukemias of animals. Virus-like particles, similar to the avian and murine leukemia RNA viruses, have been found in the plasma or sera of a high proportion of leukemia patients, especially children. Similar particles have also been observed in thin sections of bone marrow or lymph node cells of some patients with leukemia. The significance of these findings remains to be determined, for it has not been demonstrated as yet that these particles possess biological activity similar to the virus particles of the animal leukemias.

Recently a lymphoma peculiar to children in Central Africa has been described (Burkitt's lymphoma). Particles with the structure of herpesvirus have been detected in nuclei, not of the original lymphoma, but in nuclei of cells derived from the tumors and propagated as continuous cell lines. There is no indication as yet that the particles observed are infectious. Even if they prove to be one of the known herpesviruses or a new member of the family, the problem remains as to whether they are etiologically related to the malignancy. Similarly, infectious reovirus has been recovered from a number of Burkitt tumors. Findings of this nature exemplify the great need for caution in attributing an etiologic relationship to viruses, or other infectious agents, which may be mere passengers located in the tumor. Indeed numerous reports have claimed isolation of infectious cytopathic agents from human leukemia, either in vivo in mice, or in vitro in various tissue cultures. Upon further study it was found that the viruses isolated either were agents indigenous to the inoculated animal, or turned out to be mycoplasma which accidentally contaminated the cell cultures inoculated with the test material.

Recent electron microscopic studies reveal the finding of yet another type of virus-like particle, namely particles indistinguishable from papovaviruses, in autopsy material from patients suffering from progressive multifocal leukoencephalopathy (a disease superimposed on either chronic lymphatic leukemia or other diseases of the reticuloendothelial system). These observations also await further tests as to whether specific biologic activity accompanies the morphologic findings of virus-like particles.

Another similarity between human leukemia and the virus-induced leukemias of animals, especially avian leukemia, comes from studies of cells derived from leukemia

patients and studied in long-term tissue culture. It had been observed that cells of various organs derived from chickens infected with avian myeloblastosis virus undergo a spontaneous conversion to myeloblasts when propagated in vitro. As with the experimental in vitro transformation of normal chicken target cells by this virus, the spontaneous proliferation of myeloblasts in cultures of infected organs is attributed to the transforming capability of the virus. Similarly spontaneous conversion to immature lymphoid cells has been found in fibroblastic cultures of bone marrow cells derived from children with acute leukemia or infectious mononucleosis. These lymphoid cells can be propagated in culture indefinitely and have been extensively studied. Lymphoid and myeloid cell lines have been derived from peripheral blood cells of patients with lymphocytic or with myelogenous leukemias; lymphoid cells derived from Burkitt's lymphomas have also resulted in permanent cell lines.

However, attempts to demonstrate a cytopathic, or interfering, or cell-transforming agent in any of these human leukemia cells have thus far failed. Thus, in contrast to the findings in virus-induced animal leukemias, in which infectious virus can be demonstrated with relative ease, work on human leukemia has been suggestive of viral causation but it has not been rewarding as to actual isolation of an active biological agent etiologically related to the disease.

There may be many reasons for this failure. First, there is the obvious drawback of the lack of susceptible host for in vivo experiments. It is hoped that the current trials with newborn primates in the laboratory may yield positive results in the years to come. Second, the concentration of infectious virus in the naturally occurring disease of man may be very low. Third, the tissue culture system used to demonstrate the transforming capacity of material derived from the patient or from cell lines grown in culture may not be sensitive enough. As shown with avian myeloblastosis virus, only specific target cells undergo transformation. Fourth, if indeed human leukemia is caused by a virus, the responsible virus may be a defective one and special procedures may be needed to demonstrate its presence. Thus many avenues remain open in the search for a viral agent in human leukemias and lymphomas, in which models offered by the RNA tumor viruses of animals may be followed.

In view of the finding, with few exceptions, that solid tumors in animals induced by the adeno- and papovaviruses are free of infectious virus or of infectious nucleic acid, it is not surprising that innumerable attempts to isolate viruses from a variety of solid human tumors have met with complete failure.

The new knowledge of virus-induced T antigens in tumors (or in transformed cells) and of T-antibodies in the tumor-bearing animals has led to similar immunologic studies of cancer in man. Several attempts, unsuccessful as yet, have been made to test sera of cancer patients for the presence of complement-fixing or immunofluorescence antibodies to possible T antigens in the patient's own tumor cells.

The finding that the DNA tumor viruses induce these T antigens, not only in the cells they transform but also in the early stages of cytocidal virus replication, offers a model for similar studies with known viruses of man not yet implicated in animal carcinogenesis. Under the sponsorship of the National Cancer Institute, cooperative studies using the sera of cancer-bearing patients are being planned. Candidate viruses for such studies include not only the adenoviruses but also other viruses of man. For example, strong candidates include members of the herpesvirus group, such as simplex, zoster, and cytomegalovirus, all known to exist in a latent state in man for long periods of time.

A converse approach employs antisera of tumor-bearing animals that contain T-antibodies for the known tumorigenic viruses—those in papova and adenovirus groups. These antisera are being studied to determine if they will detect the presence of virus-induced T antigens in human tumors.

The techniques for searching for viruses in human cancer have become much more sophisticated than those currently used in the virus diagnostic laboratory. However, investigators must still contend with the problem of identifying "passenger" viruses present in the cancer but not in a causal relationship. Of even greater concern is the converse, the problem of identifying a virus no longer present in the cancer that it may have caused. Some of the approaches that have proven fruitful in the study of malignant changes in cells and animals might illuminate the problem of malignant transformation in relation to human neoplasm.

—Matilda Benyesh-Melnick, M.D.,
Houston.

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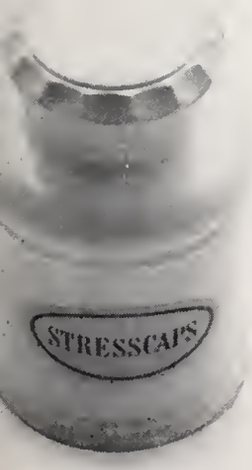

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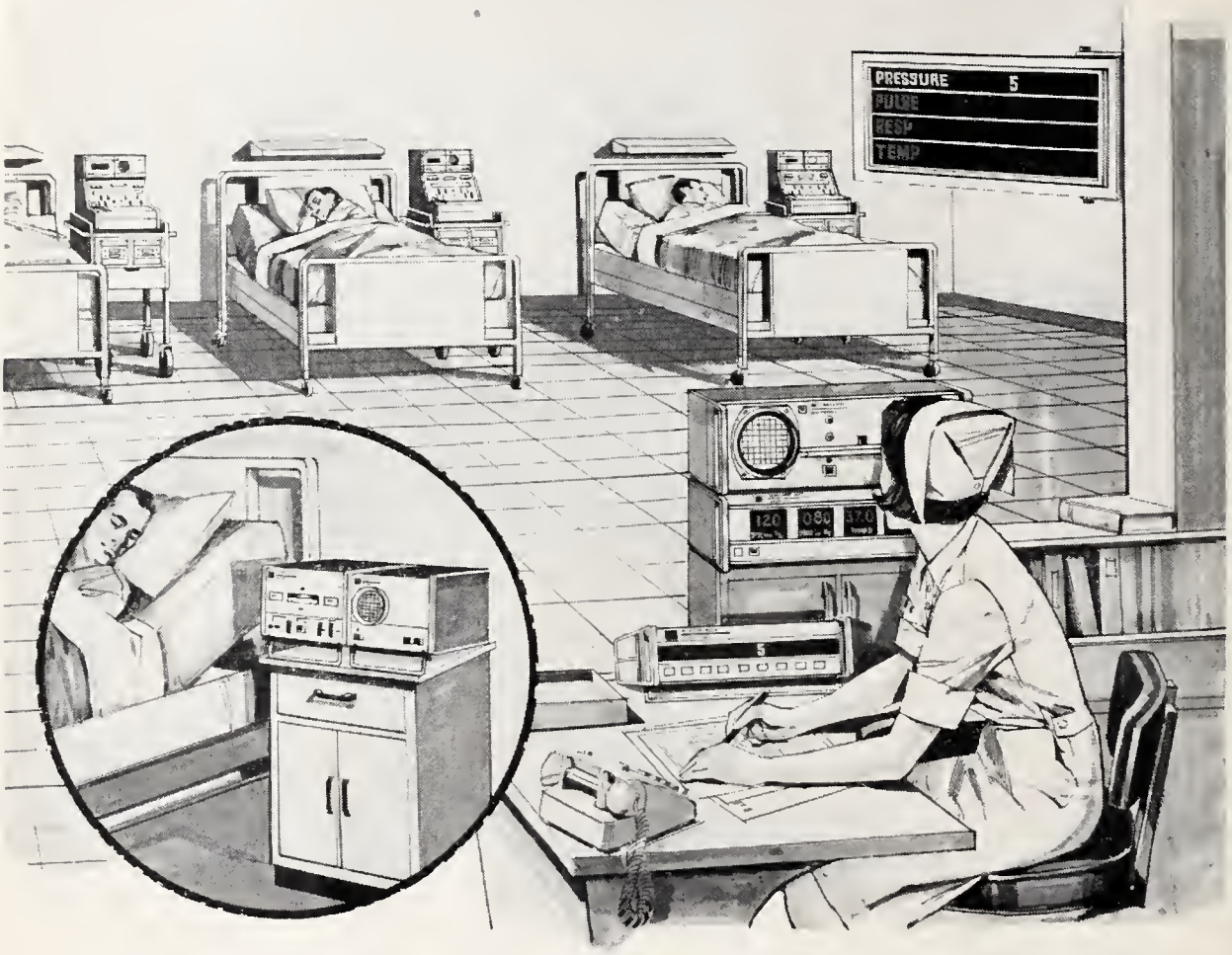

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TITLE XIX—MEDICAID

Perhaps little attention was given TITLE XIX of Public Law 89-97 until the State of New York recently passed a plan whereby a family of six with an annual net income of over $10,000 could qualify for free medical care.

Texas physicians have always believed that medical indigents should receive the best available care whether in our offices, city or county hospitals or state facilities. We have rendered free medical service, which can be conservatively estimated at some $100,000,000 each year, to these less fortunate Texans, year after year.

Presently, some twenty-four states expect to have their TITLE XIX plan operational by the end of 1966. Texas and many other states need implementing legislation to put this law into force. Each state is allowed considerable leeway in defining the extent or limits of TITLE XIX, but final approval by the United States Department of Health, Education and Welfare is mandatory.

This week a special committee was appointed with Dr. Charles Max Cole, Dallas, as chairman. This committee will serve in an advisory capacity to the State Department of Public Welfare, and is charged with the responsibility of seeking legislation to provide care for the medically indigent citizens of Texas. The committee will also study the impact of such legislation on medical education, on the general and city-county hospitals, and on medical practice.

The committee invites your suggestions.

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meet the authors

IRVING A. RATNER is in private practice of pediatric surgery in San Antonio. **Congenital Duodenal Obstructions of Infancy, p. 44.**

PETER ZANCA is acting chairman of the Department of Radiology, South Texas Medical School, San Antonio. **Infusion Pyelography in Infants and Children, p. 49.**

ALEX K. MUNSON, Austin specialist in child and adolescent psychiatry, has, in addition to four children, a household of neurotic cats, carefully diagnosed, but unrecorded for medical annals. For us, he discusses the **Minimally Brain-Damaged Child, p. 54.**

ROBERT E. ROSSMAN practices dermatology in Tyler. **Pseudoxanthoma Elasticum, p. 60.**

DON RUSSELL, resident in internal medicine at Southwestern Medical School, Dallas, presents this month's **Clinicopathologic Conference, p. 66.**

WALTER C. BARNES was an army surgeon (Pennsylvania and Eniwetok) and was fellow to Dr. Guy W. Horsley, Richmond, Va., before moving to Texarkana. **Management of Inguinal Hernia in Infants and Children, p. 40.**

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Children: 3 to 6 months — 3 mg. (½ tsp.* t.i.d.)
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1 to 2 years — 5 mg. (½ tsp. 5 times daily)
2 to 5 years — 6 mg. (1 tsp. t.i.d.)
5 to 8 years — 8 mg. (1 tsp. q.i.d.)
8 to 12 years —10 mg. (1 tsp. 5 times daily)

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily)

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the therapeutic dose.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

SEARLE *Research in the Service of Medicine*

Management of Inguinal Hernia In Infants and Children

REVIEW ARTICLES

INGUINAL HERNIA is the most common congenital anomaly treated by surgery, and inguinal herniorrhaphy is the most frequent operation performed on infants.[1] Yet great confusion and disagreement exist as to the operative management of this disorder. The literature is contradictory to the point of confusion, especially for the neophyte surgeon and the occasional operator. In 1962, Sparkman summarized the existing material, and reported his own statistical experience.[2]

This article presents an approach to the problem which has been a rational and satisfactory one for me.

Embryology

The embryologic development of the region is such that the vast majority of inguinal hernias in infancy are indirect. During the middle trimester of gestation, the developing testis descends from its original position at the lower pole of the kidney into the iliac fossa. Beyond this point, the overlying peritoneum moves with the testis as a diverticulum, the processus vaginalis peritonei. The point from which this begins will later be the internal inguinal ring. The processus vaginalis parallels the vas deferens and spermatic vessels. These structures, plus their enveloping coverings derived from the various layers of abdominal wall, comprise the spermatic cord.[3]

The gubernaculum testis, a homologue of the ovarian ligament, connects the lower pole of the testis to the scrotum. As the gubernaculum shortens, it literally draws the testis into the scrotum. By the ninth month, this descent is complete, and the distal end of the processus vaginalis becomes detached from the proximal, or funicular, portion, forming the tunica vaginalis testis. The abdominal end then obliterates, closing the internal inguinal ring.[3] If this closure fails, a *potential* hernia exists. (I emphasize "potential," since the equating of a patent processus vaginalis with an inguinal hernia is erroneous, and has led to much of the confusion in the literature. The first is a simple anatomic fact; the second, a pathologic state.) Approximately 95 percent of the indirect hernias are of the funicular type, resulting from simple failure of closure of the proximal processus vaginalis. In the other 5 percent, the entire processus remains open, leading to a complete, or scrotal, hernia. In some instances, the processus is narrowed, producing a communicating hydrocele. All hydroceles in infancy probably communicate with the abdominal cavity, and, therefore, are not true hydroceles.[4-6] Normally, the processus vaginalis closes on the left side three weeks before birth, and on the right side, two weeks before birth.[6] This presumably accounts for the fact that hernias occur twice as often on the right side as on the left.

Diagnosis

The diagnosis of inguinal hernia usually has been made before the surgeon sees the child. The mother may notice a mass in the groin while bathing the crying infant, or the older child may notice a bulge himself. Hydrocele of the cord or scrotum, or cryptor-

Careful preoperative examination will indicate whether the testicles are in the scrotum, and distinguish between incarcerated hernia and hydrocele. The contralateral side is evaluated. Surgical repair, simple high ligation and excision of the sac, includes narrowing the internal ring with nonabsorbable suture. Indications for surgery, precautions, and possible complications are reviewed, as is the work of other authors.

WALTER C. BARNES, JR., M.D.

chidism, may be the presenting complaint. Many hydroceles are mistaken for incarcerated hernias; this is not an uncommon error in the referral diagnosis.

Differentiation between incarcerated hernia and hydrocele is generally easy. (1) A child quietly sleeping or happily taking its bottle rather than crying with colic, (2) transillumination of the mass, and (3) demonstration of an empty space between mass and internal ring usually indicate a hydrocele. However, an incarceration at the internal ring can make the patent processus edematous, simulating a hydrocele. Presence or absence of the testis should be carefully noted.

Evaluation of the contralateral side before surgery is important in deciding whether or not bilateral exploratory surgery of the groin is indicated. The following factors affecting the incidence of contralateral hernias should be considered. (1) Undescended testicle or (2) presence of a hydrocele on the opposite side are absolute indications of contralateral hernia. (3) A child born prematurely or (4) a presenting hernia on the left side are relative indications to be considered along with other factors. Davis believes that there is no significance as to the side on which the presenting hernia occurs.[4] The value of the "silken glove" sign is debatable. If it is present and unmistakable, I think it is helpful; if not, it means nothing. Clatworthy and associates place little value on the sign.[8]

Sparkman believes that with careful preoperative evaluation, a contralateral hernia will be detected in 10 percent of cases.[2] This agrees with my experience, and it is not out of line with the more conservative incidences reported as the result of bilateral surgical exploration, or on careful follow-up after unilateral repair.[2,4,5,7-12] Those who advocate routine bilateral exploratory surgery quote operative incidences ranging from 50 to 90 percent.[2,6,11,13-15] Santulli and Shaw report an incidence difference of 50 percent between contralateral hernias found at routine bilateral exploration, and contralateral hernias later occurring after routine unilateral repair.[11] Potts has stated that it is actually a bit of a temptation to pull on a tiny projection of peritoneum and call it a sac. Perhaps, then, the problem is one of definition—are we referring to hernia or patent processus vaginalis?

Time and Technique of Treatment

Hernias should be repaired whenever they are found, so long as the patient weighs at least 10 lb., and there are no contraindications to surgery, such as intercurrent illness. Some authors use one month of age, rather than 10 lb. of weight, as the criterion for the earliest time for elective repair. Yarn trusses are not used except to maintain hernia reduction during illness which delays repair. Aspiration of hydroceles and injection of hernias with sclerosing substances are mentioned only to be condemned.[6] If incarceration exists, the hernia should be reduced immediately and then repaired 48 hours later.[1] In 90 percent of the cases, an incarcerated hernia can be reduced either manually or by sedating the baby and placing him

in Trendelenburg's position. If reduction is impossible, and I would caution against too vigorous an effort, immediate repair should be made.

Surgical Procedure

Most authors agree on the type of repair.[1,5,6,9,13,15-17] As with any pediatric surgery, meticulous attention to detail and gentle handling of tissues is imperative. Careful hemostasis is important. Whatever name is applied to the operative technique, an incision should be made in the abdominal fold immediately below the internal ring. Scarpa's fascia is well developed and must not be confused with the external oblique fascia. The external oblique fascia is split in the line of its fibers over the internal ring, and the fibers of the external ring are kept intact. Fibers of the cremaster muscle in the upper cord are teased apart, and the sac is identified.

Great care is taken to protect the ilioinguinal and iliohypogastric nerves, the vas deferens, and the cord vessels. By blunt and sharp dissection, the sac is freed high at the internal ring, twisted to give it substance, and sutured with nonabsorbable material. I do not advocate the use of absorbable suture; in my only cases of recurrence, catgut was used. If a complete, or scrotal, hernia exists, it is not necessary to remove the entire sac. Rather, it may be transected below the internal ring and then high ligation carried out, leaving the distal portion of the processus in situ.[4,6] This is safer than a complete scrotal dissection.

Following this, the internal ring should be narrowed by inserting one or two nonabsorbable sutures beneath the cord in the angle where the cremaster muscle and fascia derive from the internal oblique muscle. A suture is then placed in the original cremaster incision, and the external oblique fascia closed. After closing Scarpa's fascia, the skin is repaired with subcuticular sutures, and a spray dressing applied. No restriction is placed on the infant's activity, and he is dismissed from the hospital the following day if no contraindication exists.

Complications

Recurrences are usually associated with (1) a tear in the peritoneum, (2) use of absorbable suture, (3) failure to ligate the sac high, or (4) failure to narrow a wide internal ring.[4] Repair should be carried out in the manner previously described. Other complications are undescended testicle, maldescent of the testicle, sliding hernia, and direct hernia; the latter two are fairly rare.

Direct hernia necessitates opening the entire inguinal canal and mobilizing the cord; both steps are avoided in the technique described. Support for the floor of the canal is gained through a modified Bassini repair, and the remainder of the technique is carried out.

In sliding hernia there is usually no sac to be removed. Rather, the prolapsing viscus and adjacent peritoneum are reduced by insertion of several purse-string sutures. The internal ring then is narrowed and the wound closed.

When the testis is undescended, the canal is opened widely, and the testis and cord are carefully and thoroughly mobilized high into the internal ring. The scrotum is then inverted into the wound and the testis attached to the base of the scrotum by two sutures to prevent torsion. A completely patent processus vaginalis will be present, and should be obliterated by high ligation and division.[18] The remainder of the repair is routine.

After trying absorbable suture, a variety of operative techniques, and routine "blind" exploration of the contralateral side, I have arrived at the described method of management. Recurrences have been rare, and later occurrence of contralateral hernias has been no problem.

Summary

A workable plan is presented for the management of inguinal hernias in infants and children. Careful examination to detect testicles in the scrotum, incarceration, or hydrocele is imperative, and the contralateral side is meticulously evaluated for possible hernia.

At surgery, simple high ligation and excision of the sac, and narrowing of the internal ring with nonabsorbable suture is advocated. This technique has been variously called the Mitchell, Ferguson, Banks, or Potts procedure. The one-stage Gross technique is preferred for repair of cryptorchidism.

Postoperatively, no restriction is placed on the patient, and he is dismissed from the hospital the following day.

COMMENT

An accepted and conservative method of treating inguinal hernia in children is presented; Dr. Barnes also gives his modification for tightening of the enlarged internal inguinal rings. His list of indications, precautions, and complications to be avoided deserve emphasis. The risks of any treatment are evaluated with the hazards of the disease; these risks are as individual as the doctors and the patients involved. Concurrent gastrointestinal, respiratory, hematological, cardiovascular, or other mesenchymal disease adds to the risk of anesthesia and operation as well as to the risk of hernial complications such as the frequency of incarcerations, strangulations, intestinal obstruction, and perforation.

Anesthesia can be fatal; it also can result in brain and emotional trauma which may handicap the maturity of both the patient and family. Damage to the vas deferens or fallopian tubes, or the loss of or growth failure of both gonads may be the result of strangulation by the incarceration, the operation, or a combination of these. Only examination of all removed tissue along with a long-term follow-up can give some functional evaluation. Wound infection and damage to adjacent bladder or iliac vessels have occurred. Recurrences are rare, but obviously they are the lesser of the complications. Constant alertness to avoid major complications is an absolute necessity in "minor" operations.

—Luke W. Able, M.D., Houston.

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Cadmium Fumes Menace to Hobbyist

The hobbyist who uses silver solder containing cadmium metal may be playing a game as deadly as Russian roulette.

Dr. Paul Joliet, chief of the US Public Health Service's Division of Accident Prevention, warns that recent deaths related to the industrial use of silver solder containing cadmium underscore the need for hobbyists to be aware of the danger in home workshops.

Cadmium, when overheated, vaporizes and produces cadmium oxide, a highly dangerous fume which can cause death when inhaled in small quantities.

The risk lies in the fact that the fumes are practically odorless and lethal doses can be inhaled without any irritation or discomfort that would give a warning. It sometimes takes from four to eight hours for serious symptoms to develop.

Dr. Joliet pointed out that the commonly used tin-based solders do not present this hazard.

Congenital Duodenal Obstructions In Infancy

REVIEW ARTICLES

CONGENITAL DUODENAL obstruction in newborns usually is the result of malrotation, atresia, or annular pancreas, or a combination.

Malrotation

Malrotation is the commonest cause of duodenal obstruction. Normally, intestinal rotation occurs during the eighth to tenth weeks of embryonic development. The intestines move counterclockwise to position the duodenum on the right side, the jejunum on the left, and the cecum and appendix in the right lower quadrant.

Arrest in the normal sequence of intestinal rotation can occur at any stage in the process, and the resulting varieties of incomplete rotation are all classified as malrotation.

Accompanying malrotation, the root of the mesentery of the small intestine may be short, facilitating volvulus of the midgut. The jejunum, ileum, cecum, ascending and transverse colon may be twisted several times, clockwise, around the mesentery (Fig. 1). This may compress the superior mesenteric vessels and, if untreated, lead to infarction of the midgut from the jejunum to the middle of the transverse colon.

The malrotated cecum is held tightly over the junction of the second and third portions of duodenum by peritoneal bands extending from the cecum to the right gutter. Occasionally, intrinsic stenosis of the duodenum adds to the obstruction.

Clinical symptoms and signs of malrotation are mild to severe depending on the degree of obstruction and whether or not there is associated midgut volvulus.

In the first 24 hours of life, infants with midgut volvulus may show no signs or symptoms other than bile vomiting.

On the first or second day of life, the infant usually begins vomiting bile, but has little abdominal distention. He might have normal meconium stools or no stools at all.

Midgut volvulus impairing vascular function in the involved intestine can cause shock; the infant may be pale or gray, and have

Fig. 1. Appearance of the intestines in malrotation.

Presented to the TMA Section on General Practice during the 1965 Annual Session in San Antonio, May 1, 1965.

Surgical management of malrotation, duodenal atresia, and annular pancreas is described. Symptoms include vomiting bile and a little abdominal distention. A "double bubble" of air seen on plain flat and upright roentgenograms is a helpful diagnostic sign.

IRVING RATNER, M.D.

tachycardia, abdominal distention, and no peristaltic sounds.

Preoperative management includes immediate passage of a nasogastric tube (usually at least size 10 F.) and emptying the stomach to prevent aspiration of bile. Laboratory determinations should include serum electrolytes, blood urea nitrogen (BUN), complete blood count and urinalysis.

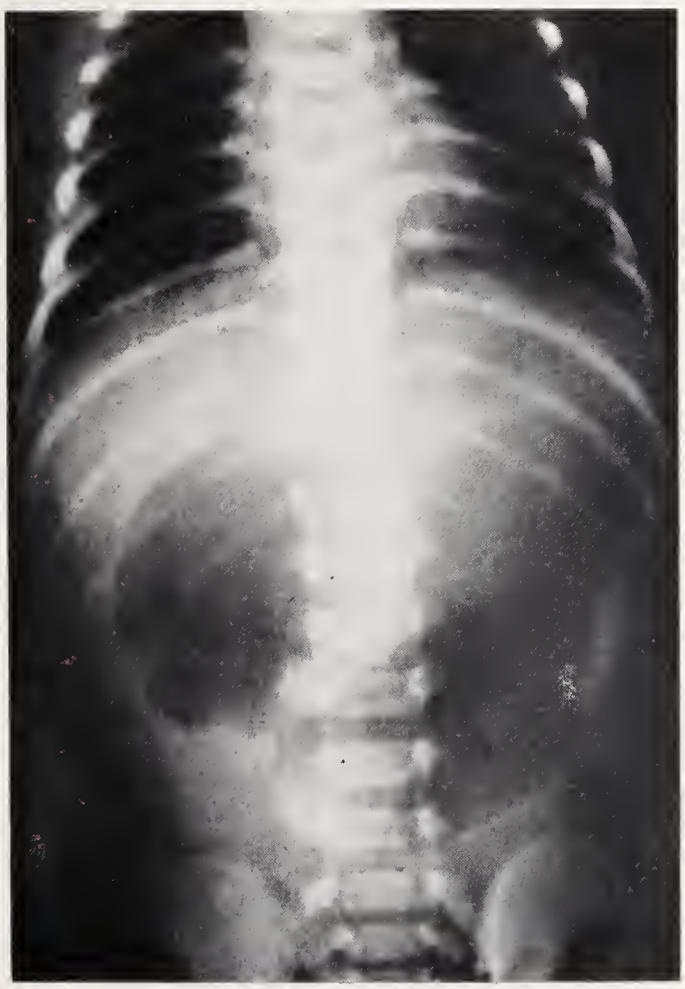

Fig. 2. Plain flat roentgenogram of the abdomen shows two bubbles of air in the upper portion.

Plain flat and upright roentgenograms frequently show a "double bubble" of air—one in the stomach and one in the dilated proximal duodenum (Figs. 2 and 3). If the duodenal obstruction is complete there may be no other air in the intestinal tract.

Although barium enema examination often does not help determine the cecum's position in infants, this study will occasionally demonstrate a malrotated colon and will always determine the colon's patency. This knowledge is valuable during subsequent explora-

Fig. 3. Upright roentgenogram shows "double bubble" of air in upper abdomen and lack of intestinal gas in lower tract.

tory surgery of the abdomen. Roentgenograms of the upper gastrointestinal tract usually are not necessary, particularly if the "double bubble" is seen on plain films of the abdomen.

Intravenous fluids (40-50 ml. per lb. of body weight) may be necessary preoperatively if the infant is older than 24 hours and has been vomiting large amounts. This is usually given as 5 percent dextrose in 0.2 to 0.35 percent saline, and the volume may be doubled if the infant is dehydrated. Skin turgor, skin and mucous membrane appearance, the degree of fullness of the fontanelles, and urine output are useful indicators of degree of dehydration, and it is neither necessary nor desirable to catheterize the infant's urinary bladder. Urine specific gravity, BUN, and hematocrit determinations are also helpful guides. If the baby has needed fewer than three diaper changes in eight hours, he is probably dehydrated.

If the infant is anemic, he should be given a blood transfusion (usually not exceeding 10 cc. per lb. of body weight) before surgery.

It is desirable to operate as soon as possible lest infarction of the intestine occur.

The anesthesia of choice varies with the experience of the anesthesiologist. Drop ether, cyclopropane, and fluothane are all safe in experienced hands and if used in small quantity. No attempt should be made to achieve a great amount of relaxation. We do not use or recommend the use of muscle relaxants unless the anesthesiologist has considerable experience in their use *in infants.* Endotracheal intubation is desirable particularly if cyclopropane or fluothane is used. A size 18 polyethylene catheter is inserted into the saphenous vein at the ankle just before surgery.

The abdominal cavity is opened through a mid, right rectus, muscle retracting incision. It is necessary to remove the intestines from the abdominal cavity to determine whether there is midgut volvulus. If so, it is reduced by rotating the midgut counterclockwise (Fig. 4). A warm saline pack is then placed over the small intestines to aid return of viability. Should the midgut be infarcted and nonviable it may require resection, possibly from jejunum to midtransverse colon. An aseptic, closed, one-layer, end-to-end anastomosis is then performed. Occasionally an infant will survive such a massive resection, although he will have considerable nutritional difficulty. Failure to resect infarcted intestine will lead to certain death of the infant.

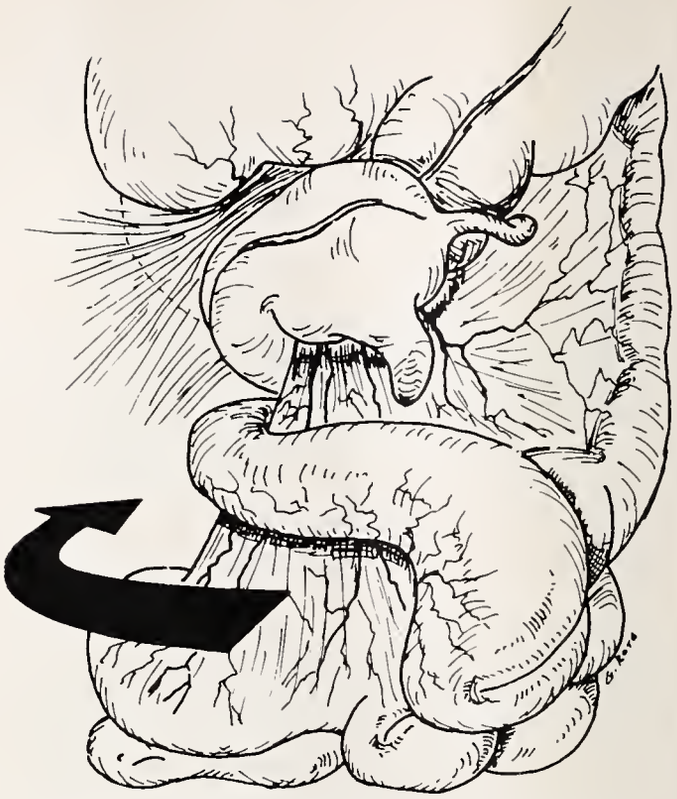

Fig. 4. Midgut volvulus is reduced by counterclockwise rotation. A warm saline pack aids return of viability.

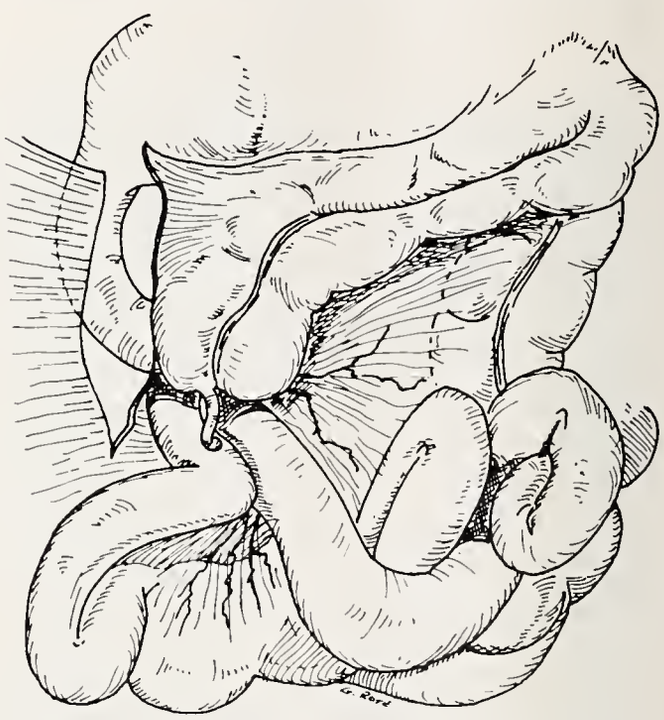

Fig. 5. Peritoneal bands are divided to release the cecum, relieving pressure from the duodenum.

The peritoneal sheet (Ladd bands) which binds the cecum to the right upper quadrant is incised (Fig. 5). Usually, a few small, short, cecal vessels must be divided to free the cecum. The cecum and ascending colon are placed in the left upper quadrant of the

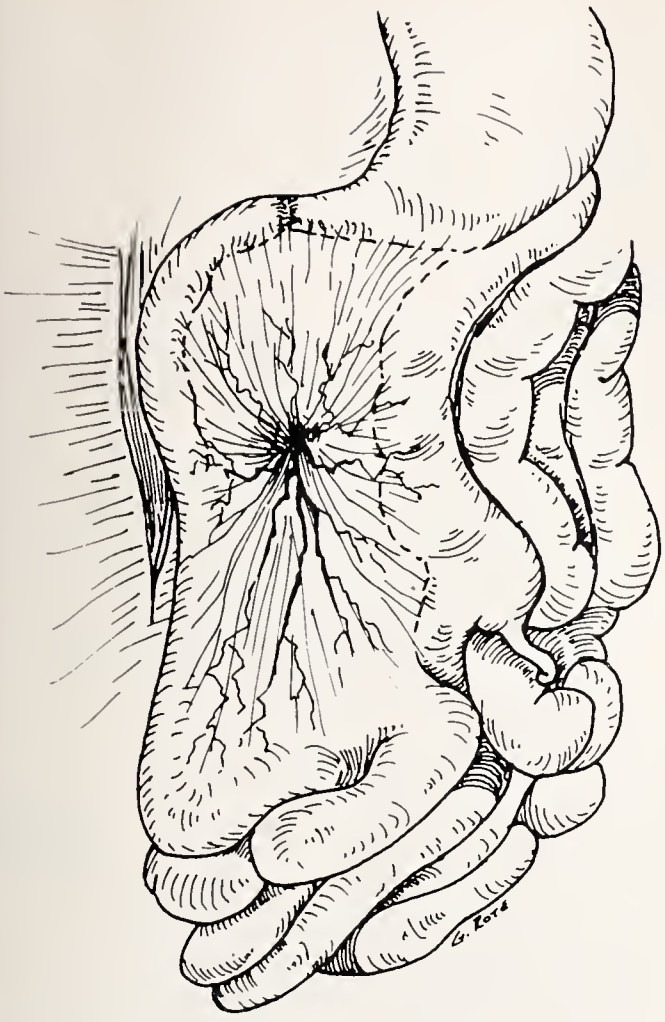

Fig. 6. At the conclusion of the operation, the duodenum has a straight, unobstructed course on the right side and the cecum and ascending colon are placed in the left upper quadrant of the abdominal cavity.

abdominal cavity, and the duodenum has a straight, unobstructed course on the right side (Fig. 6).

Should there be duodenal stenosis in addition, there should be no hesitation in performing a two-layer, side-to-side, isoperistaltic, duodenojejunostomy. A direct surgical approach to atresia or stenosis in this region of the duodenum is extremely hazardous, because in infants this size the pancreatic ducts cannot be seen, and may be injured inadvertently.

Postoperatively, nasogastric drainage and parenteral fluids are continued until peristalsis returns. This usually takes four or five days. During this time, the infant receives 30 to 40 ml. per lb. of body weight of 5 percent dextrose in 0.25 percent saline, plus replacement of measured gastric losses with Ringer's lactate. After the first postoperative day, 2-3 mEq. of potassium chloride per 100 ml. of intravenous fluid is given. Serum electrolytes are monitored every third day as a guide to fluid replacement. Urine output may be estimated by the number of diaper changes per eight hours.

When peristalsis returns, the nasogastric tube is removed and sugar water is fed to the infant. If he tolerates two ounces every two to three hours without vomiting, he can advance to boiled skim milk and then to his usual formula during the next 24 to 48 hours.

If the infant cannot tolerate sugar water or skim milk feedings after 7 to 14 days, it is likely that an operative complication, probably intestinal obstruction, has developed. Exploratory surgery should be performed forthwith before all his body reserves are depleted. Infants with malrotation who are treated within the first three days of life, and before infarction of intestine occurs, can have a normal life.

Duodenal Atresia

Duodenal atresia, a less common cause of obstruction, can involve all layers of the duodenum or only the mucosal layer. In either case the obstruction usually is just distal to the ampulla of Vater at the junction of the second and third portions of the duodenum.

The etiology of duodenal atresia is not known; some believe the disorder results from incomplete recanalization of the duodenum from its solid tube stage of development, and others believe it is secondary to a vascular accident affecting the duodenum early in embryonic development.

The symptoms are similar to those of duodenal obstruction secondary to malrotation. Bile vomiting with mild upper abdominal distention begins during the first day of life. There may be one or two meconium stools before bowel movements cease. Peristalsis usually is normal. Occasional gastric peristaltic waves may be visible.

Preoperative and anesthetic management is the same as for patients with malrotation.

At surgery, a retrocolic, two-layer, isoperistaltic, side-to-side duodenojejunostomy is performed to bypass the atretic segment. No attempt is made to resect the atretic segment lest the pancreatic ducts, which are not visible, be inadvertently injured. Before closing the anastomosis, a number 8 F. catheter placed into the jejunum allows infusion of 4-5 cc. of a 0.05 percent solution of methylene blue, and the surgeon gradually "milks" the dye down to the cecum to test patency of the remainder of the small intestine. Occasionally multiple segments of intestinal atresia must be resected.

Postoperative care is as previously described.

The likelihood of survival decreases considerably if the infant is premature or if he has other congenital anomalies such as heart defect, esophageal atresia, or imperforate anus.

Annular Pancreas

The least common cause of duodenal obstruction in infancy is annular pancreas. It is believed that when bilobate ventral pancreatic buds fail to fuse, the pancreas subsequently encircles and compresses the second portion of the duodenum, causing varying degrees of obstruction. Occasionally the symptoms are so mild that the patient reaches adulthood before his condition is diagnosed.

If the obstruction is complete, or nearly so, the clinical findings are indistinguishable from those of duodenal atresia.

Management is the same as for duodenal atresia. No attempt is made to divide the annular pancreas lest fatal pancreatitis result.

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Sex Is Factor in Cancer Survival Rates

Among patients operated on for lung cancer, women live longer than men. Scientists at the National Cancer Institute, National Institutes of Health, have confirmed this earlier finding. In their study of 504 women and 511 men, the Public Health Service statisticians also identified several factors that help to explain this sex difference in survival rates: the type of tumor, extent of disease, and amount of surgery performed.

A major difference was found between men and women in type of tumor, with adenocarcinomas and alveolar cell tumors occurring about three times more frequently in women than in men. Among patients with these types of tumors, survival rates were substantially higher for women, while among patients with epidermoid carcinoma—the most common type of lung cancer in men—survival rates in men and women were about the same. Among both men and women with adenocarcinomas or alveolar cell tumors, three of every four had growths confined to one lobe; whereas among patients with epidermoid carcinomas, women were more likely than men to have a tumor of limited extent.

The more frequent occurrence of adenocarcinomas and alveolar cell tumors in women, and the more frequent occurrence of tumors limited to one lobe account for the fact that, among surgically treated patients, almost one-half of the women, compared to one-fourth of the men, had only part of the lung removed. However, among all patients with localized disease treated by lobectomy, the outlook remained substantially more favorable for women than for men. Thus, part of the survival advantage in favor of women remains unexplained by the present study.

Infusion Pyelography In Infants and Children

PETER ZANCA, M.D.

Technique, advantages, and successful use of drip infusion pyelographic examination of infants and children is reported. Large volumes of contrast medium with physiologic saline solution were used, and side reactions were nonexistent in this series.

CLINICAL MEDICINE

DRIP INFUSION of contrast medium for pyelographic study of the urinary tract in adults is a useful, dependable, and safe procedure; its advantages over the conventional intravenous injection technique have been reported.[1-3] For the past two years we have had good success with infusion pyelography in the examination of infants and children. Using large volumes of contrast medium (meglumine iothalamate 60 percent), we obtained good or excellent films of the collecting urinary system in 27 of 34 patients. Early and precise anatomical and pathological diagnoses have been established.

This article describes the technique of this examination for infants and children, and reports its usefulness and safety as an adjunct to intravenous pyelography, or as a substitute for retrograde pyelography in selected cases.

As far as is known, the drip infusion pyelographic technique for infants and children has not been described previously.

Material

Between Dec. 10, 1963, and May 5, 1965, 34 infants and children in Robert B. Green Memorial Hospital, San Antonio, underwent infusion pyelography. Of these patients, 22 were thought to have pathologic conditions of the urinary tract, and 12 had various other conditions. Clinical diagnoses of the urinary tract abnormalities included glomerulonephritis, pyelonephritis, hematuria, kidney trauma, cystitis, tumor of the kidney, possible congenital anomaly of the urinary tract, and obstructive uropathy. Included in the second group of pathologic conditions were Hodgkin's disease, histiocytosis X, retinoblastoma, arachnodactyly, possible mixed sex, intestinal obstruction, abdominal mass, and "failure to thrive." The patient's ages ranged from 16 hours to 14 years. There were 14 boys, 19 girls, and 1 child in whom hermaphroditism was suspected. Only 5 of the 34 patients had undergone previous routine intravenous pyelographic examination, and they later had infusion pyelographic examinations because of indeterminate radiographic results. The remaining 29 patients were selected at random. Table 1 shows the results of infusion pyelography in the 34 patients.

Technique

Attention to details when performing the infusion pyelographic examination in infants and children is essential to good roentgenographic technique and for optimum pyelographic films. Consistently good results have been achieved with the following technique:

1. Preparation of the patients consists of a cleansing enema until the return is clear; there is no dehydration.
2. Abdominal compression bands are not used.
3. An anteroposterior (AP) scout kidney, ureter, and bladder film is taken and checked by the radiologist for technique.
4. Conray (meglumine iothalamate 60 percent), 2 cc. per kg. of body weight is mixed with an equal volume of normal saline solution and placed in an infusion bottle.
5. A 23-gauge needle is placed in a scalp, antecubital or leg vein, and the diluted contrast medium is allowed to drip rapidly into the vein. A total of about 150 cc. of the solution will be infused in about 15 minutes.
6. A carbonated beverage is given to the

patient 5 or 10 minutes before the beginning of the infusion to increase the gastric gas bubble, allowing better visibility of the calyceal structures.

7. A proportional small amount of the contrast medium is used as an intravenous test dose. If there is no immediate reaction the remainder of the solution is infused.

8. Pyelograms in the AP projection are taken 10, 15, 20, and 30 minutes from the commencement of the infusion. Each film is reviewed by the radiologist, and oblique, lateral, prone and spot films are taken if needed.

9. Additional AP, lateral, and oblique cystographic films and voiding cystourethrograms are obtained, if indicated. Nephrotomograms and delayed urograms are performed for differential diagnosis of cysts from tumors and for renal function.

Results

There were no immediate or delayed allergic or toxic reactions in any of the patients. Good or excellent quality films were obtained in 27 of the 34 cases. In this group, a prolonged nephrogram effect was produced, allowing excellent visibility of the outline, size, and position of the kidneys. Anatomically, the calyces, renal pelves and ureters were clearly seen in their entirety; and the urinary bladder outline was enlarged and densely opacified. The high density and large volume of contrast medium in the bladder allows excellent cystography, cystourethrography, and voiding urograms. In four cases the urograms were of only fair, though diagnostic, quality. In one of these cases an enlarged abdomen from an intra-abdominal mass partially obscured shadows of the collecting urinary system. In the other three cases in which only fair quality was obtained, and in all those in which the quality was poor, intestinal gas and feces were superimposed over the collecting urinary systems. Qualitatively, the best diagnostic films were those taken 20 and 30 minutes from the beginning of the infusion.

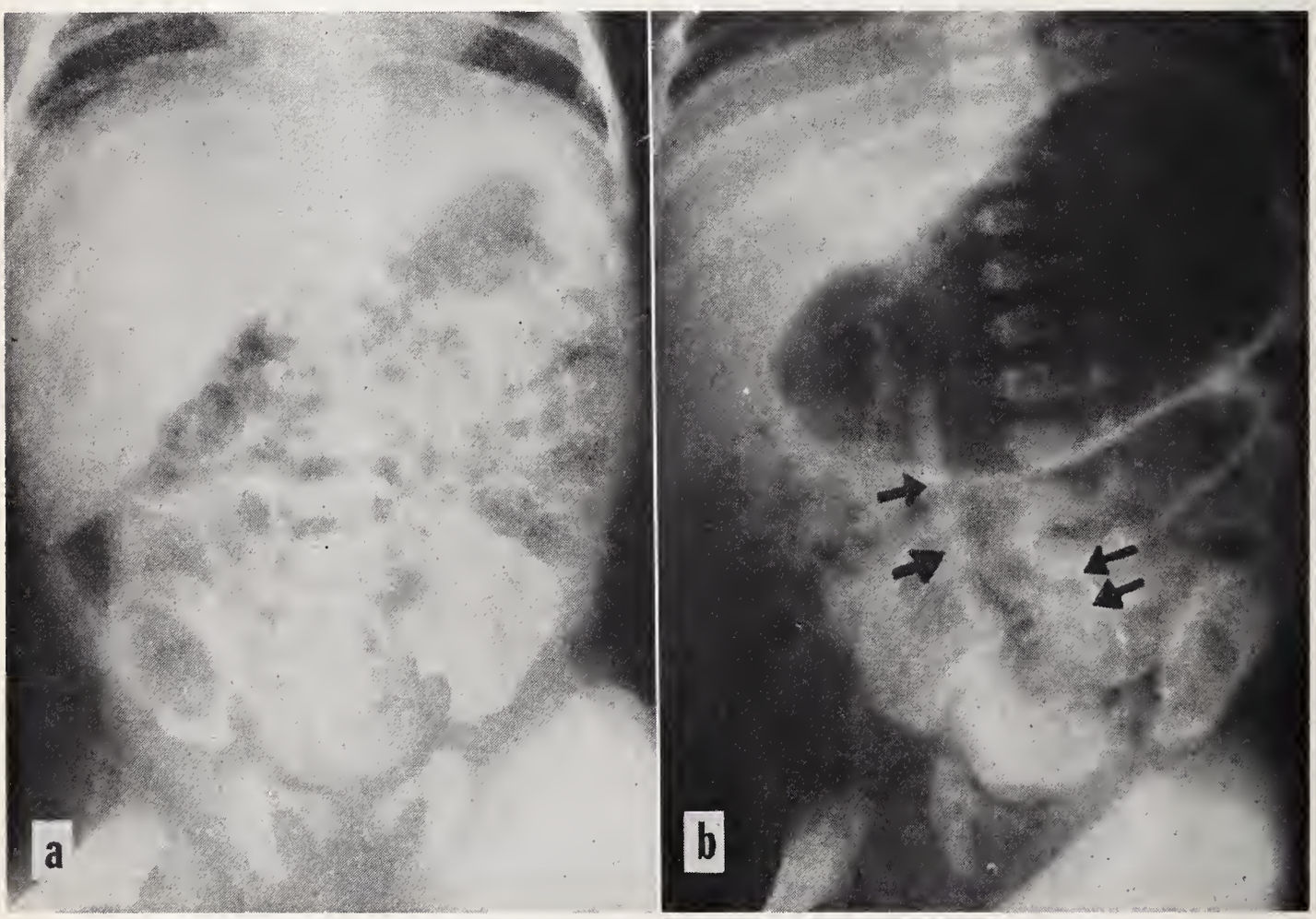

Fig. 1. Clinical diagnosis: suspected mixed-sex. a. Routine intravenous pyelogram shows incomplete filling of the collecting urinary system. This examination was considered to be unsatisfactory and indeterminate.

b. A drip infusion pyelagram performed 10 days after initial intravenous pyelogram demonstrates a normal collecting system on the left side. On the right side the calyces and renal pelvis appear normal, and ureterectasis is seen. The urinary bladder outline is normal. Arrows point to right and left ureters.

TABLE 1.

Symptoms	No. Cases	Ages	M	F	Positive Jet Stream Sign	Contrast Medium	Film Quality: Poor	Fair	Good	Best Films	Reaction	Urogram: Normal	Abnormal
Urinary tract	22	Newborn to 14 yrs.	8	14	10	Maximum 107 cc.	1	1	20	20-30 min.	0	16	6
Miscellaneous	12	Newborn to 13 yrs.	6 (1 mixed-sex)	5	1	Maximum 68 cc.	2	3	7	20-30 min.	0	6	5 (1 "indeterminate")
Totals	34		14 (1 mixed-sex)	19	11	2 cc./kg.	3	4	27	20-30 min.	0	22	11 (1 "indeterminate")

Twenty-two patients had normal urograms, 11 had abnormal pyelographic findings, and in one patient the results were indeterminate. Close clinical follow-up of these cases, retrograde pyelographic examinations, and surgery confirmed the interpretations obtained from the infusion pyelographic examinations.

The ureteral jet stream phenomenon, described as being positive in about 35 percent of all children with urinary tract infection,[9] was positive in 10 of our 22 patients with urinary tract symptoms. Only 1 of 12 patients with other diseases and symptoms had a positive jet stream sign (Table 1).

Illustrative films from satisfactory and diagnostic infusion pyelographic examinations are shown in Figs. 1-4.

Discussion

Routine intravenous pyelography in infants and children is frequently an unsatisfactory diagnostic technique because of inability to prepare the patient properly or because of insufficient filling or concentration of the contrast medium in the collecting urinary systems. Additional technique and procedures, which include prone, upright, oblique and Trendelenburg position and spot films; abdominal compression bands; second dose injections; and the use of carbonated beverages have been introduced to help achieve more optimum results.[4, 5] These techniques and devices enhance the chances of producing better quality urograms, but are time-consuming and require additional radiation ex-

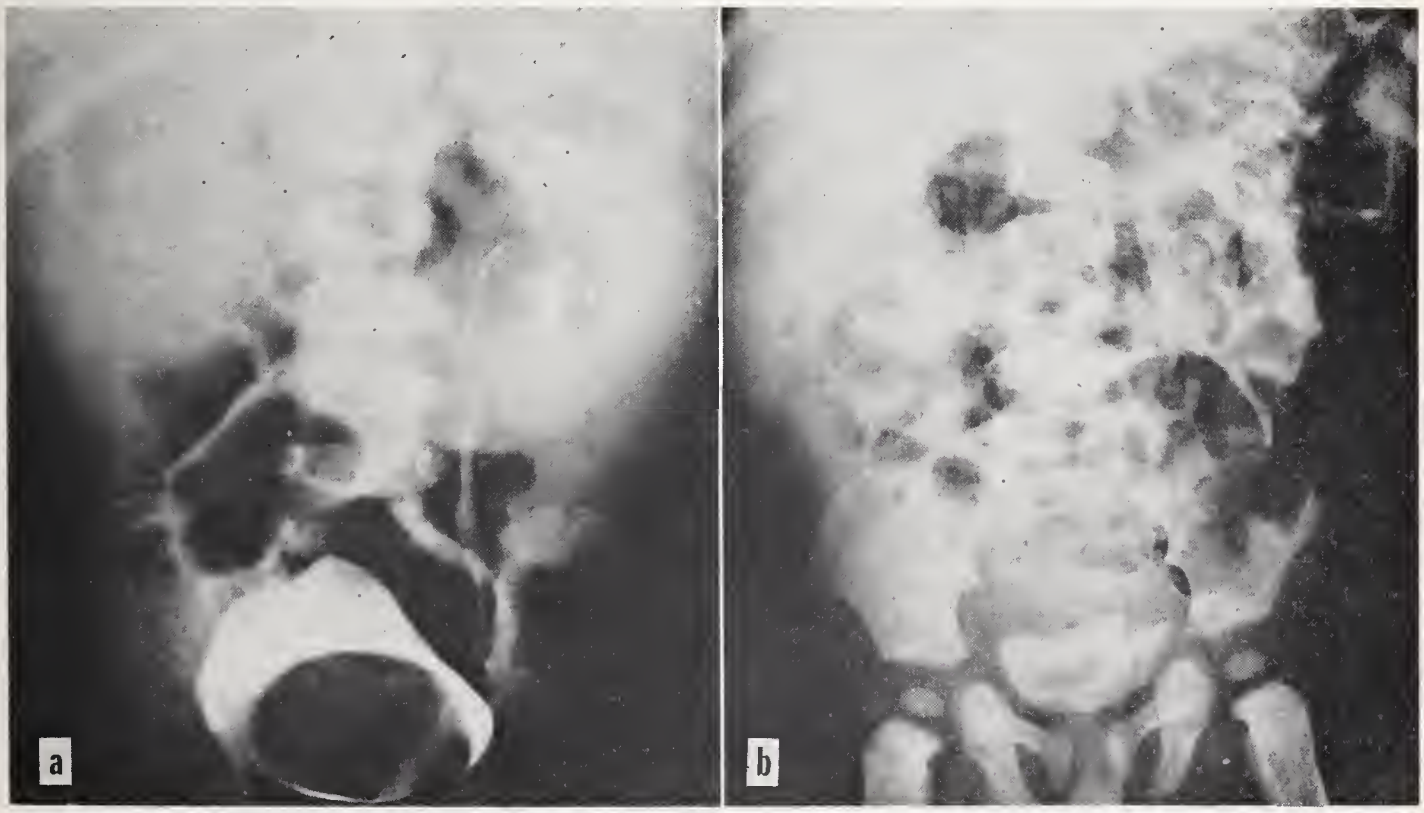

Fig. 2. Clinicol diognosis: possible kidney tumor, right side. a. Infusion pyelogrom shows o normol collecting urinary system in its entirety on the left side, and o positive ureterol jet stream sign. The right kidney is nonfunctioning. The kidney shodow on the right side oppeors enlorged, and the rodiolucency in the urinary blodder represents o ureterocele. At surgery, hydronephrosis on the right side ond o ureterocele were found.

b. Postoperotive routine introvenous pyelogrom poorly demonstrotes the collecting urinory system on the left side.

posure for the patient. In spite of these additional techniques, results in a significant number of examinations are inconclusive. In such cases, we recommend adoption of the drip infusion pyelographic examination, such as is used clinically in adult patients.[5]

Side reactions from large volumes of contrast medium in infants, children and adults have been minimal, and are no greater than those encountered during routine intravenous pyelographic examinations.[1,2,6-8] In our series, there were no complications and no allergic or toxic reactions.

Our patients, examined by the infusion technique, were not dehydrated. We, therefore, depended on a large volume of contrast medium to flood the collecting urinary system, and thus got greater filling of the calyces, renal pelves, ureters and urinary bladder. The large volume of dye increases the density of these structures and allows better and easier identification of the anatomy and pathologic conditions of the urinary tract.

The positive ureteral jet stream phenomenon in 10 of our 22 patients confirms the observation made by Nevin and associates of its presence in children and infants with urinary tract disease.[9] This sign was present in 17 of 67 adult patients having symptoms of urinary tract infection.[10]

The uses and advantages of routine intravenous pyelography and of infusion pyelographic examinations for infants and children may be summarized as follows:

1. Routine intravenous pyelography.

a. This examination should be done to study renal function. Time of appearance and differences in concentration of the contrast medium are noted. Delayed films are taken.

b. It should be used for studies of renal hypertension.

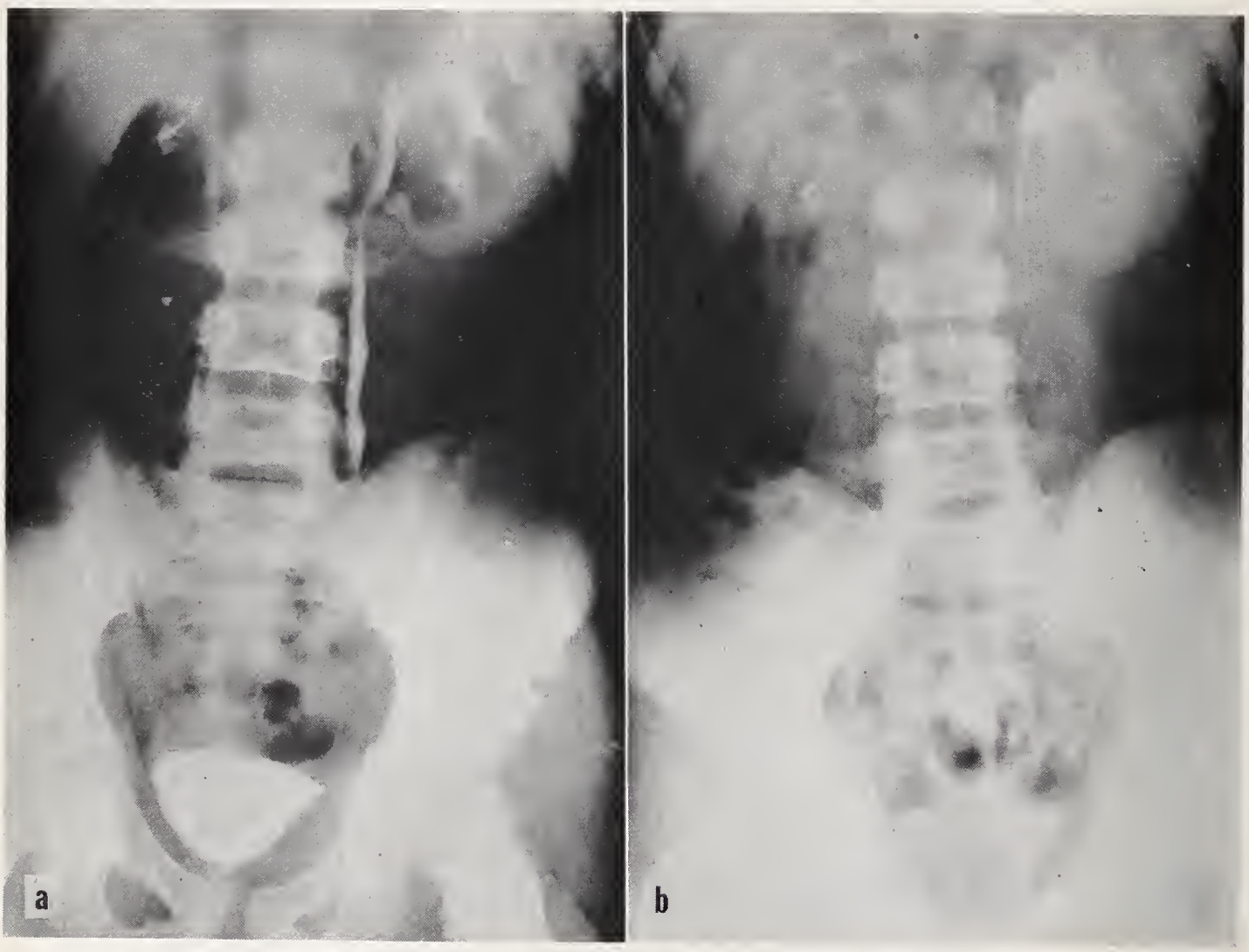

Fig. 3. Clinical diagnosis: possible rupture of kidney as result of automobile accident. a. Film taken 20 minutes after beginning of infusion shows a small, hypoplastic right kidney. There is no extravasation of contrast medium, and visibility of the collecting urinary systems is excellent. Note fracture of the left hemipelvis.

b. Routine 12-minute intravenous pyelogram taken six weeks after trauma shows healing of the fracture, but visibility of the collecting urinary systems is poor as compared with Fig. 3a.

c. It is recommended as a survey examination to detect congenital anomalies, to study the anatomy of the collecting urinary system, and to demonstrate pathologic conditions in the kidneys, ureters or urinary bladder.

2. Infusion pyelography.

a. This examination should follow routine intravenous pyelographic examinations which give inconclusive results.

b. It should be used for better delineation of the anatomy of the collecting urinary system.

c. It should be used to obtain a prolonged nephrogram effect.

d. It should be used when nephrotomography is contemplated.

e. It can be used in cases of renal failure.

f. It is useful and a good substitute for a cystogram, and can be converted into a cystourethrographic voiding examination.

g. Since AP urographic films are of optimum quality at 20 or 30 minutes, radiation exposure can be reduced by taking only one or two films during each examination.

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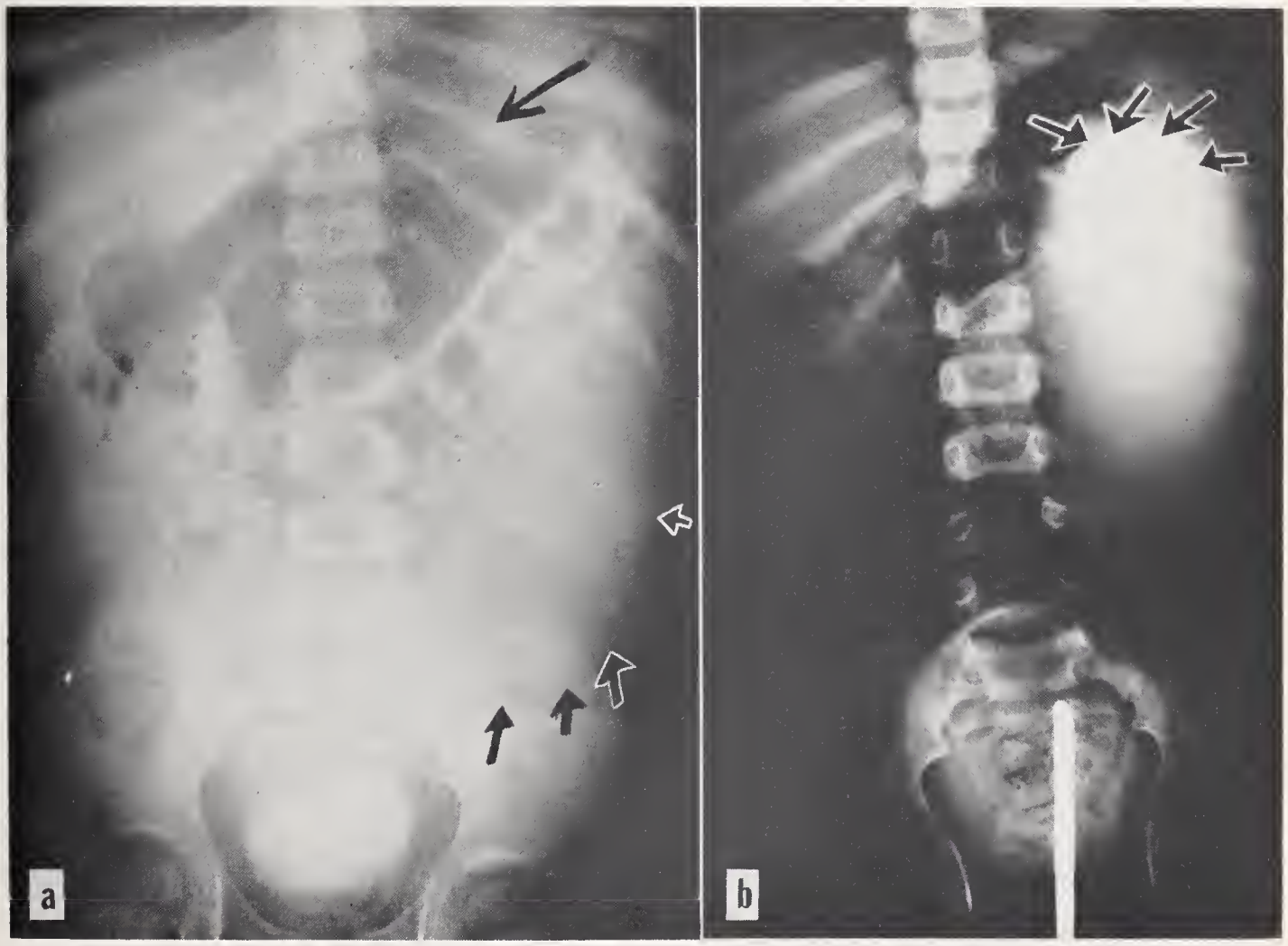

Fig. 4. Clinical diagnosis: abdominal mass, left side. a. 30-minute infusion pyelogram shows normal upper collecting urinary system on the right side and caliectasis in upper collecting system on the left side (arrows). In addition, the left renal shadow is enlarged. These findings on the roentgenogram suggest hydronephrosis on the left side. b. Retrograde pyelogram on left side confirms severe hydronephrosis. Upper arrows point to severe caliectasis.

The Minimally Brain-Damaged Child With Behavioral Problems

REVIEW ARTICLES

CORRECT DIAGNOSIS and effective management of the minimally brain-damaged child challenge the family physician, the pediatrician, and the psychiatrist. Minimal brain damage (MBD) results from impairment of brain tissue from any cause. The chronic brain disorder is virtually irreversible, and although the loss may be almost imperceptible clinically, the chronic brain syndrome leaves some permanent disturbance of memory, judgment, orientation, comprehension and intelligence functioning, motor coordination, and affect, or any combination thereof. Behavior problems are usually secondary to the functional handicap and to the child's social environment.

In this article, the descriptive diagnosis of minimal brain damage is limited to children with evidence of chronic brain impairment, with nondefective intelligence, and without additional major neurologic or systemic disorder. Should mental retardation, convulsive disorder, cerebral palsy, or other systemic disorder be present, the minimal brain dysfunction may accompany the primary disorder and complicate treatment.

Each minimally brain-damaged child has his own cluster of signs and symptoms. Each sign is based on many factors including basic personality matrix and other constitutional factors, stage of development at time of brain injury, type of damage, possible association with organic disease, and the reaction to the resulting handicap by the child, his family, and others. With these variables, a child with chronic brain injury may have any combination or grouping of signs and symptoms, none of which individually indicate brain damage. Many of these signs can be found in transient emotional reactions in otherwise normal children as well as in children with functional and character disturbances.

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Evaluation Procedure

The diagnostic procedure should include obtaining an adequate history, documented school reports, evaluation of the family's stability and mode of interaction, and mental status and neurologic examinations. Psychological testing and an electroencephalogram may be indicated.

A child under five is seldom seen for evaluation. Many symptoms do not become apparent until the child enters kindergarten or elementary school and is placed in a semi-structured situation in open comparison with his peers. The peaks of referrals usually occur when the child is in the third grade where reading skills are emphasized more, and in the seventh grade where the curriculum is less structured than in elementary schools. Frequently, the school initiates the referral process. Commonly, parents report that they are dealing with a psychiatrist only because the teacher thinks their child has a learning problem. Teachers and parents complain also of disobedience in school, generalized irritability, restlessness, easy frustration, silly and irrelevant speech, hyper- or hypoactivity, inability to play with other children, impulsiveness, poor participation in sports, and poor academic performance in relation to capability.

Diagnostic Criteria

Criteria generally used for determination of minimal brain damage include:

1. *Special learning deficit.*—Scholastically poor performance in a specific area such as reading,

Proper diagnosis and treatment of the minimally brain-injured child can result in clinical improvement. Parental understanding is essential although other treatment must be geared for the individual child.

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mathematics, or spelling; difficulty with conceptual thinking and relating to abstractions such as time, size, and direction.

2. *Preceptual-motor deficits.*—Poor ability to print, write, or draw appropriately for his age; possible inability to recognize letter and number reversals.

3. *General coordination deficits.*—Clumsiness; possible varying degrees of difficulty with fine or gross motor coordination or both.

4. *Hyperkinesis.*—Fidgitiness and restlessly driven motor activity; possible uninhibited speech or thought patterns.

5. *Impulsiveness.*—Inability to check impulses, especially in new or unstructured environments; this easily leads to conflicts with demands established by family, school, or society.

6. *Emotional lability.*—Extreme nervousness; quick reaction to minimal stress; reaction often intense but usually brief.

7. *Short attention span and easy distractibility.*—Poor ability to concentrate; pronounced more when child deals with abstract material or when multiple stimuli produce distractions. However, when interest is aroused in a specific subject, there is ability to concentrate.

8. *"Soft" neurological signs.*—Includes transient strabismus or nystagmus, dysdiadochokinesia, poor finger dexterity and coordination, mixed or confused laterality, or both, speech deficit or delay without structural cause, asymmetry of reflexes, disturbances of muscle tone and movement, equivocal pathologic reflexes, positive palmomental reflex, or hippus phenomenon.

9. *Borderline or abnormal electroencephalogram.*—Although "experts" are not in complete accord, most agree that there is a more than chance relationship between borderline or abnormal EEG and the minimally brain-damaged child.

In obtaining the history, special emphasis should be placed on the prenatal, birth, neonatal, and developmental aspects. The effects of rubellae and thalidomide ingestion illustrate the possible intrauterine complications of maternal illness and medications. The trauma of birth, coupled with excessive analgesia and narcosis, may produce hypoxia significant enough to cause structural damage to the still-developing brain tissue.

The developmental history is probably not too reliable, unless there have been compulsive records kept. Significant would be noticeable lags in comparison to siblings or peers, or major regressions of previously developed abilities. Areas to be considered in the social and family history are the parents' and siblings' reactions to the affected child and their modes of expressing their feelings and frustrations.

Discussion with the family or school officials may arouse the physician's suspicion of minimal brain damage; the diagnostic interview then can serve as an arena in which the child is allowed to present his psychopathology. Additional insights into the family's interactions can be gained by observing the child and his parents as a unit while they are in the waiting room and in the office. Their manner may provide clues about their understanding.

Unless there is a high degree of suspicion, the routine neurologic examination usually will not uncover the more subtle signs. Should one sign be discovered, others will usually be detected on careful examination. The neurologic examination should be done in a warm and relaxed manner. In some cases, it may be necessary to structure a performance task in the office to produce frustration. The way in which the child copes with this test can be observed.

The reliability of psychological testing is directly related to the training, experience, and reliability of the clinical psychologist who administers the tests. The minimal test battery should include a complete Wechsler Intelligence Scale for Children (WISC) or equivalent, the Bender Visual Motor Gestalt Test, and a standardized reading test such as Gray's. Often the psychologist's observation of how these tests are executed is significant in establishing demonstrable organic abnor-

mality. Those children who show the most involvement with visual motor incoordination often demonstrate figure rotation, perseveration, fragmentation, or haphazard arrangements of the designs in the Bender test. Although differences between verbal and performance scores on the WISC are important, the full-scale IQ score has little direct relationship with the diagnosis of minimal brain injury. In the WISC test, there are patterns of the specific subtests which do reflect organicity. Although projection tests such as Rorschach and Draw-A-Person help determine personality patterns, they are not sensitive enough to document minimal brain damage. Many routine classroom-administered "psychological tests" measure primarily verbal ability. This function is not overly affected by organic injury.

An electroencephalogram is not a reliable enough index of brain damage or dysfunction to be required for the diagnosis of this syndrome. Children with clinically substantiated brain damage may produce normal tracings, and definitely abnormal tracings have been obtained in persons with no known correlation with abnormal brain function or personality structure. Many parents also view the "brain wave" test as a magical procedure that will offer the complete solution. Availability of EEG facilities and cost are important factors. An EEG should be made whenever possible if there is clinical suspicion of the presence of an epileptogenic process, localized or focal neurologic signs, progressive or regressive changes in behavior previously within normal limits, or a penetrating head injury. Probably the most common EEG findings (except for tracings compatible with an epileptogenic or convulsive disorder) are multiple spikes. Many believe this is a result of scar tissue irritation on normal cerebral tissue. Investigators have shown some relationship between the "14 and 6" tracing phenomena and the impulsive acting-out child.

Case Reports

Case 1.—A nine-year-old boy brought by his parents for evaluation had repeated the first grade and was given social promotions to the second and third grades.

His teacher reported that he was failing all subjects, had no number concepts, and could not spell. He could, however, read. He had few friends and even children who seemed to accept him in the classroom played cruel pranks on him during recess. The teacher could not understand his poor scholastic performance since his score was average on a class achievement test.

There were no significant problems noted during the perinatal period. Growth and development were within normal limits in comparison to his older siblings. His medical history was essentially negative, and there were no known sequelae from the usual childhood illnesses. He had no history of extreme febrile episodes, convulsions, or serious head trauma.

The most remarkable features of his mental status were concreteness in thinking and inability to handle abstract material comfortably.

He had a lateral nystagmus on close accommodation with a pronounced hippus phenomenon and a minor degree of pass-pointing. Intelligence functioning was within the dull normal range, and performance ability was more impaired than the verbal ability. The patient also had noticeable difficulty in performing tasks involving visual motor coordination and in comprehension of reading material.

The mother's major concern was his poor school performance which she blamed on "poor teaching." The father identified with the child in his scholastic problems, but was emotionally detached from him. Both had been permissive and overprotective, and neither could accept the thought that the child had emotional problems. The mother viewed this as a reflection that she was a poor parent. With the father's detachment and the mother's overprotectiveness, the boy had assumed a passive, "anti-anger" attitude toward his classmates. Recognition of his intellectual deficiencies made it easy for him to assume the role of the "goat." His anxiety seemed to be the result of not being able to acceptably express his negative feelings.

The parents were told that their son needed a well-structured home life in which more appropriate expectations would be made consistently for his behavior and performance. They were to emphasize his ability to achieve through short-term goals. Their family physician was to be contacted about medication to relieve anxiety. Remedial reading as well as special classroom assistance was recommended.

Case 2.—A nine-year-old boy had been diagnosed as having minimal brain damage with symptoms of hyperactivity and poor scholastic performance. He was started on medications which the parents later discontinued. They also did not accept counseling offered.

One year later, school officials were still concerned about the child's continued poor school performance and he was again referred for evaluation.

Except for occasional enuresis until age seven, the medical history did not provide possible etiology for his problems. The parents had had some marital difficulties because the boy's maternal grandparents lived with the family. The father was often away from home, and the mother had become overprotective, overindulgent, and inept at setting behavioral limits.

The boy was cooperative during the examination, although his general mannerisms and behavior pattern were immature. His psychomotor activity was increased. He attempted to explore the office and seemed driven to touch each item. His major preoccupation with the "badness" of his brother appeared to be a projection because he used terms his teachers had used in describing him.

When a telephone call interrupted the interview, the child became anxious and wanted to know if his mother was all right. Later, he reported that he loved his mother more than his father because she "is sweet and does things for me." His speech was explosive and lacked modulation. His general level of productivity and ability to cope with test situations gave an impression of low normal intelligence. His memory recall for past events was adequate although questionably impaired for recent recall. His apperception of general material was good; however, he could not handle abstract material comfortably. He had poor judgment and no discernible insight into his problems.

There were no pathologic reflexes and his sensory and motor components were intact. Except for a rotary nystagmus on close accommodation, the cranial nerves were intact and functional. There was postural stooping, a tendency to waddle, clumsiness in executing gross coordinated movements, poor finger dexterity, dysdiadochokinesia, as well as mixed and confused laterality.

Psychological tests were similar to test results obtained two years previously. Although he continued to show evidence of organic malfunction, his intellectual functioning ability was well within the bright normal range. He had considerable difficulty dealing with tasks involving complex hand-eye coordination. His new learning ability was appropriate to his intellectual level although he was definitely limited in ability to deal with abstract concepts. The psychological impression was chronic brain syndrome; he had a full-scale IQ score of 116.

It was thought that he had a chronic brain syndrome of unknown etiology, and that he was emotionally immature. The latter was a secondary result of his mother's overprotection and understimulation, coupled with his father's noninvolvement.

He was started on medications (Suvren, 50 mg. orally three times a day, and 100 mg. every night, and Mellaril, 25 mg. orally three times a day.) He was given supportive and interpretive psychotherapy. He responded by ventilating frustrations and negative feelings which had developed from his feelings of inadequacy and from his dependency situation at home. A psychiatric social worker, using our recommendations, counseled with the parents. We suggested that the father become more active in the day-to-day contact with his son, that he set short-range goals that his son could handle capably. Both parents were advised to increase his responsibility by reasonable and consistent expectations, and by setting up a more structured home milieu. Our treatment plan concerning the boy's scholastic needs was also given to his school.

After several months, the parents had sufficiently mobilized their resources to handle their needs more adequately. The child had shown sufficient improvement to terminate therapy, and was referred back to the family physician for follow-up care and continued medications.

Case 3.—A 17-year-old boy was referred by his family physician, at the parents' request, for reevaluation of continued poor school performance secondary to "brain damage." The major problem reported was excessive talking interspersed with periods of extreme lethargy.

Minimal brain damage had been diagnosed at age four. Repeated examinations and tests during his first two years of school substantiated the diagnosis. Initially, he had symptoms of hyperactivity, short attention span, dyslexia, and poor coordination. Organic malfunction was apparent from the results of psychological testing, and he had an abnormal, although nonspecific, EEG.

At age seven, he was started on dextroamphetamine. At the time of evaluation, he was taking 2 to 4 spansules of 15 mg. each per day.

His hyperactivity was the most impressive feature. His mental status examination was suggestive of a toxic condition. Because realistic appraisal of brain injury could not be made while he was on sufficient dosages of a medication known to have toxic effects with prolonged use, he was hospitalized and the medication slowly withdrawn during the next four days. By the seventh hospital day, none of the symptoms present on admission was evident. The following week, the evaluation was completed. An extensive battery of psychological tests, sensitive for organic dysfunction, indicated normal functioning ability. An EEG with both wake and sleep recordings was also normal. Neither "soft" nor "hard" neurologic signs were found. In repeated interviews with the patient no clinical evidence of toxicity or organic malfunction could be detected. The major findings were extreme immaturity and a developing passive-aggressive personality structure. Additional work with the family disclosed interactions and attitudes regarding the previous diagnosis of minimal brain dysfunction. These were believed directly related to his personality psychopathology.

The parents were informed of the findings, and recommendations were made for long-term family oriented outpatient therapy. Several weeks after the boy was discharged from the hospital, the parents expressed dissatisfaction at our inability to find evidence of their son's brain damage. They had contacted another physician who had reordered dextroamphetamine for "brain damage," presumably based on the history supplied by the parents.

Differential Diagnosis

The diagnosis of minimal brain damage is based on history, neurologic examination, and psychological testing with or without an EEG. Hyperactivity is common in children with anxiety reactions or transient emotional problems. Poor scholastic performance may also be the result of mental retardation, passive-aggressive personality traits, or of social and cultural understimulation. Emotional lability or learning problems in forming meaningful relationships can be traits of the psychotic or autistic child. Adequate, detailed history and qualified psychological testing are often enough for differentiation; however, in many instances, referral to a diagnostic clinic or psychiatrist may be necessary before an absolute diagnosis can be made.

Prognosis

Clinical improvement in the minimally brain-injured child is possible even though the brain cannot regenerate cerebral tissue.

Often, children with a previous well-documented history of minimal brain damage at age seven or eight are free of signs and symptoms when reexamined in their teens. Although the theory that the child will outgrow it comes to mind, improvement is probably related to the mind's ability to successfully compensate for the structural and functional deficit and from the beneficial effects of specialized educational techniques and favorable home environment.

Other Considerations

Even after diagnosis of minimal brain damage, a secondary diagnosis may be necessary to describe the child's reaction to the pressures of his own and others' expectations. Often a child with minimal residual damage has become severely handicapped by infantile behavior, resulting from parental overprotection and emotional suffocation. Conversely, transient psychosis and disturbed behavior have occurred in children with obvious brain damage who have been pressured for unrealistically high scholastic achievement.

After recovering from the initial reaction, the family may create a false environment for the child. In this insecure setting, he is excused from responsibility and is given special consideration in contrast to his siblings and peers. This type of family maladjustment can only produce an emotional cripple, who, by being excluded from life experiences, is inadequate in coping with normal conflicts.

A child with this problem may serve as the unknowing target for the unrealistic desires, needs, and unresolved childhood problems of the parents. In such a family situation, this child might be labeled as "bad" or "uncooperative"; other siblings can take advantage and perpetuate the family myth by being, in comparison, "goody-goody" children. Frequently, the minimally brain-damaged child is forced into the role of being the lowest in the pecking order in family or school.

Management

The treatment program must be tailored to each patient. The more understanding and accepting the parents are, the fewer the secondary symptoms that complicate treatment. The secondary symptoms may require resolution before a treatment plan can be effective. Drug therapy is often useful, especially when anxiety and hyperkinesis are present. Response to drug therapy is an individual matter, and the choice of drugs is based upon personal preference and experience. The amount of faith one has in his prescription is possibly as significant as the pharmacologic effect of the drug. My personal experience has been primarily with Mellaril and Suvren. There are numerous reports indicating improvement after using Librium, Valium, Deaner, Tindal, and dextroamphetamine. Dextroamphetamine appears most effective in children under age ten. It should be used carefully since dependency and secondary mental changes may occur; children showing spike phenomenon on an EEG seem to have less tolerance to dextroamphetamine than do children with normal EEG tracings.

Work with the family is a matter of educating them about realistic expectations of their child's abilities and in methods by which their child can best develop his abilities. The family must set up a well-structured pattern for acceptable behavior and activity which is realistic and which can aid the child in developing a sense of accomplishment and worthwhileness.

Close cooperation is needed with school personnel. Communication with the teacher may aid her in planning a more realistic educational program for the child. Unless he has concurrent mental retardation, he does not qualify for a special education class established for the "educable mentally retarded" child. Likewise, placing him in a segregated slow group in a regular classroom setting emphasizes his "dumbness" rather than allowing for his special dysfunction. Facilities for remedial reading and speech therapy may be needed.

Parents should ask their local school authorities to organize special education classes for the minimally brain-injured child. In cases 1 and 2, the parents were able to encourage their local school board to start such classes, established according to suggestions of the Texas Education Agency.

Summary

The "salvage value" of most children with some degree of brain dysfunction warrants adequate evaluation and counseling with the child and his parents. The goal of the treatment program is to develop, according to individual needs, the best utilization of the child's abilities. Drugs, psychotherapy, coun-

seling with the family, and proper placement in school are used.

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♦ Dr. Munson, 1000 E. 32nd, Austin.

American Academy of Pediatrics Outlines Latest Immunization Procedures

Oral poliovirus vaccine is the most effective immunization procedure for the prevention of polio in children, and should be used in community-wide vaccination programs and for routine immunization in infancy and childhood, reports the American Academy of Pediatrics in its newly-revised 15th edition of the "Report of the Committee on the Control of Infectious Diseases" (The Redbook).

The report points out that inactivated polio vaccine is also effective, but that evaluation reveals a clear-cut superiority of oral polio vaccine because of "ease of administration, immunogenic effectiveness, protective capacity, and potential for the eradication of poliomyelitis."

The Redbook also recommends that:

1. Immunization procedures against diphtheria, pertussis, and tetanus should begin at between 2 and 3 months of age.

2. Three doses of DTP vaccine (diphtheria, tetanus toxoids, and pertussis) should be administered at 4- to 6-week intervals, with simultaneous administration of monovalent or trivalent oral polio vaccine.

3. A fourth dose of DTP and trivalent oral polio vaccine should be given one year later, and another dose of DTP should be administered at 3 years of age. The pertussis vaccine is then eliminated, and tetanus diphtheria boosters are repeated at 6 and 12 years.

4. Children ready to enter school should again receive a single dose of trivalent polio vaccine.

5. Initial vaccination against smallpox should be administered sometime between 15 and 18 months during a relatively cool season, and should be repeated at ages 6 and 12.

6. Children should be tested for tuberculosis at between 9 and 11 months of age, and should be tested annually until age 4, and thereafter every two years. Also, TB tests should be given before the administration of measles vaccine. The report also lists five schedules of measles immunization, emphasizes the greater effectiveness of the live virus vaccines, and recommends that all infants receive measles vaccine "by the end of the first year of life."

The "Redbook" contains a completely rewritten section on the use and abuse of immune serum globulin, an updated section on immunization requirements for foreign travel, a completely new section on parasitic diseases, and a revised and updated section on drug dosages.

Pseudoxanthoma Elasticum

CASE REPORTS

PSEUDOXANTHOMA elasticum usually is not considered a disorder of the pediatric age group. However, visible evidence of this rare systemic disease may appear during childhood and typical skin changes have been noted at birth.[1] A recent review reported that 25 of 200 cases (12.5 percent) were first diagnosed in the 0-10 age group.[2] Since the diagnosis is not commonly established until adult years, it is probable that some patients with early clues are being overlooked.

The following report describes the gross and microscopic changes of pseudoxanthoma elasticum found in an apparently healthy 10-year-old boy. Common systemic manifestations and possible complications of the syndrome are discussed.

Case History

On March 21, 1964, a 10-year-old boy was observed to have visible skin changes suggestive of pseudoxanthoma elasticum. His mother had noticed a peculiar wrinkled appearance of his neck when he was born, but had been advised to ignore it. In 1963, results of studies done elsewhere to rule out xanthoma apparently were within normal limits.

There was no family history of any similar disorder. His growth and development had been normal with no serious illnesses, bleeding episodes, or operations.

Examination on March 21, 1964, revealed no abnormalities except for a yellow linear and reticulated papular eruption involving the lateral neck, shoulders, axillae, and antecubital skin (Fig. 1). A skin biopsy specimen was typical of pseudoxanthoma elasticum (Fig. 2).

The results of an extensive work-up, including complete physical examination, electrocardiogram, x-rays of the skull, chest and extremities, and lipid fractionation were normal. Blood pressure was 110/70 mm. of mercury, and all peripheral pulses were palpable. Repeated ophthalmoscopic examinations failed to reveal angioid streaks.

Comment

The original observers[3, 4] stressed the similarity between gross skin changes in pseudoxanthoma elasticum and true xanthomas. Biopsy and histopathologic analysis of accessible skin lesions revealed abnormal dermal elastic tissue, and for many years pseudoxanthoma elasticum remained classified among the rare dermatoses.

Gronblad[5] and Strandberg[6] in 1929 reported the high incidence of visual disturbances associated with angioid streaks of the retina. They correctly implicated an elastic tissue abnormality as the common pathogenic denominator of the skin and eye involvement. Other sporadic case reports[7, 8] with autopsy findings confirmed the widespread systemic nature of the disorder.

Currently, pseudoxanthoma elasticum is classified among the inherited diseases of connective tissue.[9] Characteristically the

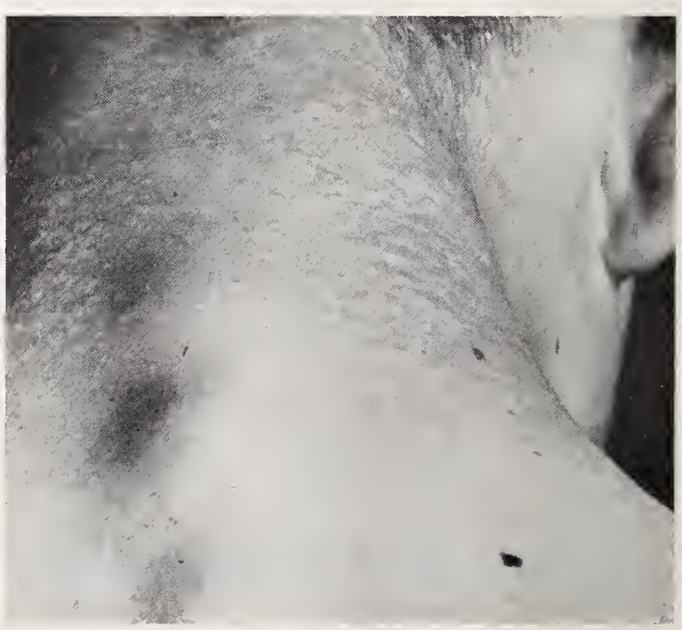

Fig. 1.

A 10-year-old boy has typical skin lesions of pseudoxanthoma elasticum, but no evidence of eye or systemic involvement as yet. Visible alterations of the skin frequently are the earliest evidence of this inherited systemic disorder, and awareness of their significance may aid diagnosis in children.

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W. P. JOHNSON, M.D.

skin, eyes, and cardiovascular system are involved. The most common pattern of inheritance appears to be an autosomal recessive one, although other modes may occur.

Clinical Findings

Skin Manifestations.—Recognition of the visible skin lesions is paramount in the early diagnosis of the disease. Early changes most frequently occur on the neck and axillae and may precede systemic involvement by many years. Other sites of predilection include the antecubital and popliteal spaces, inguinal folds, umbilicus, abdominal wall and buccal mucosa.

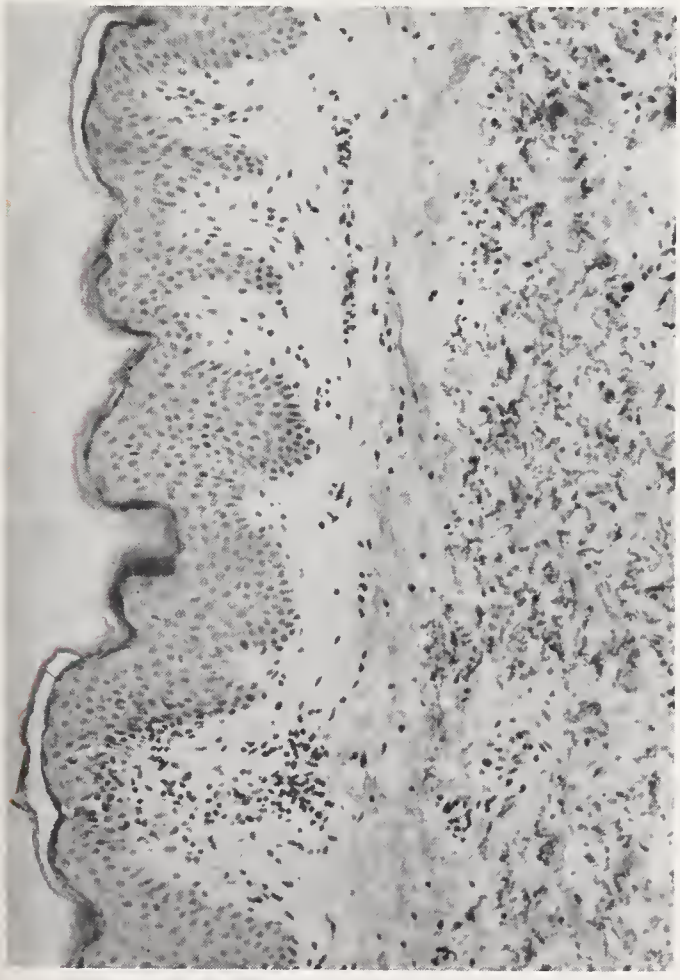

Fig. 2. X 71.5.

The primary lesions are small, soft, ivory-to-yellowish papules often arranged in a linear or reticular pattern resembling a plucked chicken. Later they may coalesce to form plaques or thick, lax folds of skin. The skin lesions may remain stationary or may progress.

Microscopically, swollen, fragmented and clumped fibers with the staining characteristics of elastin are found in the middle and lower dermis.[10] Normally, elastic fibers do not stain well with hematoxylin and eosin, but they stain intensely in pseudoxanthoma elasticum because of an affinity for calcium. This can be demonstrated by specific calcium stains or microincineration techniques.[10] The abnormal elastic fibers have occasionally been demonstrated in grossly uninvolved skin.

Eye Manifestations.—Two-thirds of patients with pseudoxanthoma elasticum are first seen because of visual disturbances. Loss of central vision is the most commonly described symptom and quite often it is preceded by blunt trauma to the eye. The frequency of this symptom in children is not known.

Ophthalmoscopic examination reveals angioid streaks in 85 percent of patients with pseudoxanthoma elasticum. These appear as zig-zag, tapering streaks radiating from the disc. They are wider than normal vessels and do not branch.

It is now thought that angioid streaks represent cracks in an abnormal Bruch's membrane. The outer layer of this structure is derived from the choroid and is composed of dense elastic fibers. In pseudoxanthoma elas-

ticum, normal strength and elasticity is diminished, and force exerted upon this brittle membrane produces fractures clinically manifested as angioid streaks.[1]

Other chorioretinal findings include hemorrhage, scarring and pigmentary abnormalities. Symptoms occur with the onset of macular involvement and may progress to severe impairment. Multiple pregnancies may aggravate visual impairment and progressive deterioration may warrant sterilization.[11]

Cardiovascular Manifestations.—Vascular disturbances are common among older patients with pseudoxanthoma elasticum but are rare in children. Clinical manifestations are secondary to elastic tissue degeneration, calcification, arterial insufficiency, and hemorrhage and may involve cerebral, coronary, visceral, or peripheral vessels. Vessel calcification visible on roentgenograms has been reported as early as age nine.[12] Vascular complications in children have led to intermittent claudication, diminished peripheral pulses, and gastrointestinal bleeding from weakened vessels.

Prognosis and Management

The chief advantage of early diagnosis is in preventive measures and genetic counseling. The aggravation of visual impairment by blunt trauma should preclude participation in vigorous contact sports. Bleeding episodes in these patients should be managed in a conservative manner. Surgical exploration in such situations is seldom warranted. Redundant, unsightly folds of the face and neck can often be corrected by cosmetic surgery.

There is no effective method to treat or retard the premature degenerative vascular changes of pseudoxanthoma elasticum. Symptoms of vascular insufficiency can be treated symptomatically, but in general the disease is slowly progressive and chiefly responsible for premature death.

Genetic counselling depends upon the particular pattern of inheritance.

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Fatal Parachute Fall 1 in 17,000

When an American sports parachutist jumps from a plane, his chances for a fatal accident are about 1 in 17,000, the American Medical Association says. This is a per-participant fatality rate 80 times higher than that of football. Many parachuting deaths are due to inexperience, carelessness, and failure to comply with regulations. Contrary to widespread belief, parachute failure isn't the only major cause of accidents. About one-third of all recent parachute deaths have been due to drowning after accidentally landing in water. Another third occurred when jumpers opened their chutes too late, or simply failed to pull the rip cord.

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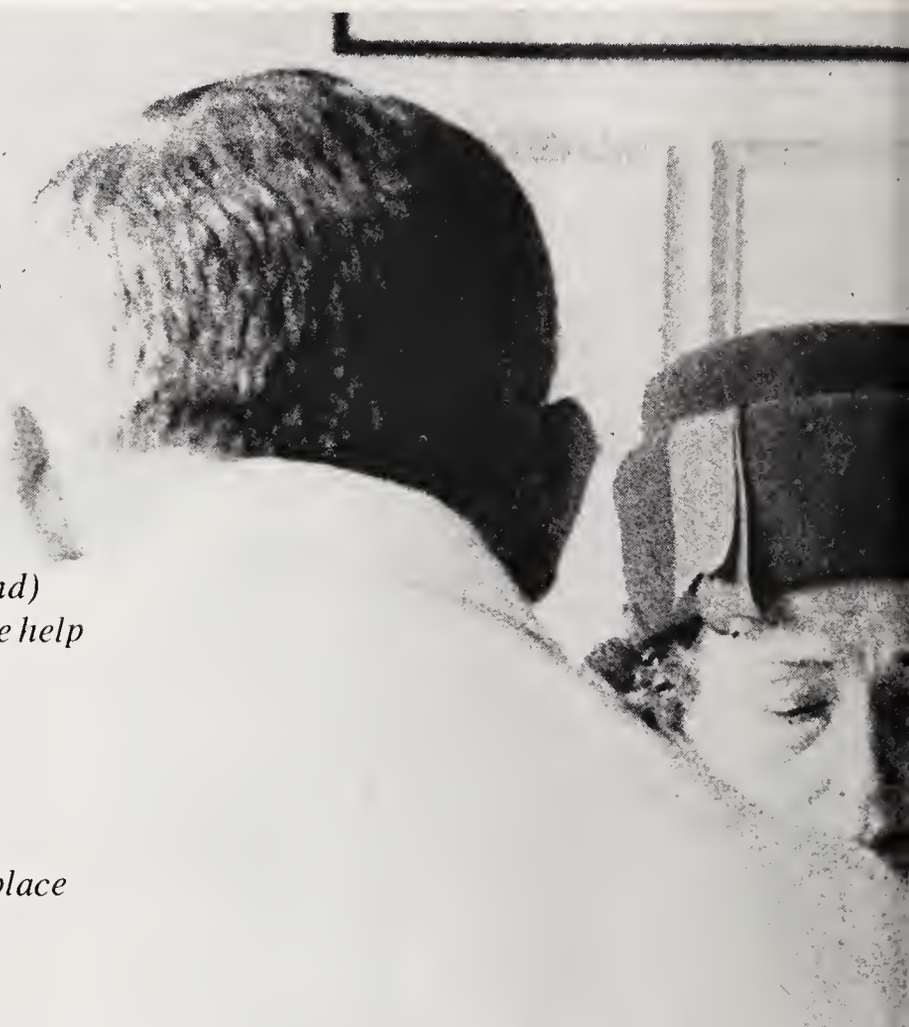

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McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

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Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

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Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

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Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

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Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.

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Clinicopathologic Conference

Don Russell, M.D.; Edward Christensen, M.D.; Norman W. Carter, M.D.;
Eugene P. Frenkel, M.D.; J. Donald Smiley, M.D.; and Bruce D. Fallis, M.D.

DR. DON RUSSELL: This was the only Parkland Memorial Hospital admission for this 17-year-old white boy; he was transferred from another hospital because of renal failure and seizures. The patient was apparently well until Aug. 28, 1965, when he began to complain of malaise, dark urine, and pain in multiple large joints. He was seen by a physician who treated him as an outpatient with tetracycline. During the next two days, nausea, vomiting, and periorbital edema developed in addition to earlier symptoms. At that time (Aug. 30) he was admitted to Midcities Osteopathic Hospital. There was no previous history of dark urine. At age four, he had urinary retention following treatment with sulfa drugs; this was relieved by catheterization and did not recur. The patient had been hard of hearing since early childhood, had a history of chronic otitis media, and wore a hearing aid. There was no family history of deafness or renal disease.

Physical examination at Midcities Hospital showed blood pressure 150/90 mm. of mercury, periorbital edema, and pretibial edema. Urinalysis showed specific gravity, 1.023 with 4+ protein; 100 red blood cells (RBC) per h.p.f., and coarsely granular casts. Blood chemistry values were hemoglobin (Hgb.), 12.5 gm. per 100 ml.; sodium, 134 mEq. per liter; potassium, 6.2 mEq. per liter; chlorides, 100 mEq. per liter; carbon dioxide, 21 mEq. per liter; urea nitrogen (BUN), 104 mg. per 100 ml.; cholesterol, 107 mg. per 100 ml.; total serum protein, 4.4 (1.35/3.05) gm. per 100 ml. Erythrocyte sedimentation rate was 26 mm. per hour. Blood and urine cultures were negative, as were lupus erythematosus preparations. Urine output initially was 600-700 ml. per day but subsequently dropped to about 200 ml. On the second hospital day, the patient spiked a temperature to 101 F. A short systolic murmur was heard, and conjunctival hemorrhages were noted. The diagnosis was subacute bacterial endocarditis, and penicillin, 2 million units per day, was administered. Urine output continued to decrease, and the blood became more azotemic. After peritoneal dialysis on Sept. 3, the patient's BUN level dropped to 65 mg. per 100 ml., and serum potassium to 4.7 mEq. per liter. Dialysis was repeated Sept. 15, and that day the patient's temperature spiked to 103 F. He was treated with 60 million units of penicillin during about 24 hours. At 4 a.m. on Sept. 16 he began to have generalized seizures which were controlled with 600 mg. of short-acting barbiturates. Later that day, he was transferred to Parkland Memorial Hospital.

When admitted, he was comatose. His pulse rate was 120; blood pressure, 150/100 mm. of mercury; respirations, 20; and temperature, 100.2. He had slight periorbital edema, and his pupils were equal and reactive. No conjunctival hemorrhages were seen. His optic fundi were normal. There was exudate in both ear canals, and the tympanic membranes were inflamed. His neck was supple, his chest was clear, and his heart was

This is the second in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School; guest editor for the series is Dr. Bruce D. Fallis, professor of pathology. Other participants in this report are Drs. Don Russell, resident in internal medicine; Edward Christensen, assistant professor of radiology; Norman W. Carter, Eugene P. Frenkel, and J. Donald Smiley, assistant professors of internal medicine.

A 17-year-old boy was hospitalized with acute glomerulonephritis, etiology unknown. Oliguria, progressive azotemia, uremia with gastrointestinal bleeding, *Pseudomonas* septicemia, and pulmonary edema were present; the patient died after two months.

not enlarged. A short, early systolic murmur was present along the left sternal border. His abdomen and genitalia were normal. A stool test for occult blood was positive. There was 2+ pedal edema. Neurological examination revealed generalized hyperreflexia with bilateral Babinski response. Laboratory values obtained were: RBC, 12.1; WBC, 12,000 with a left shift. Urine was loaded with RBC's with 4+ protein and many coarse granular casts. BUN, 96 mg. per 100 ml.; electrolytes, normal; total serum protein, 6 (1.9/4.1) gm. per 100 ml.; calcium, 7.4 mg. per 100 ml.; phosphorus, 5.5 mg. per 100 ml.; and cholesterol 246 mg. per 100 ml.

The patient was alert, oriented, and had no residual neurological symptoms on the second hospital day. He had no further seizures. A diagnosis of lupus nephritis was made on the basis of positive lupus erythematosus preparations on Sept. 22. At that time, treatment with prednisolone (100 mg. per day) began, but the patient did not improve. 6-Mercaptopurine, 100 mg. daily, was added to his medications on Oct. 8 and was continued throughout his hospitalization. His hospital course was marked by continued oliguria, progressive azotemia, uremia with gastrointestinal bleeding, and terminally by *Pseudomonas* septicemia and pulmonary edema. Peritoneal dialysis was performed twice, and hemodialysis four times. A permanent shunt for hemodialysis was attempted twice but had to be removed because of recurrent clotting. Blood and urine cultures after the last hemodialysis on Oct. 19 grew *Pseudomonas aeruginosa*. This was treated with colistin. On Oct. 26 pulmonary edema developed, and the patient was digitalized. Peritoneal dialysis was started, but the patient died after two dialysis runs.

Radiologic Findings

DR. CHRISTENSEN: There are several chest x-rays. On admission the cardiac outline was normal and remained so until Oct. 25, at which time the heart became grossly enlarged, the pulmonary vasculature congested, and the patient was in obvious cardiac failure. On Oct. 6 he had small bilateral pleural effusions, but the pulmonary vasculature was essentially normal then. Terminally, he had an infiltrate in the left lung base, and probably had pneumonia in the left lower lobe.

Films of the abdomen on admission revealed distended loops of large and small bowel, which radiographically had the appearance of an adynamic ileus. On Oct. 19, after the pleural effusions became evident, he had a diffuse haze of increased density over the whole abdomen, probably the result of ascites. This obscured the liver and kidney shadows, which we did not see on any of our films.

X-rays of the skull and cervical spine showed fusion of the second and third cervical vertebrae, and the odontoid process was anomalously developed and peculiarly shaped. On the right side, the first and second ribs were fused.

Clinical Discussion

DR. NORMAN CARTER: This patient was first hospitalized with acute glomerulonephritis. This is a condition characterized by the development of edema, a congestive state usually associated with hypertension, usually some degree of azotemia and probably always a decrease in glomerular filtration rate, and proteinuria and urinary sediment containing red cell casts. Although many conditions give rise to red cell casts, large numbers of easily identified ones are seen in acute glomerulonephritis.

The most common cause of acute glomerulonephritis is a poststreptococcal immune state. On the other hand, probably all the collagen diseases can cause acute glomerulonephritis, but the most frequent association is with systemic lupus erythematosus. Acute glomerulonephritis can also develop in patients with hereditary nephritis. It also can be an exacerbation of chronic glomerulonephritis. Finally, it can be the onset of chronic glomerulonephritis.

In this patient, the original findings were

TABLE 1. *Results of Laboratory Studies.*

	Entered Parkland														
	8/30	9/4*	9/15	9/16*	9/19	9/21*	9/24	9/28*	10/1†	10/5†	10/7	10/9†	10/19	10/20†	10/25
Hemoglobin	12.5	13.4	12.4	12.1		8.8	10.1	8.7	8.0	7.9	8.0		8.7		5.0
WBC	6,400		17,000	12,000											
Stool guaiac				2+		4+	4+	4+	Neg.	1+			4+	4+	4+
Urine:															
Specific gravity	1.023			1.025						1.020					
Protein	4+			4+						4+					
RBC	100 +			20						25					
WBC	5			50						Loaded					
RBC casts	None			Present											
24-hr. protein	320 mg.				400 mg.										
Creatinine clearance	6 cc./min.				8 cc./min.										
Urine culture	Neg.			Neg.		*Pseudomonas*				Neg.			*Pseudomonas*		
VDRL	Neg.			Neg.											
Fasting blood sugar	96			166											
BUN	104	65	91	96	144	81	120	53	71	62	105	57	145	68	152
Creatinine				7.4	7.4	7.2	9.5		6.2	9.6	8.6	4.8	6.7		6.8
Sodium	134	137	137	140		141									
Potassium	6.2	4.7	6.7	5.3	4.2	4.7	6.4	4.6	5.2	4.8	4.8	3.9	5.1	4.1	
Chlorides	100	93	98	94		97									
Carbon dioxide	21	24		24		28	28		25		25	28	26	23	19
Total serum protein	1.35/3.05			1.9/4.1											
Cholesterol	107			246											
Calcium				7.4	8.3										
Phosphorus				5.5	10.4										
SGOT				60											
LE preparation	Neg.	Neg.				Pos.	Pos.		Neg.						
Antinuclear						1+									
Latex fixation						Neg.									
ASO titer					50 U										
Blood culture	Neg.	Neg.		Neg.					Neg.	Neg.		Neg.	*Pseudomonas*		*Pseudomonas*
Urine output	700 cc.	(100-300)		(0 - 350 cc.	→									)	800 cc.?
Blood transfusion						1 Unit P.C.									1 Unit P.C.

* After peritoneal dialysis.

† After hemodialysis.

consistent with those of acute glomerulonephritis. The problem, then, is to identify the etiology. First, was there an underlying chronic renal disease? In this patient, the question of hereditary nephritis arises, because otitis media and nerve deafness since early childhood are frequently associated with this condition. Ultimately, the diagnosis of hereditary nephritis was dropped, because no other family members had any form of renal disease. There was no previous history of renal disease in this patient; apparently this was the onset of his illness.

The arthritis involving multiple large joints is an important point in the onset of his illness, since it suggests the diagnosis of collagen disease; systemic lupus erythematosus would be the first choice. Apparently this was considered originally, and his serum was checked for the LE phenomenon. Other things can help differentiate between simple poststreptococcal acute glomerulonephritis and acute glomerulonephritis associated either with chronic glomerulonephritis or collagen disease. First, patients with simple poststreptococcal nephritis initially tend to have normal serum albumin concentration, normal or high urine specific gravity (a high urine osmolality), and normal or even a high normal phenolsulfonphthalein excretion. In an acute exacerbation of chronic renal disease, chronic urinary protein loss and impairment of renal function cause hypoalbuminemia, low urine specific gravity, and impaired phenolsulfonphthalein excretion. In this patient, on the first determination, serum albumin concentration was low, and the globulin level was somewhat high. However, I would caution that when albumin concentration decreases, the chemical fractionation of albumin and globulin is tenuous, and electrophoresis is required for proper evaluation of plasma proteins.

In this patient, oliguria and azotemia ultimately required dialysis. This in itself removes the probability of simple poststreptococcal acute glomerulonephritis. Profound oliguria for any length of time, or azotemia requiring dialysis, is extremely rare in poststreptococcal nephritis.

Ultimately the patient had generalized seizures which were probably the result of hypertension and hypertensive encephalopathy. However, it is interesting to note that 60 million units of penicillin were given to him when he was essentially anuric; under these conditions, convulsions have been reported.[1]

At admission at this hospital, the laboratory findings were unchanged, which indicates that this disease began as an acute glomerulonephritis but has some manifestations of a more severe disease than the nephritogenic type of nephritis. At the time of admission, the specific gravity was 1.025; that is spuriously high as the result of measuring specific gravity with a refractometer in the presence of proteinuria. Corrected values for true urine osmolality were much closer to the serum osmolality. In addition to the numerous casts including red cell casts in the urine, there were lipoid bodies, a finding more consistent with chronic than with acute disease.

We have been impressed by a group of patients whose onset of illness resembles acute glomerulonephritis but is accompanied by significant azotemia, oliguria and in one instance anuria, low serum albumin suggesting a massive loss of protein early in the course of the disease, and a urine sediment of the so-called telescoped type, that is, consisting of all the inflammatory cells usually found in acute nephritis but also containing casts consistent with chronic disease, i.e., waxy casts, granular casts, and lipoid bodies in addition to red blood cell casts.[2, 3] These patients, as a group, do poorly clinically.[4] We have a small series of patients whose histories and laboratory findings are similar to this patient's. In all of the patients, the course has been progressively downhill, and steroid therapy has not been of any benefit. All these patients died within two years, and several of them died after a brief period. This clinical picture has been previously described as subacute glomerulonephritis, but it is not a good term because it tends to make one think of a direct relationship to acute glomerulonephritis, and this implies that it might be a poststreptococcal state. In rare instances, it might be, but more frequently it is part of a more chronic disease such as a collagen disease or hereditary nephritis. The renal morphology in these cases is quite similar, and likewise has been called a subacute glomerular lesion. I think that this, also, and for the same reasons, is not a good term. These cases do fall into a group, and should be separated from chronic glomerulonephritis that is characterized by a long latent period or a long period of nephrosis, and acute glomerulonephritis with complete recovery. In other words, this is a severe disease with a steady downhill course and a relatively short duration.

When the lupus erythematosus preparations were positive, prednisolone therapy was begun, but there was no clinical or laboratory improvement. It was, therefore, suggested that we might supplement steroid therapy with immunosuppression with 6-mercaptopurine, and, therefore, attempts were made to dialyze the patient for a period of time, at least long enough to expect a response from 6-mercaptopurine. The dose of 6-mercaptopurine was relatively small and usually has not been associated with profound toxic manifestations. We expected that it would take at least ten days before any therapeutic response would be seen. After this patient had received the drug for some ten days, we agreed that there had been, again, no clinical or laboratory response. Our final diagnosis was rapidly progressive glomerulonephritis due to systemic lupus erythematosus.

DR. EUGENE FRENKEL: I agree with the diagnosis of lupus nephritis. The LE test was strongly positive. Since the initial studies of Schwartz and Dameshek,[5] immunosuppression has been used for a variety of clinical circumstances. Some of the problems in this case are: (1) What is the metabolic fate of 6-mercaptopurine? (2) Is 6-mercaptopurine the drug of choice? (3) What is the temporal relationship of the anticipated response?

First, a word about 6-mercaptopurine metabolism: the major route of excretion is through the kidney, and the half-life of the drug after one dose is about five hours. In the presence of normal renal glomerular filtration, 50 percent of it is identified in the urine within five hours, 15 percent as thiouric acid and the remainder as inorganic sulfate. Beyond the first five hours, small quantities are identifiable in the urine as inorganic sulfate, but one can never account, via renal excretion, for all the 6-mercaptopurine. Because renal excretion is the major route of elimination, one may question the use of the drug in this anuric patient. Few data are available in such clinical circumstances, although studies are presently under way in this institution.

Second, a more current problem related to the use of 6-mercaptopurine in diseases such as this is the comment of Aisenberg[6] relative to species specificity. Though 6-mercaptopurine is an effective immunosuppressive agent in rabbits, it may not be the best agent in man. A derivative of 6-mercaptopurine, azathiopurine (Imuran), produces a greater degree of immunosuppression in man than comparable doses of 6-mercaptopurine. By contrast, this derivative is a poor agent in the rabbit, so there seems to be some species specificity. Thus, for immunosuppression in these patients, it is reasonable that Imuran be the drug of choice.

With regard to the third question, how long should such a patient be dialyzed, or how long should one hope for a response, I don't know of any good specific human data. We do have data in another form of immunosuppression, where a circulating anticoagulant to antihemophiliac globulin had developed in three patients. After 6-mercaptopurine was utilized in these patients, no change in the titer of the anticoagulant antibody was noted until at least two weeks of therapy. It was beyond the 15th day that the first drops in titer occurred, and the first decrease in bleeding tendency occurred after the third week.[7] Perhaps it takes somewhat longer than the time that we followed this patient.

Finally, the dose should be considered. Shearn[8] used 3 mg. per kg. of body weight per day of 6-mercaptopurine in some steroid-resistant nephrotic syndromes, and noted response in two lupus erythematosus patients. Toxic effects were frequent, with leukopenia in most cases and severe thrombocytopenia in half. Considering the dose we use to treat acute leukemia, 2.5 mg. per kg., Shearn's doses are large, and it is not surprising that he encountered clinical problems. For immunosuppression, we have used 1.5 to 2 mg. per kg., and we have not had significant difficulty with thrombocytopenia and leukopenia. Whether this represents inadequate immunosuppression, we cannot state. As we mentioned, in our studies of the circulating anticoagulant where a "marker" existed in terms of the titer of that anticoagulant, this dose range was adequate and safe.

DR. J. DONALD SMILEY: The presence of pain in the large joints at the onset of the disease, fever, hyperglobulinemia, and positive LE preparations offer substantial support for the diagnosis of systemic lupus erythematosus with rapidly advancing nephritis. I shall discuss two aspects of this problem: first, the use of 6-mercaptopurine therapy in such patients; and second, some recent studies done in mice that relate to the etiology of systemic lupus erythematosus.

When does one use steroids in treating lupus and when 6-mercaptopurine? Several

groups[9, 10] and also the Rheumatic Diseases Unit here, have shown that systemic lupus erythematosus should be treated with adequate steroids to suppress as much activity of lupus nephritis as possible. Steroid use becomes a problem, however, in a patient who has azotemia and severe hypertension. Steroids raise the patient's blood pressure even further, so that occasionally the nephritis is made worse and even hypertensive encephalopathy occurs. In one of our patients, with severe azotemia and what appeared to be a terminal episode, 6-mercaptopurine (1.5 mg. per kg.) was begun as a desperation move. That was three years ago. The patient showed a dramatic clinical response; her BUN came down and she continued in remission as long as 50 mg. per day of 6-mercaptopurine was maintained. During one interval of withdrawal of the drug, she again relapsed with increased evidence of renal insufficiency. We have since seen several other systemic lupus erythematosus patients who showed improvement on 6-mercaptopurine.

In our case today, we might ask whether or not we waited too long before introducing the 6-mercaptopurine. The disease appeared about Aug. 28. It was not until Oct. 8 that 6-mercaptopurine was begun. Perhaps the delay in therapy could account for lack of response. This is not to say, however, that every patient with lupus erythematosus who has advanced renal disease will show response. Some, treated to leukopenic levels with high doses of 6-mercaptopurine, have had unremitting renal disease and death.

In the renal lesion in systemic lupus erythematosus one can stain both gamma globulin and complement in the glomeruli, suggesting an immunologic etiology for the glomerulonephritis. Some recent studies in mice offer interesting possibilities for the etiology and pathogenesis of systemic lupus erythematosus. A similar disease has been described[11] in New Zealand black mice with identical renal lesions, positive antinuclear factors in the serum, positive LE preparations, hypergammaglobulinemia, and hemolytic anemia. In these animals, disease develops at about nine months of age, prior to which they appear normal. One cannot transfer the disease directly to other animals, but the F_1-hybrids with other mouse strains often show even more severe disease, so that it appears related to some intrauterine or genetic factor.

Another condition in mice that has been more carefully studied and is more fully understood[12] is the so-called "late disease" of lymphocytic choriomeningitis (LCM). If LCM virus is given to mice in utero or during the first three days of life, the mice become infected but do not develop any obvious disease. They appear normal, gain weight, and reproduce. At about 14 months of age, however, they show onset of hemolytic anemia, thrombocytopenia, hepatosplenomegaly, hyperglobulinemia and glomerulonephritis. They do not have arthritis, LE cells, or antinuclear factor, but rare glomeruli show wire loops resembling systemic lupus erythematosus nephritis. A mouse carrying the LCM virus, apparently immunologically tolerant to it, has no antibodies to LCM and a high titer of virus in the blood, and will eventually develop "late disease." If such a mouse is joined by parabiosis to an immune mouse that has recovered from LCM, then the immunologically tolerant mouse immediately develops "late disease," presumably initiated by onset of immunity against the virus, but with many of the features of an autoimmune state. It is interesting to consider the autoimmune features of systemic lupus erythematosus in comparison with these two similar mouse diseases, and to wonder if systemic lupus erythematosus also could represent a break in immunological tolerance to a carrier virus state.

Postmortem Findings

DR. BRUCE FALLIS: The patient was a somewhat small, although by no means dwarfed, young man with the typical moon facies of Cushing's syndrome, secondary to the steroid therapy. There were multiple venepunctures and many petechiae of the skin. In addition, there were the wounds produced by the peritoneal dialyses; associated with these were numerous fibrous peritoneal adhesions, but there was no acute peritonitis. There were 800 ml. of clear serous fluid in each pleural cavity, and both lungs showed considerable compression atelectasis. There were 50 ml. of serous fluid in the pericardial sac and approximately 300 ml. of ascitic fluid. The left kidney weighed only 10 gm. and consisted of an irregular cluster of roughly spherical cysts with thin, translucent walls. No normal renal parenchyma was present on the left side. The upper third of the left ureter was a fibrous cord with no lumen. The lower

part of the left ureter was patent. There was a small but patent artery running to the left kidney. Microscopic study of the left kidney revealed multiple cysts lined by low cuboidal epithelium, small clusters of poorly formed tubules, and a loose, interstitial tissue containing fat cells, a few lymphocytes, and many small blood vessels. No glomeruli were found. We interpreted this as a unilateral multicystic kidney, or cystic dysgenesis of the kidney. This is a congenital renal disorder thought to result from improper development of the ureteric bud. In multicystic kidney disease, the ureter on the involved side is absent or atretic, and the kidney is represented only by a cluster of cysts without recognizable renal parenchyma. Multicystic kidney disease is unilateral and not hereditary and carries a good prognosis, unlike polycystic kidney disease which is bilateral, hereditary, and eventually lethal.

With the exception of the multicystic left kidney, an incidental autopsy finding not important in the patient's death, the autopsy findings were those of systemic lupus erythematosus. The major changes involved the right kidney, heart, and spleen.

The right kidney was large and weighed 360 gm. as the result of compensatory hypertrophy in response to the congenitally small left kidney. Its capsule stripped with ease. The shape of the right kidney was normal, and its external surface was smooth and mottled pinkish-gray with a few petechiae. There were large foci of severe acute pyelonephritis, characterized by small, yellowish foci of necrosis surrounded by zones of fresh hemorrhage. On the cut surface of the kidney, the architecture was intact, the pyramids were somewhat congested, and the cortical tissue was grayish-yellow. The histologic changes were those of severe proliferative subacute glomerulonephritis (Fig. 1). All the glomeruli were severely involved; they were hypercellular and more distinctly lobulated than normal. Prominent fibroepithelial crescents were present, and the capillary tufts were shrunken and appeared ischemic with inapparent lumens. Periodic acid-Schiff stains revealed thickening and duplication of basement membrane material. The interstitial tissue showed edema and slight fibrosis and contained a slight infiltrate of lymphocytes. There was moderate atrophy of the tubular epithelium, and many epithelial cells contained prominent hyaline droplets. The small arteries and arterioles were normal. The more characteristic features of lupus nephritis, that is, variability of involvement of glomeruli, the focal fibrinoid necrosis of the tufts, and hematoxylin bodies, were not present, and the renal lesions in this case could not be differentiated from ordinary subacute glomerulonephritis.

In addition to the glomerulonephritis, there were large areas of acute necrotizing pyelonephritis (Fig. 2). These changes were predominantly in the renal pyramids, although they did extend in some areas into the cortex, and we viewed them as an ascending pyelonephritis. There were smudgy basophilic areas of necrosis in the renal medulla, surrounded by edema and a relatively scant cellular infiltrate. The paucity of the inflammatory reaction is a reflection of leukopenia, steroid therapy, and the caus-

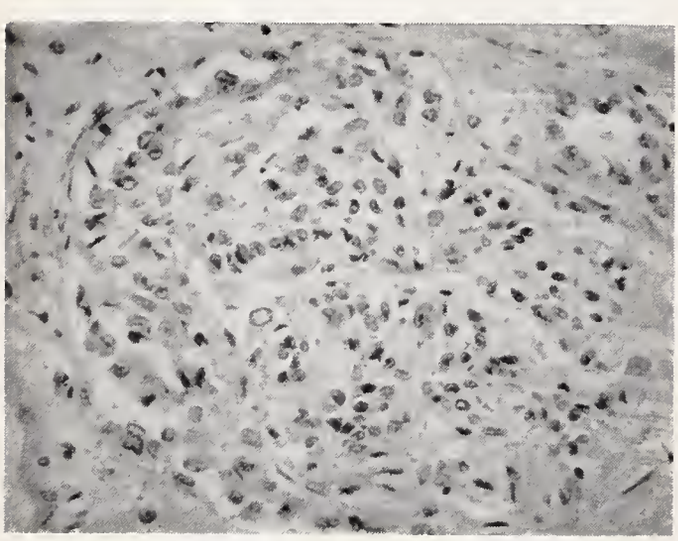

Fig. 1. Kidney. One hypercellular glomerulus is shown. The capillaries are inapparent, and many synechiae bind the glomerular tuft to Bowman's capsule. X 344.

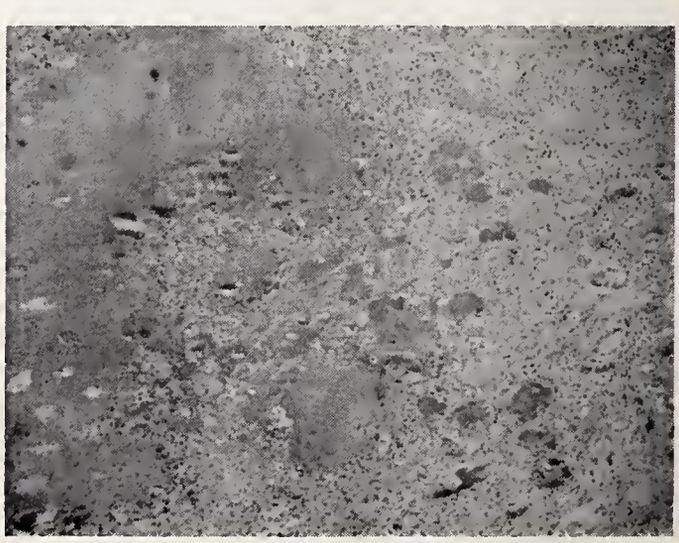

Fig. 2. Kidney. Note the smudgy, necrotic tissue (left) in contrast to the normal medullary tissue (right). Relatively few inflammatory cells are present. X 80.

ative organism, *Pseudomonas aeruginosa*. In the urinary bladder there was bullous and hemorrhagic cystitis. The prostate contained irregular yellowish-gray foci of necrosis. Microscopic study revealed the same type of necrotizing inflammation in the prostate that was present in the kidney. The lower urinary tract infection, caused by *Pseudomonas aeruginosa*, presumably began as a complication of the patient's indwelling catheter, and was followed by ascending infection of the right kidney.

The heart weighed 330 gm. and was somewhat dilated but not hypertrophied. The only significant lesion was a grayish-yellow vegetation on the line of closure of the aortic valve. The other valves were unremarkable. Microscopically, the valve was thick and fibrotic, and the vegetation consisted of irregular foci of smudgy, fibrinoid necrosis, granulation tissue, and mononuclear inflammatory cells. These changes were present on both the ventricular surface of the aortic valve and deep in the sinus of Valsalva on the reverse side. The distribution on both surfaces of the valve is common with the endocarditis of systemic lupus erythematosus (atypical verrucous endocarditis of Libman and Sacks) but is rarely seen with degenerative verrucous endocardiosis, bacterial endocarditis, and rheumatic fever.

The spleen weighed 190 gm. and was grossly unremarkable. Microscopic examination showed concentric periarteriolar fibrosis, involving mainly the central arterioles of the white pulp (Fig. 3). This onionskin periarteriolar fibrosis is characteristic of systemic lupus erythematosus. The lymph nodes were slightly enlarged throughout the body. The lymphoid tissue, as the result of steroid therapy, was somewhat hypocellular. The sinusoids, however, showed a nonspecific proliferation of large reticuloendothelial cells. The bone marrow was quite hypocellular with dilated sinusoids and a few clusters of hematopoietic cells. The hypocellularity of the marrow is the result of the 6-mercaptopurine therapy; there was moderately severe but probably reversible damage of the bone marrow. The thymus showed slight hyperplasia of epithelial reticulum cells and a relative reduction of lymphocytes. The changes were slight and probably nonspecific. The thymic changes described by Hutchins and Harvey[18] in cases of systemic lupus erythematosus were not present.

The lungs weighed 1,430 gm. and showed severe edema. On microscopic examination there were poorly demarcated foci characterized by smudgy interstitial necrosis, deposition of fibrin in the alveoli, and a scant cellular infiltrate. Some of the alveolar epithelial cells in these foci were large, plump, and partially detached; their nuclei were enlarged and contained a central purple inclusion body with the chromatin marginated along the darkly staining nuclear membrane (Fig. 4). This change indicates cytomegalic virus inclusion infection of the lungs. This is a rare disease in adults and was presumably caused in this case by the debilitating primary disease, the immunosuppressive therapy, and the steroid therapy. This viral infection was terminal and probably not of great consequence. Other lesions discovered at autopsy included acute pseudomembranous esophagitis due to *Pseudomonas aerugi-*

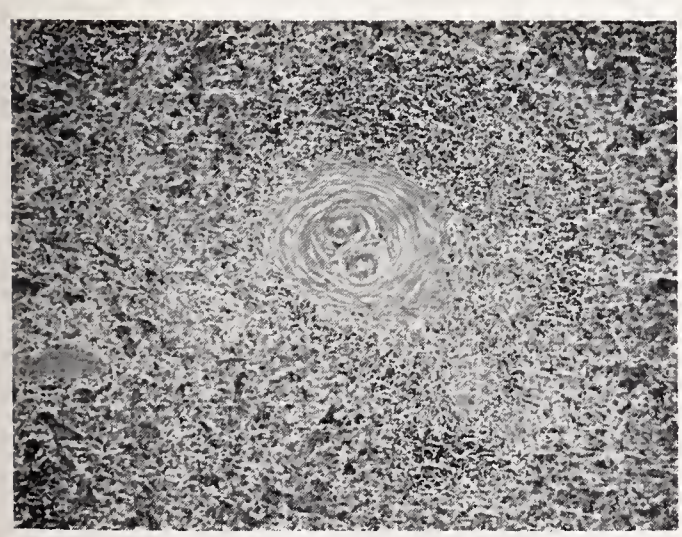

Fig. 3. Spleen. In the center of the field are two small central arteriales with cancentric periarterialar fibrasis. X 80.

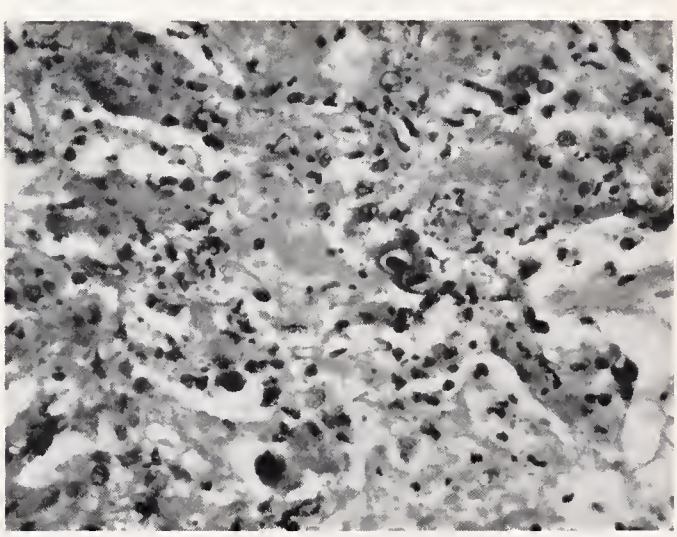

Fig. 4. Lung. Nate the twa large, dark intranuclear inclusians (center). The alvealar septa are smudgy and necratic with many fragmented nuclei. X 344.

nosa, cloudy swelling of the liver, and testicular atrophy.

In summary, the postmortem diagnosis was systemic lupus erythematosus, the major lesions being subacute glomerulonephritis, periarteriolar splenic fibrosis, and organizing verrucous endocarditis of the aortic valve. Effects of treatment included iatrogenic Cushing's syndrome and hypoplastic bone marrow with leukopenia. Death resulted from a combination of *Pseudomonas* infection of the urinary tract and esophagus, and focal interstitial pneumonitis due to cytomegalic virus.

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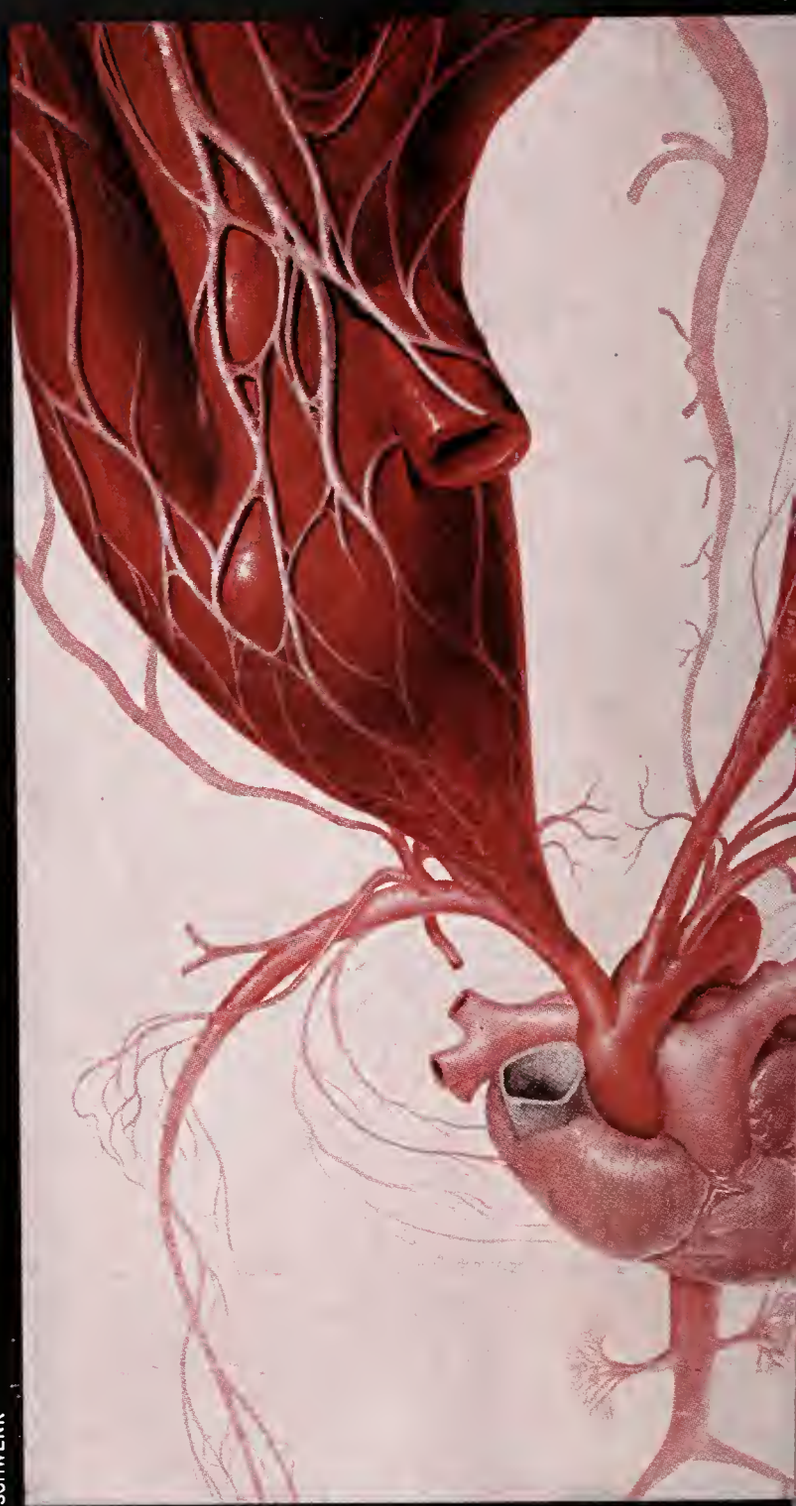

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Ralph Schutzman

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2. Are your assistants doing some of the book work in the evenings and on the weekends? More often than not, a pegboard system will enable them to finish today's work today.

3. At the end of the month, is there a bottleneck in getting out the statements? It is possible to have the statements ready to mail on the closing day of the month.

4. How are the cash collections (payments today for services performed today)? You do not need a pegboard to increase your cash collections, but the system operates in such a way that cash collections generally increase. If you collect the full amount for services on the day they are performed, fewer month-end statements need to be mailed.

5. When the patient wants to pay, does your office assistant always know the charge without having to ask you? There should be a smooth flow of this information from doctor to assistant.

6. Is your assistant or bookkeeper worried because she is responsible for all the cash, and because you have not established controls whereby she can prove that she is handling it correctly? Are you concerned about the cash? You need tight control, and your bookkeeper deserves protection.

7. Are you sure that all charges are accounted for? You may find that your pres-

ent system could be improved to guard against lost or forgotten charges.

8. Do you know whether or not your patients like your bookkeeping procedures? A pegboard system reduces "doctor-bill shock," and promotes a feeling of general efficiency. Most patients like it.

Other Considerations

Assuming that one or more of these points has been concerning you, what other factors should be considered? Your situation is individual, and your office procedures should be designed by an expert who will study your needs and devise an efficient, individualized system for you.

In an individual practice, the specialty normally is the greatest determining factor in office procedure. An anesthesiologist may find a "back office" operation to be quite efficient, but a general practitioner probably needs a "front office" system. Physical layout of the office is not too important.

In a partnership practice, specialty is likewise a factor, but the details of the partnership must also be considered. Is the partnership set up on a flat percentage of receipts and expenses, or is it based on the amount of charges and receipts created as a result of each doctor's work? If more than one doctor sees the patient, who receives the first payment, and how is this easily determined? Here, the physical layout of the office could be important.

In a group practice or large clinic, the physical layout of the office is extremely important, and all of the variations involved must be carefully considered. In each situation, a pegboard system can be designed to provide a fast routine with a great deal of control.

Methods

With any of the following procedures, you will have correct month-to-date and year-to-date totals of your production and of your cash.

In a "front office" operation, all bookkeeping is done at the front desk. The forms and the pegboard are set up before the day begins, and Fig. 1 shows how a patient is registered by writing his name on the transaction slip. At the same time, the name is transferred to the day sheet. At this point, it is also possible to insert the patient's balance onto the transaction slip; some doctors like to know this when they are with the patient. Transaction slips are glued together lightly so that banks of 25 can be placed on the board at one time. After the patient's name is entered, the transaction slip is lifted off the pegboard and is used as a charge ticket; it is included with the medical history card or envelope which goes to the doctor.

If standard fees are established, the doctor checks the service he has performed; if standard fees have not been set up, or if the service is an unusual one for which the assistant would not know the charge, the doctor writes the amount of the fee on the transaction slip (Fig. 2).

How does the charge ticket get back to the front office? You or your nurse could take it if one of you customarily ushers the patient to the front. Usually, however, the doctor simply gives the charge ticket to the patient and asks him to give it to the office assistant. While the patient was with the doctor, his financial history and month-end statement were selected; now they are placed on the proper line on the day sheet, the charge ticket is placed over the statement, and the charges are written on it at the top (Fig. 3). At this point, the assistant tells the patient what the charge is. She may or may not ask if he would like to pay now, depending on the doctor's preference.

Fig. 3 shows a payment of the previous balance. As soon as the amount received is written on the transaction slip, the slip becomes a receipt for the patient. Entering the new balance transforms the transaction slip into an immediate statement. Except for writing the day and hour of the next appointment (which would also make the transaction slip an appointment slip) and entering the amount of the check or cash on the deposit slip, all of the writing in the bookkeeping procedure is complete.

The patient's financial history has all the figures that were written on the transaction slip—so does the month-end statement, which can be used as a follow-up, since the patient has already received one statement. All of this information is also on the day sheet. To prove the figures on the day sheet, run adding machine tapes of the different columns; this automatically proves the figures on all other records. (A fringe benefit of carbonless paper is that it does not smudge your assistant's neat dress or white uniform.)

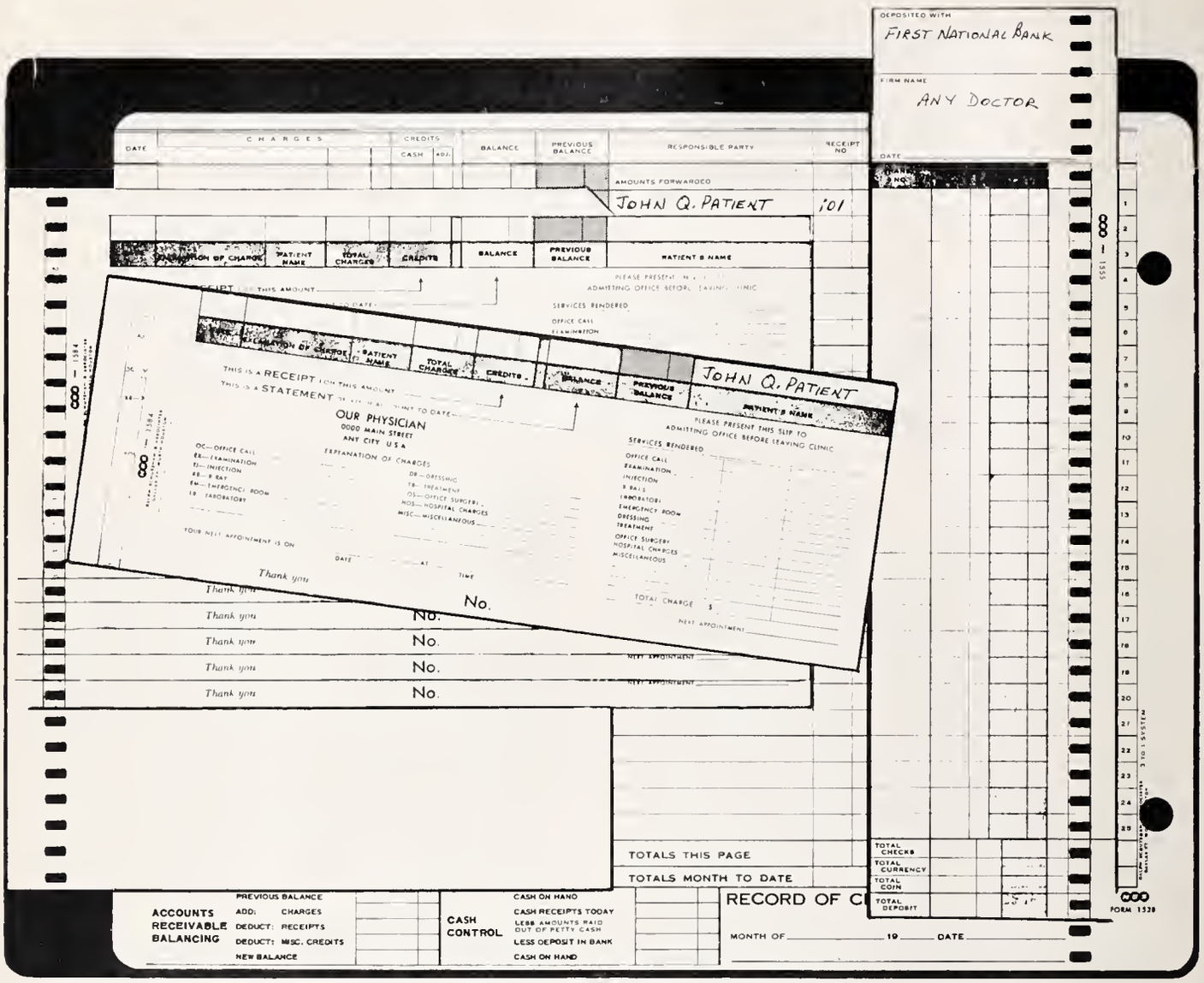

Fig. 1.

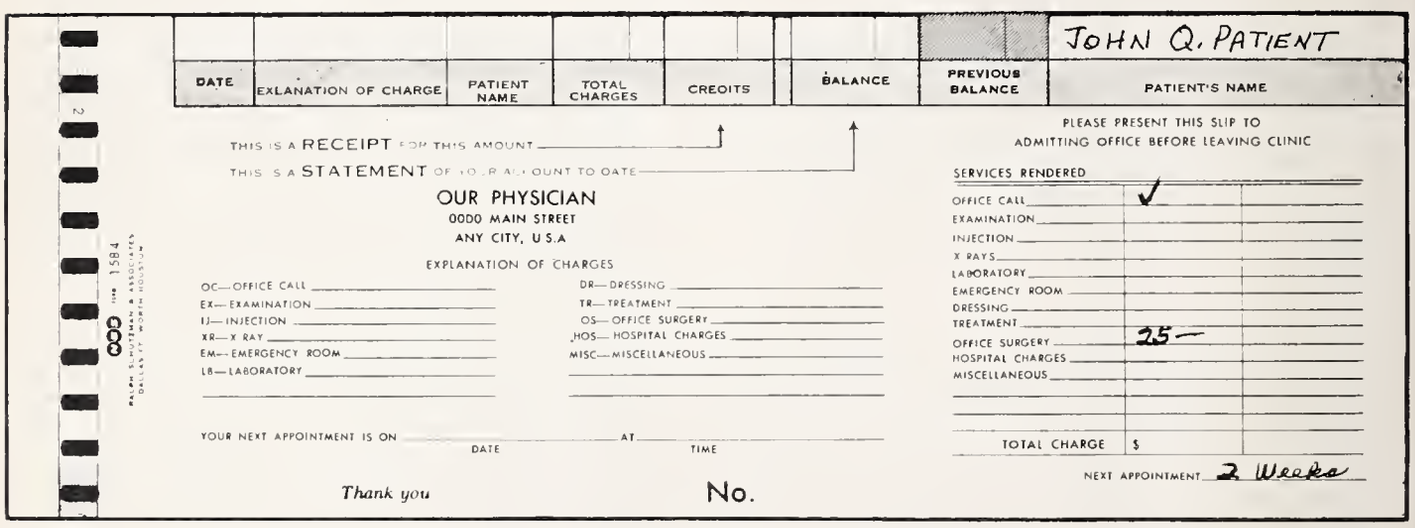

Fig. 2.

In a back office procedure, no pegboard transaction slip is used. Charges and credits go to the bookkeeper in the usual way. Instead of posting charges and credits only on the patient's financial history, the assistant writes them simultaneously on the month-end statement, financial history, and the day sheet. Proving the day sheet automatically proves the figures on the other records. At the end of the month, the statements are ready to be mailed.

A hand addressor takes less time than

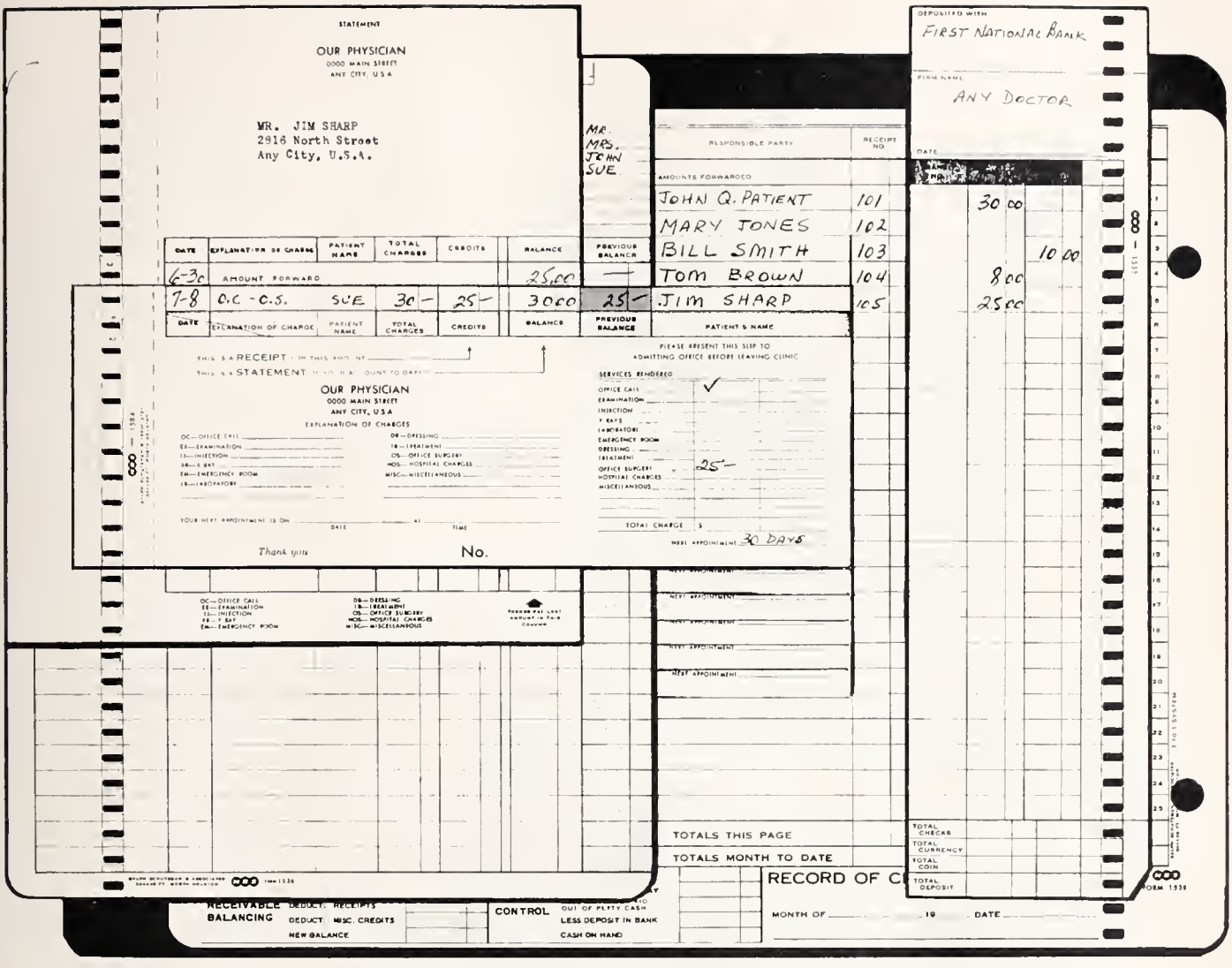

Fig. 3.

typing, and can be used to put the patient's name and address on the statements.

Variations

Variations of these procedures depend on the situations, and on the likes and dislikes of the doctor and his staff.

The financial history can be printed to look like a statement, so that at the end of the month a facsimile can be sent to the patient. Another variation utilizes perforated transaction slips; one portion serves as a charge ticket, receipt, appointment slip and statement, and the other as the month-end statement.

Electronic data processing systems are used by many physicians. Their output is fantastic in quantity and speed; the quality of output, however, depends on the quality of input. The pegboard system is being used to raise the quality of computer data. If it is used as a complete front office procedure, the patient's financial history remains in the doctor's office. In some procedures, only a transaction slip and day sheet are used. In either case, the accuracy of the information can be verified before it is put into the computer and used to prepare patients' statements. From this same information, electronic data processing equipment can provide a breakdown of fees by type of service, and analyze the age of past-due accounts.

There is also a pegboard system that simplifies writing statements for past-due accounts, and also analyzes the age of these accounts, and provides a systematic collection procedure.

One more bookkeeping chore—keeping track of expenses—can be simplified by the technique of collating forms so that as each check is written, all necessary bookkeeping for disbursements is simultaneously completed. Deductible items are clearly indicated for income tax purposes.

Costs

The pegboard itself is inexpensive; most

boards cost $12 or less. Although it must be emphasized that the efficiency of any system depends on the quality of personnel who use it and on the quality of their supervision, the pegboard system is simple and requires no special training for an assistant.

In most cases, standard forms make up the pegboard system. Special forms for special situations are available at a slightly higher cost than standard. Manufacturers specializing in pegboard systems charge nothing for making a survey of present office procedures and making recommendations, nor do they charge for training the personnel in the use of a pegboard system.

Periodic Checkup

If you are as good a businessman as you are a professional man, you will have your accountant check your books periodically, regardless of what procedures you use or how smoothly they operate. Your accountant can spot any new developments, keep your general ledger, provide a "case history" which will be valuable if any crisis occurs, and offer sound advice pertinent to the business side of your practice.

♦ Mr. Schutzman, Ralph Schutzman & Associates, 2816 Hall Street, Dallas 75204.

Flu and the Power of Positive Resistance

Some of us may have mental resistance which helps fight off the flu after infection, while others become ill because they are psychologically vulnerable.

This is indicated by research reported the *Archives of Internal Medicine.*

There is even a suggestion—although the statistical evidence is not strong—that mental make-up helps prevent the infection itself.

When physicians tested 480 men employees at the Army Biological Laboratories, Fort Detrick, Md., they found 96 who could be classed as "psychologically vulnerable" to illness, and another 78 who were termed "intermediates."

The remaining 306 were "nonvulnerable," a term indicating a relationship between their psychological health, as determined by testing, and their speed of convalescence from previous illnesses.

When an outbreak of Asian influenza struck the installation, the "vulnerable" men reported illness almost two and one-half times as frequently as the nonvulnerable.

In both groups, only one in every six men infected by flu virus actually became ill. There was a slightly larger percentage of infection, however, among the vulnerable.

The differences between these infection rates were not statistically significant, but they are "suggestive" of an increased rate of infection in psychologically vulnerable persons, the authors said.

Flu vaccinations proved ineffective in this instance, and did not affect results of the survey. Infection rates were the same among vaccinated and unvaccinated men.

The authors point out that the men at Fort Detrick were particularly suited for this type of study. They were of approximately the same age, and had a high level of education.

brings peace to the hyperactive colon

CANTIL®
(mepenzolate bromide)

helps restore normal motility and tone

"In 40 of 44 cases of irritable or spastic colon, Cantil [mepenzolate bromide] or Cantil with Phenobarbital reduced or abolished abdominal pain, diarrhea and distention and promoted restoration of normal bowel function . . . Cantil [mepenzolate bromide] proved to be singularly free of anticholinergic side-effects . . . Urinary retention, noted in two cases was eliminated in one by reducing dosage."[1]

IN BRIEF: One or two tablets three times a day and one or two at bedtime usually provide prompt relief. Cantil with Phenobarbital may be prescribed if sedation is required.

Dryness of the mouth or blurring of vision may occur but it is usually mild and transitory. Urinary retention is rare. Caution should be observed in prostatic hypertrophy—withhold in glaucoma. Cantil with Phenobarbital is contraindicated in patients sensitive to phenobarbital.

Supplied: CANTIL (mepenzolate bromide)—25 mg. per scored tablet. Bottles of 100 and 250. CANTIL with PHENOBARBITAL—containing in each scored tablet 16 mg. phenobarbital (warning: may be habit forming) and 25 mg. mepenzolate bromide. Bottles of 100 and 250.

1. Riese, J. A.: Amer. J. Gastroent. 28:541 (Nov.) 1957

LAKESIDE LABORATORIES, INC. Milwaukee, Wisconsin 53201

LAKESIDE

PRODUCTS
FOR PATIENTS
YOU SEE
EVERY DAY

DACTILASE®

Each tablet contains:
Dactil® (piperidolate hydrochloride), 50 mg.;
Standardized cellulolytic* enzyme, 2 mg.;
Standardized amylolytic enzyme, 15 mg.;
Standardized proteolytic enzyme, 10 mg.;
Pancreatin 3X** (source of lipolytic activity), 100 mg.; Taurocholic acid, 15 mg.

*Need in human nutrition not established.
**As acid resistant granules equivalent in activity to 300 mg. Pancreatin N.F.

In chronic or acute indigestion, fluttery, gassy stomachs obtain prompt, gratifying relief through the antispasmodic, surface anesthetic and enzymatic activity of Dactilase. Dactilase decreases hypermotility and pain and reduces the production of gas. Dactilase does not induce stasis, but helps restore normal tone. It has little or no effect on enzyme secretions, but *adds* enzymes, thus contributing to the digestive efficiency of the patient.

Side Effects and Contraindications: Dactilase is almost entirely free of side effects. However, it should be withheld in glaucoma and in jaundice due to complete biliary obstruction.

Administration and Dosage: One tablet with, or immediately following, each meal. Tablets should be swallowed whole.

Supplied: Bottles of 60 and 250.

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For cold hands and feet, nothing beats hot stoves—but they *are* awkward to carry around. Now Gerilid, in good-tasting take-along chewable tablets can provide rapid vasodilation of peripheral circulation, bringing real warmth to the extremities and decreasing sensitivity to sudden temperature change. Patients *like* Gerilid and *know* they are getting relief.

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Each chewable tablet contains: nicotinic acid (niacin) 75 mg. and aminoacetic acid (glycine) 750 mg.

Administration and Dosage: One or two chewable tablets 3 times a day before meals. If flushing is objectionable, dosage may be lowered. However, tolerance to flushing usually develops without loss of efficacy in regard to vasodilation. The recommended dosage should not be exceeded.

Side effects: Occasional lightheadedness or transient itching which may disappear with continued use. There are no known contraindications; however, caution is advised when there is a concomitant administration of a coronary vasodilator.

Supplied: Packages of 50 chewable tablets.

Also available in liquid form as Geriliquid®, in bottles of 8 and 16 ounces.

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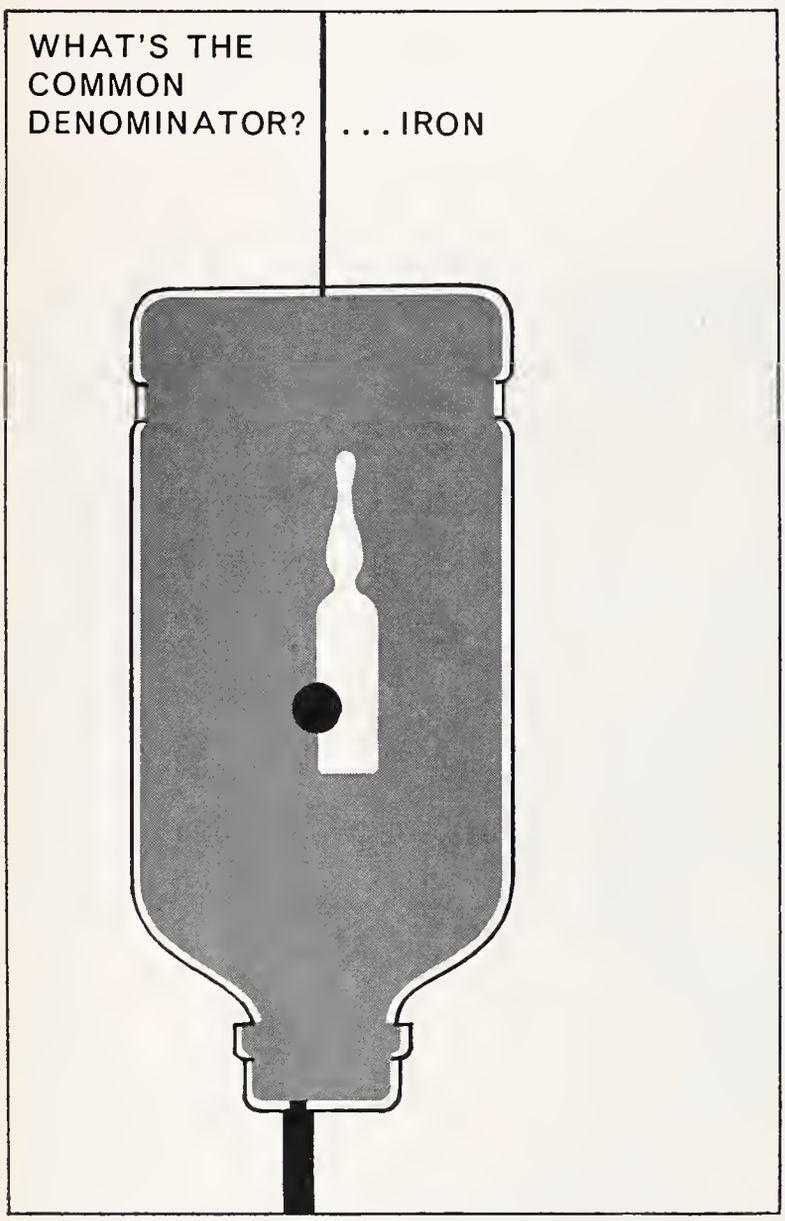
In fact, there's as much iron...250 mg. ...in a 5 cc. ampul of Imferon (iron dextran injection) as in a pint of whole blood. When iron deficient patients are intolerant of oral iron...or orally administered iron proves ineffective or impractical...or if the patient cannot be relied upon to take oral iron as prescribed, Imferon (iron dextran injection) dependably increases hemoglobin and rapidly replenishes iron reserves.



IMFERON®
(iron dextran injection)

IN BRIEF: ACTION AND USES: A single dose of Imferon (iron dextran injection) will measurably begin to raise hemoglobin and a complete course of therapy will effectively rebuild iron reserves. The drug is indicated only for specifically-diagnosed cases of iron deficiency anemia and then only when oral administration of iron is ineffective or impractical. Such iron deficiency may include: patients in the last trimester of pregnancy; patients with gastrointestinal disease or those recovering from gastrointestinal surgery; patients with chronic bleeding with continual and extensive iron losses not rapidly replenishable with oral iron; patients intolerant of blood transfusion as a source of iron; infants with hypochromic anemia; patients who cannot be relied upon to take oral iron.

COMPOSITION: Imferon (iron dextran injection) is a well-tolerated solution of iron dextran complex providing an equivalent of 50 mg. in each cc. The solution contains 0.9% sodium chloride and has a pH of 5.2-6.0. The 10 cc. vial contains 0.5% phenol as a preservative.

ADMINISTRATION AND DOSAGE: Dosage, based upon body weight and Gm. Hb/100 cc. of blood, ranges from 0.5 cc. in infants to 5.0 cc. in adults, daily, every other day, or weekly. Initial test doses are advisable. The total iron requirement for the individual patient is readily obtainable from the dosage chart in the package insert. Deep intramuscular injection in the upper outer quadrant of the buttock, using a Z-track technique, (with displacement of the skin laterally prior to injection), insures absorption and will help avoid staining of the skin. A 2-inch needle is recommended for the adult of average size.

SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylatoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

CONTRAINDICATIONS: Imferon (iron dextran injection) is contraindicated in patients sensitive to iron dextran complex. Since its use is intended for the treatment of iron deficiency anemia only it is contraindicated in other anemias.

CARCINOGENICITY POTENTIAL: Using relatively massive doses, Imferon (iron dextran injection) has been shown to produce sarcoma in rats, mice and rabbits and possibly in hamsters, but not in guinea pigs. The risk of carcinogenesis, if any in man, following recommended therapy with Imferon (iron dextran injection) appears to be extremely small.

SUPPLIED: 2 cc. ampuls, boxes of 10; 5 cc. ampuls, boxes of 4; 10 cc. multiple dose vials.

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NEWS

AMA Unanimously Names Dr. Rouse To Serve as 1966-67 President-Elect

Dr. Rouse is also a contributor + avid supporter of H.L. Hunt's Reflections Foundation wh. is conveniently forgotten in this editorial

DR. MILFORD O. ROUSE of Dallas was unanimously chosen president-elect of the American Medical Association during its 1966 Annual Convention, June 26-30. The election took place Wednesday, June 29.

DR. ROUSE

Dr. Rouse, a gastroenterologist and clinical professor at the University of Texas Southwestern Medical School, has made significant contributions to activities of the AMA, Texas Medical Association, and Southern Medical Association, as well as to several other medical organizations. He has received many honors and awards both within and without medical circles.

Dr. Rouse has been for three years speaker of the AMA House of Delegates and was previously vice-speaker of the House for four years. He has served as chairman of the Texas delegation to the AMA and chairman of the AMA Advisory Committee on Medicine and Religion. He is an ex officio member of the AMA Board of Trustees.

Positions held by Dr. Rouse in TMA include president, 1956, and chairman of the Council on Medical Education and Hospitals, 1948-54. He received the Distinguished Service Award in 1964.

In the Southern Medical Association Dr. Rouse has been president, 1959-60, chairman of the Section on Gastroenterology, 1952, and recipient of the Distinguished Service Award, 1965.

Dr. Rouse will be installed as AMA president in June, 1967, at the Association's Annual Convention.

AMA Acts On Principle Of Individual Responsibility

Texas delegates contributed significantly to the adoption of a statement of policy on the concept of individual responsibility by the American Medical Association at its 1966 Annual Convention.

The action by the AMA's top policy-making body represented an endorsement of the principles of individual responsibility as expressed in a resolution presented by the Texas delegates. The resolution pointed out that the medical profession must fulfill its responsibility to maintain the quality of care. Texas delegates emphasized that the obligation is best served by a personal relationship in which the physician is responsible directly to the patient for care and for medical services rendered. The patient, in turn, assumes as his personal obligation the responsibility for paying the physician for professional services rendered.

The Texas resolution stated that the doctrine of individual responsibility is fully compatible with the American way of life, and the traditional approach to financing the purchase of goods and services. It stressed that the principles of individual responsibility are voluntary.

The Nebraska, Oklahoma, and South Carolina delegations joined in presenting similar resolutions on the principles of individual responsibility. The House of Delegates subsequently adopted a report of that doctrine prepared by the AMA Council on Medical Service.

In its report, the AMA Council expressed its belief "that the doctrine of individual responsibility is a clear and straightforward assertion of the basis of free-enterprise private medicine wherein the responsibility of the physician to provide medical service to the patient is matched by the responsibility of the patient to compensate the physician for that service." The Council declared that direct financial dealings with any patient, whenever possible, are ethical. The Council did not declare that it is inappropriate or unethical to accept compensation for service under other circumstances which the physician feels to be equitable to the patient and himself.

In its commentary on the resolutions presented by the four state delegations, the AMA Council on Medical Service concluded that it was not the intent of proponents of that doctrine to diminish cooperation between physicians and underwriters of insurance in the private sector of the economy or to increase the technical administrative problems of those companies in claims processing. The Council also voiced its view that a physician, in subscribing to the doctrine of individual responsibility, is free to exercise his own volition in his dealings with "third parties," and that he

does not bind himself in any way to refuse payments from "third party" paying agencies of which he approves, or in any other way commit himself to a rigid system of financial dealing with patients.

The Council report emphasized that physicians should become acutely aware of the manifest superiorities of direct billing as compared with taking assignments from patients for services rendered under the Medicare program. It emphasized that, under ordinary circumstances, direct billings of patients will prove superior to the acceptance of assignments.

The Council on Medical Service pointed out that under Public Law 89-97, the physician is legally entitled to set his own valuation upon his services, and to bill the patient directly for services rendered. That approach will be the least productive of misunderstandings with patients, the least demanding of the time of the physician and his office assistants, and the least disturbing to normal physician-patient relationships. If the direct billing approach is used, the Council stressed that the physician will not become involved in the multitude of complexities which embrace the taking of assignments. Specifically, by using direct billing, the doctor would not need to concern himself with questions as (a) the eligibility status of the patient and the extent of Medicare coverage; (b) the distinction between those services which are "covered" and those which are "excluded"; (c) the question as to whether a patient already has paid his Part B deductible premium; (d) the 20 percent coinsurance provisions with the inevitable need for double-billing when assignments are accepted; (e) the relation of what the physician feels his service is worth as opposed to what the carrier defines as a "reasonable" charge; (f) the presence or absence of complementary coverage; and (g) the use of the prescribed form.

Acting upon a resolution presented by the Texas and Kansas delegations, the American Medical Association House of Delegates adopted a resolution at the 1966 Annual Convention expressing deep gratitude and affection for former President and Mrs. Dwight D. Eisenhower upon the occasion of their golden wedding anniversary.

General and Mrs. Eisenhower celebrated their anniversary on July 1. It marked the first 50th wedding anniversary to be celebrated by a former President of the United States and his wife in 119 years. President and Mrs. John Quincy Adams celebrated a golden wedding anniversary in 1847.

The resolution authored by the Texas and Kansas delegations pointed out that former President Eisenhower has given a lifetime of service to his country, and through his dedication, freedom still lives in the world today.

House Speaker To Address TPA 87th Annual Meeting

Ben Barnes, speaker of the Texas House of Representatives, will deliver the major address at the 87th Annual Meeting of the Texas Pharmaceutical Association. The meeting will be held Aug. 17-20 in El Paso.

Barnes, a member of the National Legislative Conference Committee on Federal-State Relations, will speak on "Group Action in Politics" at the association's final session on Saturday afternoon, Aug. 20.

The meeting program will feature the theme "The Welfare of Pharmacy" and will include three other speakers.

Dr. William S. Apple, American Pharmaceutical Association executive director, will open the meeting Thursday with a talk on "Professional Partners," a preview of future relations of the Texas and American pharmacy groups. Dr. Apple will also address the association meeting Friday, Aug. 19, on "The Community Pharmacist in Institutional Practice."

Dr. George Archambault, US Public Health Service officer, will speak Thursday afternoon on "Medicare—Its Direct Application to the Profession of Pharmacy."

Commissioner John H. Winters of the State Board of Public Welfare will speak Thursday afternoon on "Expansion of Kerr-Mills Under Title 19."

The Friday afternoon session will feature a panel discussion on "Institutional Procedures for Pharmacy Consultants Under Medicare."

The TPA meeting will be based at El Paso's Hotel Cortez. The Woman's Auxiliary of TPA and the Drug Travelers Association of Texas will be meeting simultaneously at the El Paso Del Norte and Plaza hotels respectively.

Occupational Health Council To Sponsor Oregon Congress

The 26th Annual Congress on Occupational Health, sponsored by the American Medical Association's Council on Occupational Health, will be held Sept. 26-28 in Portland, Ore.

The Congress will be held at the Sheraton Hotel and Memorial Coliseum in conjunction with the 92nd Annual Session of the Oregon Medical Association and the 13th Annual Pacific Northwest Occupational Health Congress.

Discussion subjects will include the search for the tools of cancer prevention, sprains and fractures, impairment and disability problems, cardiac disability and workmen's compensation, aerospace medical problems, and returning to work following sickness or injury.

Additional information can be obtained from the Council on Occupational Health, American Medical Association, 535 N. Dearborn, Chicago 60610.

Medicare Seminars Planned

Texas Nursing Home Association will hold four Medicare seminars in the month of August.

Locations and dates of these seminars are as follows: (1) Dallas, Sheraton-Dallas Hotel, Aug. 9; (2) San Antonio, El Tropicano Motor Hotel, Aug. 10; (3) Houston, Sheraton-Lincoln Hotel, Aug. 11; and (4) Lubbock, Holiday Inn Parkway, Aug. 12.

Those wishing further information should write to John M. Crawford, Executive Director, Texas Nursing Home Association, 6225 US Highway 290, Austin.

Flying Physicians To Meet

The Flying Physicians Association will hold its 12th annual meeting Sept. 11-16 at the Dunes Hotel, Las Vegas.

Meeting sessions will be primarily concerned with discussions of various medical disciplines as they might ultimately relate to general aviation.

Further information can be obtained from Dr. James A. Martin, 3501 Gaston Ave., Dallas 75246. Dr. Martin is president of the association's Texas chapter.

Direct Billing Position Given By Blue Cross-Blue Shield

The position of Blue Cross and Blue Shield on providing benefits for services of members of the medical specialty groups when the patient is billed direct and not through the hospital was the topic of a discussion recently by members of the Committee on Liaison with Blue Shield of the Texas Medical Association.

In answer to questions being asked, it was reported that there are many variations of types of coverages provided by Blue Cross and Blue Shield covering some 1,934,123 persons and that many Blue Cross contracts provide benefits for such services when ordered by the attending physician and supplied by the hospital but do not have any benefits for such specialty services when provided by and billed direct by the physician. Benefits under such contracts cannot be split or divided between the hospital and the physician. Only the hospital benefits can be paid and no payment can be made to the physician under these contracts.

Many contracts do have some type of such benefits and some 1,358,000 persons have some type of major medical coverage including benefits for such services. In view of these facts, it was unanimously agreed that Blue Cross-Blue Shield would pay only benefits which are clearly specified and would adhere strictly to the provisions of its contracts. Committee members agreed that Blue Cross-Blue Shield should endeavor to amend contracts at time of renewal and develop new contracts to provide professional benefits in keeping with practices established and followed by the physicians and hospitals.

The Hospital and Physician Relations Committees of the Blue Cross-Blue Shield Board of Directors have unanimously agreed with this recommendation.

Blood Bank Organization Announces Name Change

Southwest Blood Banks, Inc., has officially changed its name to Blood Services.

The reasons for the name change were outlined by W. Quinn Jordan, executive director of the organization. "The change was made because of the many diversified fields of blood banking we have moved into in the last few years," he said. "The fields of blood banking, blood derivatives, blood storage, blood research, and blood insurance are all areas within which our organization works," Mr. Jordan continued.

Blood Services was formed in Phoenix, Ariz., 1943. Today the organization reportedly is the largest medically-sponsored, not-for-profit blood-banking system in the United States. It operates blood banking services in Texas, California, Nevada, New Mexico, Louisiana, Mississippi, Arkansas, Wyoming, Montana, North Dakota, South Dakota, and Arizona.

Mr. Jordan further pointed out that the change of name will not affect the services performed for the people in the organization's service communities.

Blood Services provides for the blood needs of 160 communities in Texas through 270 medical institutions. District offices are located in El Paso, San Antonio, Lubbock, Houston, and Harlingen, with branch offices in San Angelo and Laredo.

Health Service Will Issue Measles Protection Vaccine

The US Public Health Service will buy at least one and one-half million doses of vaccine during the next year to protect preschool children against measles, Surgeon General William H. Stewart has announced.

The vaccine will be offered to health departments receiving project grants under the national Vaccination Assistance Act. The Public Health Service will pay 68 cents a dose for the vaccine, including gamma globulin to be used in conjunction with the vaccine to minimize reactions.

The purchase was made in connection with the health service's cooperative effort with state and municipal health departments to eradicate measles, one of the most serious and widespread childhood diseases. Many communities have staged mass campaigns to avert epidemics or to prevent seasonal outbreaks. Since the licensing of the measles vaccine in 1963, some 12 million doses have been given in the US, and the number of cases of measles has dropped from 385,000 cases reported in 1963 to 266,000 reported in 1965. It is estimated that reported cases represent only one-tenth of the actual cases.

The Public Health Service reports that 150 million people are currently being served by 76 state and local health departments which have immunization projects in effect under the grants program of the federal government. Measles has an attack rate of 90 percent among unprotected children under 15, disabling thousands of children each year and causing up to 500 deaths.

Paper Permanence Problem Facing Medical Libraries

The *New York State Journal of Medicine* reports that many librarians are becoming concerned about the disintegration rate of paper made since 1870.

Many books and journals published in the first half of the twentieth century, it is feared, may not last until the year 2000. In the average research library more than 75 percent of material published before 1940 may be unusable in 25 years.

About 1870 a highly acid alum resin was introduced into the paper making process. Paper containing a high portion of ground wood disintegrates more rapidly than that with relatively little ground wood content. Low wood content paper, however, is expensive and has a relative transparency allowing undesirable show-through. Such paper can be coated in order to gain opacity and offset acidity of the paper itself.

The Paper Standards Committee of the American Chemical Society has made a long-term study of the paper longevity problem and has made recommendations for improvement of paper stock.

Some steps that can be taken toward preservation of scientific records include reprinting of short runs by publishers on high quality rag paper for libraries and microfilming of journal volumes for library distribution. Microfilm, however, is a comparatively new innovation and is not certain to have a long life, though it is thought that under proper storage conditions microfilm will be able to outlast paper.

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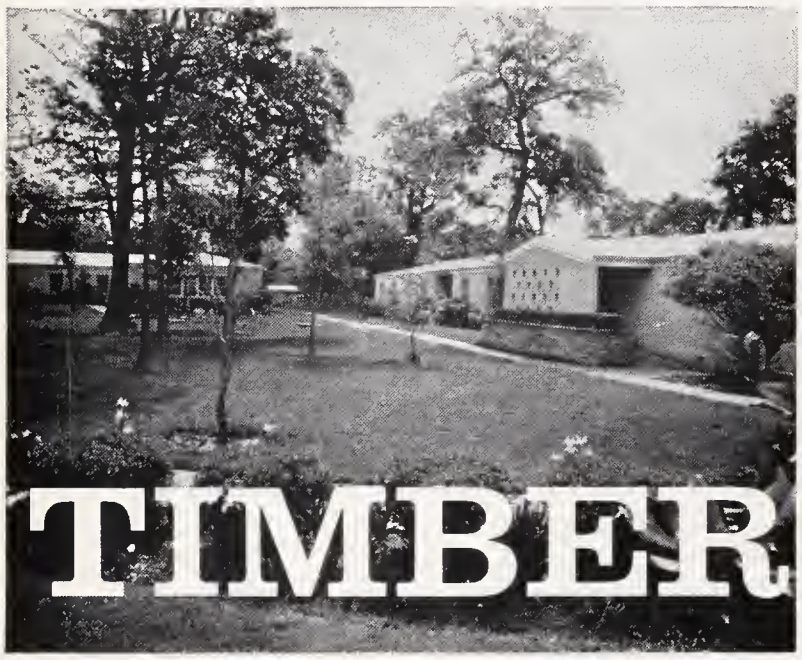

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Conference on Infant Deaths Scheduled for San Francisco

The American Medical Association Committee on Maternal and Child Care will sponsor a national conference on infant mortality Aug. 12-13 at the Fairmont Hotel in San Francisco.

The conference is open to chairmen and members of all state and county maternal and child care committees; perinatal and maternal mortality committees; state health department directors of maternal and child health; medical school faculty members in departments of obstetrics and gynecology, pediatrics, and preventive medicine; and other interested physicians and representatives of groups concerned with the problems of infant mortality.

Effective approaches for continuing the reduction of infant mortality will be explored. Participants in special workshops will study the problems of identifying high-risk patients, organization and delivery of special care services, manpower utilization, continuity of reproductive care, and prematurity. Special speakers will discuss the subjects of perinatal mortality studies, obstetric and pediatric education programs, research questions, expectant parent and sex education, and population control.

Those wishing registration information should write to Fred V. Hein, Secretary, Committee on Maternal and Child Care, American Medical Association, 535 North Dearborn St., Chicago 60610.

Physical Medicine Groups To Hold Annual Meetings

The 44th annual session of the American Congress of Physical Medicine and Rehabilitation and the 28th assembly of the American Academy of Physical Medicine and Rehabilitation will take place Aug. 28-Sept. 2 at the Sheraton-Palace Hotel in San Francisco.

Featured items on the program include "Symposium on Space Medicine and Technology and Physical Medicine and Rehabilitation: Common Interests and Problems"; "Symposium on Strokes"; "Symposium on the Clinical Management of Disability"; "Seminar on Bio-Medical Engineering: Approaches to Problems on Human Disability"; and five scientific sessions.

Dr. Lewis A. Leavitt of Houston, president of the American Congress of Physical Medicine and Rehabilitation, will preside over the first session of the congress and present the presidential address.

Oklahomans Plan Meeting

The Oklahoma chapter of the American Academy of General Practice will hold its Red River Valley Conference at Lake Murray Lodge, Ardmore, Okla., on Aug. 28.

Further information can be obtained from Dr. Roger Reid, chairman, 1001 15th NW, Ardmore, Okla.

Name Changes Sought By University of Texas

The University of Texas Board of Regents issued a resolution on May 31 proposing name changes in several divisions within the University of Texas System. The recommendations are part of an effort to make more uniform the legal titles of the University System components and to more readily identify these components with the University.

The proposals to be placed before the 60th Texas Legislature would change:

1. The Main University of Texas at Austin to the University of Texas at Austin;
2. The University of Texas South Texas Medical School to the University of Texas Medical School at San Antonio;
3. The University of Texas School of Public Health to the University of Texas Institute of Environmental Health at Houston;
4. The Institute for Dental Science to the University of Texas Dental Science Institute at Houston; and
5. The University of Texas Dental School to the University of Texas Dental School at Houston.

Other proposed changes consist of combinations as follows:

1. The University of Texas Medical Branch at Galveston to include the University of Texas School of Medicine at Galveston, the University of Texas School of Nursing at Galveston, and the University of Texas Medical Branch Hospitals at Galveston (including the John Sealy Hospital, the Rosa and Henry Ziegler Hospital, the Marvin L. Graves Hospital, the Children's Hospital, the Randall Pavilion, the R. Waverley Smith Pavilion, the John W. McCullough Out-Patient Clinic, and the Moody School for Cerebral Palsied Children;
2. The University of Texas Dental Branch at Houston to include the University of Texas Dental School at Houston, and the University of Texas Dental Science Institute at Houston; and
3. The University of Texas at

Frank Williams, porter for 14 years at Texas Medical Association headquarters building, receives a diamond pin on the occasion of his retirement. C. Lincoln Williston, executive secretory, makes the presentation. Mr. Williams, fondly known by many members of the Association, officially retired on April 29.

Houston to include the University of Texas Dental Branch at Houston, the University of Texas M. D. Anderson Hospital and Tumor Institute at Houston, the University of Texas Graduate School of Biomedical Sciences at Houston (including a Division of Continuing Education for Physicians), and the University of Texas Institute of Environmental Health at Houston.

The Board of Regents is also considering asking the Legislature to change the name of the University of Texas Southwestern Medical School at Dallas to the University of Texas Medical School at Dallas.

NTA Pamphlet Promotes Public Education Program

The National Tuberculosis Association is sponsoring an educational program on respiratory diseases.

The program is intended to (1) provide information to the public on emphysema and other chronic respiratory diseases, and (2) create public awareness of local, state, and national association involvement with these health problems.

The association has placed advertisements in national magazines, in local newspapers, and on radio and television. The advertisements offer a free booklet, "Your Breathing Troubles: Understand Them, Face Them, Treat Them," which can be obtained by writing NTA. The advertisements also instruct readers to consult with doctors.

The pamphlet discusses causes and symptoms of asthma, chronic bronchitis, and emphysema; and it informs the readers as to existing treatment for these conditions.

Physicians who have not received booklets may do so by contacting local tuberculosis associations or writing to National Tuberculosis Association, GPO, Box 2400, New York 10001.

Postgraduate Courses

Laboratory Refresher Training Courses, Atlanta, Ga.—Laboratory refresher courses will be offered beginning Aug. 1 by the US Public Health Service's Communicable Disease Center.

Course topics and dates are as follows: principles of fluorescent antibody microscopy, Sept. 19-23; laboratory methods in the detection of rabies, Sept. 26-30; fluorescent antibody techniques applied to bacterial identifications, Sept. 26-Oct. 7; and basic clinical chemistry, Sept. 26-Oct. 7.

Other courses will be offered in various time periods through June 30, 1967.

Application forms and further information can be obtained from the Training Office, Laboratory Branch, Communicable Disease Center, Atlanta, Ga. 30333.

Obstetrics and Gynecology, Galveston, Aug. 3-6.—A four-day postgraduate course in obstetrics and gynecology will be offered by the University of Texas Medical Branch. Also cooperating in presentation of the course are Texas Medical Association, Texas State Department of Health, Texas Academy of General Practice, and Child Health Program of the University of Texas.

Guest speakers will be Dr. Isadore Dyer, New Orleans, Tulane University Department of Obstetrics and Gynecology; Dr. R. A. H. Kinch, London, Ontario, Canada, University of Western Ontario Department of Obstetrics and Gynecology; Dr. William H. Masters, St. Louis, Reproductive Biology Research Foundation; and Dr. Tiffany J. Williams, Rochester, Minn., the Mayo Clinic Department of Gynecology.

Subjects under study will include mechanism of intravaginal contraceptive failures, primary ovarian failure, surgical management of infertility, psychiatric approach to sexual problems, perinatal mortality, postpartum hemorrhage, carcinoma of the cervix in pregnancy, diseases of the female urethra, and severe accidental trauma in pregnancy.

"She's always sick. Are there any condominium hospitals?"

For further information and for registration write to Department of Postgraduate Education, University of Texas Medical Branch, Galveston.

An obstetrics and gynecology course for nurses will also be offered from 2-5 p.m., Aug. 3-5. Those interested should write Miss Dorothy Felis, OB-GYN Supervisor, UTMB Hospitals, Galveston.

Pediatrics Postgraduate Medical Education, University of Colorado School of Medicine, Estes Park, Colo., Aug. 1-5.—The ninth annual five-day postgraduate course in pediatrics has been scheduled by the Department of Pediatrics and the Office of Postgraduate Medical Education, University of Colorado School of Medicine.

In addition to the University of Colorado faculty, members of the guest faculty will include Dr. Marvin Cornblath, professor of pediatrics, University of Illinois College of Medicine, Chicago; Dr. Joseph Dancis, professor of pediatrics, New York University School of Medicine, New York; and Dr. Richard T. Smith, professor and chairman of the Department of Pediatrics, University of Florida College of Medicine, Gainesville.

Additional information may be obtained from the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver, Colo. 80220.

Dermal Pathology, Houston, Sept. 12-16—A five-day course in dermal pathology will be sponsored in September by the Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston and the University of Texas M. D. Anderson Hospital and Tumor Institute.

The course, designed primarily for pathologists and dermatologists, will review the basic concepts of dermal pathology, including embryology, histology, electron microscopy, histochemistry, inflammatory dermatoses, granulomatous dermatoses, reticuloendothelial dermatoses, and nevi and neoplasms.

Those wishing to register or acquire further information should write to Division of Continuing Education, The University of Texas Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston 77025.

Physicians Can Aid Early TB Examinations Required for School Personnel by Sept. 1

By Sept. 1, 1966, the licensed physicians of this state will have certified tuberculosis examinations for an estimated 160,000 Texans. This large segment of our population is made up of the teachers, administrators, cafeteria and custodial personnel, and all other employees of Texas public schools. Beginning this September, school employees must have proof of tuberculosis examination. Perhaps because of the vast new governmental health and medical care programs being initiated, the new act supplementing the Texas Tuberculosis Code (Art. 4477-11, V.C.S.) has gone into effect relatively unnoticed during the past year. Section 5 of this new Act, codified as Art. 4477-12, Vernon's Civil Statutes, provided that:

All school personnel, including teachers, clerical employees, supervisory personnel, bus drivers, personnel handling food and personnel performing janitorial services, shall be required to furnish the governing board of any public school in this state on or before September 1 of each year a certificate signed by a person licensed to practice medicine in this state, revealing that such school personnel have been examined for the disease of tuberculosis during a period of time not exceeding one hundred twenty (120) days prior to September 1 of each year, and revealing the results of such examination, and revealing that the results of such examination have been furnished the State Board of Health by the person performing the examination. No person shall be permitted to perform his or her duties in the absence of such certificate being furnished the governing board of the school.

The principle of universal testing for tuberculosis is one for which organized medicine has long stood and the new act, of which section 5 is only a part, was actively supported by the Committee on Tuberculosis of the Texas Medical Association and by the Association itself when the legislation was introduced in 1965. Because implementation of this law involves the participation of the licensed practitioners of medicine in this state, the ultimate success of this new legislation will depend greatly upon their understanding and cooperation.

The Texas Department of Health has mailed to local health authorities an explanation of section 5 as well as model certificates for physicians to use in certifying that the person tested is free of "active tuberculosis." The health department has anticipated that many school districts may choose to hold mass examinations of all employees and to certify these results in list form.

However the bulk of the testing will probably be done by the private physician, and he must provide the person tested with a certificate to be presented to his school board, stating that during a period of time not exceeding 120 days prior to Sept. 1 (that is, between May 4 and Aug. 31, 1966) he was examined for the tuberculosis by the certifying physician and that the results of such examination, which are to be included on the certificate, have been furnished to the State Board of Health by the certifying physician. The law specifically provides that the employee shall not be allowed to perform his or her duties in the absence of such a certificate.

In implementing this statute, the Department of Health has promulgated rules and policies for the guidance of school administrators and public health officials. These rules and policies also offer a reasonable and workable guide for the private practitioner in certifying that the employee in question has been examined. Under the health department's guidelines, an "examination" may consist of a negative tuberculin skin test. If this test results in a positive reaction, a chest x-ray must be obtained and reported to the administrative officers of the school district. A negative chest x-ray may be used in lieu of the tuberculin test. An employee who has a positive tuberculin reaction will need the report on his x-ray and the person with suspicious findings on x-ray examination will need additional examination by his physician who will decide whether or not a certificate of freedom from active tuberculosis can be issued.

While the law does not require that the certificate states that the examinee is "free of active tuberculosis," as does the certificate suggested by the health department, it does require "revealing the results of such examination." Clearly, the implication is that the physician should make a diagnosis of whether or not the examinee has active tuberculosis; the law requires the reporting of the "results of such examination" for tuberculosis and not just the results of any specific tests which may or may not prove positive. Thus, the physician must make a diagnosis as to whether or not the examinee has tuberculosis, and the mere reporting of the examinee's reaction to a particular test, such as the tuberculin skin test, will not suffice as proper certification.

State health authorities and local school officials are chiefly concerned with the implementation of this new law. Like so many regulations which require action prior to a specific deadline, the lack of publicity may result in a rush of school personnel to be tested during the last few days of August. Obviously, a positive reaction to the tuberculin skin test and the consequent necessity of having an x-ray with perhaps additional examination by a physician may involve a great deal more time than is available immediately prior to the Sept. 1 deadline. Physicians who have school personnel among their patients might provide a valuable service to those patients by reminding them of this new law and suggesting that they have their certificates filed well before the September deadline.

—Philip R. Overton, LL.B., and Sam V. Stone, Jr., LL.B., Austin, Legal Counsel, Texas Medical Association.

Almost no farming is possible on barren land above Kowloon. With persistence crops are watered.

Tribulations of Far Easterners Described by San Antonio Doctor

[Editor's Note: While on a tour of duty recently at Letterman Army Hospital in California, Dr. M. Jeanne Fairweather, chief of General Medical Clinics at Brooke General Hospital, Fort Sam Houston, flew to Tokyo to attend a medical meeting. In Tokyo an Army chaplain with whom she had served arranged for a visit to a refugee center high in the mountains of Kowloon, near Hong Kong, and another center in Portuguese Macao. The following account describes her unusual experiences at one of these remote centers.]

I LEFT TOKYO by air in a bad storm and arrived at the Hong Kong airport, still in stormy weather, landing on a narrow airstrip running straight out into the water.

A typhoon was in full sway, so I spent the first few days indoors in the beautiful Mandarin Hotel on the waterfront, watching hundreds of ships from over the world seek shelter. The shelters for the small boats were filled. With this bizarre scene to watch, I sat out the typhoon.

When the storm ceased, I crossed on the ferry to the Kowloon side to meet the Maryknoll priest, Father Trube, who is in charge of the refugee center in the mountains above Kowloon.

This remarkable New Yorker was a missionary priest in China for ten years before the Communists imprisoned him for more than two years and then expelled him. As with many other missionaries, the Communists tried to humiliate and frighten him into giving up Christianity. With two other priests, Father Trube was taken to a pagan village and put into a filthy, cold, damp, stinking pigsty, where they were left for more than a year. Their prison, with mud floors and no beds, was overrun by rats as large as cats. The little food they were given was rancid.

By the time he was expelled, the missionary priest was critically ill. In a Hong Kong hospital he was nursed back to health and offered the opportunity to return to the States. However, because of his love for his Chinese flock, Father Trube requested permission to stay in Hong Kong to open a refugee center for the miserable people who were able to escape from Red China.

This remarkable priest took my

Army chaplain friend and me through a rough pathway that passed as a street. It was so narrow that the priest's old car could hardly maneuver between the miserable shacks on either side.

The priest explained to us that this tiny area was the Walled City. Although in British territory, it still belongs to China, a cause of violent discussions between the two countries.

Despite the narrowness of the street, women used the pathway to wash their clothes, in a stream of dirty water running from pumps located at intervals along the road.

The mountainside was covered with hundreds of shacks, thrown together with tar paper, cardboard, and straw, topped with rusted tin roofs. Only a few of the shacks were made of mud bricks.

In this pitifully inadequate settlement live some 250,000 refugees, most of them Buddhists or followers of other Eastern religions.

The danger of uncontrollable fires is ever present; six great fires have already wrought disaster. In one fire, 35,000 refugees died, and the settlement was completely destroyed. The wind was strong that night, whipping the fire to a frenzy that swept the area like a tornado. In the second fire, believed to have been set deliberately by the Communists, 500 shacks housing 1,600 persons were destroyed.

The priest told of another night fire in the border town of Sha Tao Koc, which we later visited. The main alley divided the town into a British and a Red sector. The Red sector, where the fire began, had no fire apparatus. The Communists, refusing to accept British help, put up a line of armed soldiers to prevent the use of British fire-fighting equipment and water. The British had to stand by and watch the horrifying destruction and the charred bodies.

In 1962, Father Trube told us, hundreds of thousands of refugees crossed the Red Chinese border before the Communists replaced their strong border guard. Surprisingly, a large number of these people were well-to-do landowners, teachers, government officials, and officers in the armed forces. On the long trip over the mountain and across the river, they brought little more than the clothes on their backs and their knowledge. After their escape they lived in unsanitary caves and miserable shacks, and in squatter camps. Some 200,000 of them shared

Astounding workers, the Hakka women appear delicate and frail. They normally are seen carrying such heavy loads as these.

crowded quarters, sleeping in shifts with ten or more people in rooms only six feet square. Another 10,000 slept in the streets, alleys, doorways, and on roofs.

Here, the missionary priest moved into a one-room squatter shack which became the chapel, reading room, community and welfare center, employment bureau, catechumenate, and dispensary.

Father Trube told a touching story of a group of deaf and dumb youths who had fled from Shanghai. They had been marked for death by the Communists, who detest useless people. Father Trube learned sign language to communicate with them and engaged them in a co-partnership painting religious pictures. This proved so successful that it provided a livelihood for the entire group. They were the first pagan converts in the refugee center.

Not far from Hong Kong—typical ancient and present-day way of farming with water buffalo.

As families moved into the center, Father Trube attempted to help each family use its trade. Although there is almost no farming land on the barren mountain, one family found enough land to begin raising pigs.

The Communists went from shack to shack, distributing subversive literature and talking to the refugees. Since the refugees had no place to discuss openly what they heard, many sympathized with Communist doctrine. Eventually the opening of a meeting place did much to stop the spread of Communist propaganda.

A public address system was installed to broadcast local news, prayers, and music, as well as to locate lost children and to give important information in a variety of languages, including Cantonese, Hakka, Mandarin, and Swatonese. While I was there, I heard the PA system give advice on cleaning the areas around the shacks and apartments. The inhabitants were told, for instance, not to throw their garbage out of windows. It was not unusual to walk past a shack and be struck by garbage flying out of a window.

Food and medical care for these hundreds of thousands of people was an almost impossible task. Then the United States government came to their rescue. A part of the medical program was subsidized by one of the refugee aid programs in the US.

Although the US donated huge quantities of flour to the colony, the gift was difficult to use. Father Trube conceived of the idea of

noodles for feeding the refugees. There was no money to buy ovens to bake bread, but from parts salvaged from a crashed bomber, the refugees were able to build a noodle machine.

I watched a refugee pour pounds of American flour into the crude machine, add British Calorid (a liquid "whole food"), mix with water, and force the mixture through little sieve-like openings. Out came the noodles, to be caught in the bare hands of a worker who folded them into a figure eight. A third worker took the noodles outside and draped them on racks to dry.

Powdered milk is provided, but it must be taken in wagons to the foot of the mountain daily where boiled water is added in a "milk factory" and brought back up the mountain in time for 150 gallons to be distributed to children standing in well-mannered lines awaiting their share. Noodles are provided each week to 9,000 families, each knowing when to arrive for its portion.

The government is attempting to replace shacks with apartments. On Ngnau Tau Kok Road, we watched the old shacks being torn down and an area blasted out of solid rock to make way for a new building.

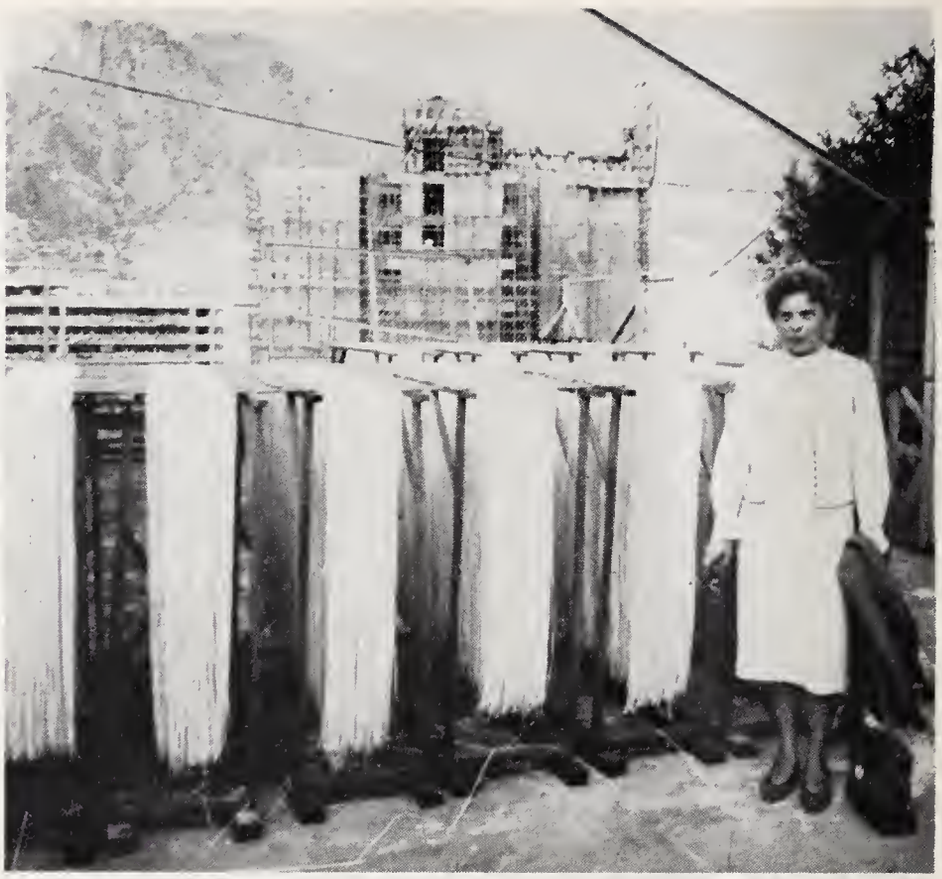
All work is done by hand. The Hakka women, wearing flat, veiled hats, appear to be delicate and frail, but are astounding workers. The Hakka are a fascinating people. The name means "guest," although they migrated from Northern China 2,000 years ago. They still have their own language and customs, and only recently have intermarried in Hong Kong. The Hakkas in Portuguese Macao, however, still remain aloof and will not intermarry.

At the dispensary there were two physicians and a nurse, all refugees. Most of the drugs were samples from the US. Among them I noticed a large quantity of appetite depressants.

"American doctors do not understand our needs," one of the doctors told me. "Our people need food, not appetite depressants."

Eighty patients are seen in the early morning. After a break for lunch, the staff rests and sees 80 more before supper. In the evening some 70 to 90 more are treated.

Many of the patients suffer from malnutrition, many from tuberculosis. X-rays are taken in a government hospital in town, the patients from the center being taken into town in truck-loads on specific days. Accepted TB medications are used, but all treatment is done on an outpatient basis.



From parts of a crashed bomber, refugees from Red China built a noodle machine to use US gifts of flour. Dr. Fairweather stands near noodles hung to dry.

Among several side trips was one to the New Territories, the land of the "Water People" some 90 miles from the mountain, which was leased from China in 1898. The new mental hospital in this area holds 1,000 patients, all of them committed for murder or other extremely violent acts, and already there is a great need for more beds.

On the way to the Red border we stopped to visit the fabulous home of a wealthy Chinese physician who had defected some years ago. After amassing a fortune during his exile, he built a home which was a replica of his home in Old China, filled with museum pieces. It is guarded now by huge black dogs as well as human guards.

The doctor told me, in perfect English, that he had a son studying medicine in California. His brother is a noted Catholic bishop who is being held incommunicado in the interior of Red China. He kept in touch with his brother, through spies, until a year ago when the last contact was broken, but it is believed that the bishop still lives somewhere as a prisoner.

We left the doctor to drive along a narrow, hairpin winding road to the Red border. Dusk was falling as we stood by the tangled barbed-wire barricade, looking across a narrow valley to the barbed wire and guards on the Communist side. A group of dirty Hakka women surrounded us, trying to sell their long-stemmed pipes and flat veiled hats for four times their worth. A few Oriental tourists at the border were buying objects from a tiny stall, lit by one small candle. I was intrigued by the beautiful pieces of ivory, jade, and cloisonne, but was warned that the prices were exorbitant. I did pick up a beautiful statue, however, and was astounded when the shopkeeper said, in a perfect British accent, "Father, tell the lady she may not buy from my shop. Customs would confiscate the things she bought. She would never get them home, because they are all from Red China."

I came away from the tiny shop without the little statue, but I brought away treasures that customs could not confiscate . . . Memories of a sad and tragic land, filled with the charm and fascination that spring from courage and hope.

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Orthopedic Surgeon Assist

A San Antonio physician who believes in tithing his time as well as his money is returning in October for a second tour of duty at a remote medical center in Korea.

Dr. Coyle W. Williams, Jr., an orthopedic surgeon, first volunteered his services last summer through the Board of World Missions of the Presbyterian Church, U.S. As a result of his request, he was sent for one month to the Presbyterian Medical Center in Chonju, 150 miles south of Seoul in rural Korea.

"The Presbyterian Medical Center in Chonju," says Dr. Williams, "is a teaching hospital and has 32 residents, so my job was to organize the graduate orthopedic teaching program, to lecture daily to the interns and residents, do demonstrative orthopedic cases, and to teach the residents basic orthopedic surgery."

Crippled children were the major challenge. Not one doctor in the area of more than three million people was doing surgery on crippled children.

"After getting organized, we did as many as eight crippled children cases per day," Dr. Williams said. "The outpatient clinic then began to fill with crippled children, to the extent that we could not handle them all, and we had to turn 40 to 50 children away every day."

Dr. Williams did 80 operations himself and assisted the staff men and residents in many of the routine orthopedic procedures. He consulted with more than 400 outpatients in the clinic, most of whom were patients with polio, cerebral palsy, tuberculosis in various forms, and osteomyelitis.

"We were hampered by the smallness of the physical plant, and we could have done more if our physical facilities had been better," Dr. Williams said.

He noted that there is a critical need for specialists in various fields. Recently he was notified of a specific and immediate need for several specialists in internal medicine; the present internist was leaving Korea this summer.

Korean youngster, victim of polio, leans on stick, the usual mode of transportation when lower limbs are paralyzed.

Crippled Children in Korea

"The medical center is in danger of losing its approval for intern training due to the leaving of its internist," Dr. Williams stated. "So several people in internal medicine could give some time during the year, perhaps from three weeks to a month to keep up the internal medicine program. This could be very valuable."

Dr. Williams was asked to return this year and plans to return during the month of October.

"Most of my work there has been with crippled children, and as this is elective surgery, they are already in the process of scheduling surgery for me and consultations mainly in this realm," he said.

The center also is in need of other specialists to visit and contribute to the teaching program, such as a urologist, psychiatrist, anesthesiologist, and an obstetrician-gynecologist.

A motion picture of the medical center is now being filmed to use in a drive for funds to build a new teaching hospital at Chonju. Already $500,000 has been raised by the women of the Presbyterian church toward this effort, but another $1,500,000 will have to be raised before the hospital becomes a reality. Dr. Williams is also showing a movie of Chonju and speaking at churches to aid the fund-raising effort.

"Besides the medical work done in Korea," Dr. Williams said, "it was very interesting to visit in Asia and learn something of the culture of the country.

"During my stay at Chonju, we were visited by Prince Yi, who is one of the last remaining princes of the Yi family that ruled Korea from 1390 to 1910. Also, Chonju was visited by President Park, who came to dedicate a new dam on the Somjin River. The medical director, Dr. Paul Crane, was invited to meet him at the Governor's Mansion, and took me along. As we entered the Governor's Mansion, everyone had to remove his shoes, which proved to be embarrassing as it was immediately noticed by everyone that I had a hole in my sock. Out of my dark blue socks stuck a big white toe!"

The short-term missionary project of the Presbyterian Church, U.S., sends especially qualified people who are needed on the mission field for a short period. The specialists pay their own travel expenses and their own expenses at the mission station.

"It is my conviction," says Dr. Williams, "that a Christian's time should be tithed as well as his money, and in my opinion this gives the physician a chance to do the most good in the shortest time."

This small Korean boy, who has had tuberculosis of the spine with a spinal fusion, prepares to put on his brace.

Pamphlets

Two pamphlets, "Mental Health Jobs Today and Tomorrow," and "How We Can Get the Nurses We Need," are available for 25 cents each from Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.

"Mental Health Jobs Today and Tomorrow," Public Affairs Committee Pamphlet No. 384, has been issued in cooperation with the National Institute of Mental Health.

The pamphlet contains discussions of education and training required for a psychiatrist, clinical psychologist, social worker, psychiatric or mental health nurse, and occupational therapist. Tuition costs and sources for scholarship information are included. Supplementary positions, such as psychiatric aides, mental health

counselors, volunteer work, and summer jobs are also described.

"How We Can Get the Nurses We Need," Public Affairs Pamphlet No. 385, is a survey of problems facing the nursing profession today. Facts about nursing careers—educational requirements, salaries, work loads, new methods, and problems—are discussed in an effort to interest capable young people in entering the nursing profession. The booklet also seeks to publicize the present and future need for more and better nurses.

"The Importance of Carbohydrates, Simple and Complex," a newly published booklet by the Cereal Institute, Inc., of Chicago, is based on research of scientific literature over the years and on current scientific research studies.

The booklet includes a basic cereal and milk low-fat breakfast pattern with a table of food values.

The 10-page booklet may be obtained free of charge from the Cereal Institute, Inc., 135 South La Salle Street, Chicago, Ill. 60603.

Personals

Dr. Stanley W. Olson of Houston resigned as dean of Baylor University College of Medicine effective June 21.

Dr. Olson, Baylor dean since 1953, had just completed a nine-month sabbatical leave, during which time he had toured medical centers in many parts of the world.

Dr. Olson was the fourth dean of Baylor University College of Medicine, the second since the school moved to Houston in 1943.

A faculty executive committee will assume Dr. Olson's duties until the appointment of a new dean is decided prior to the beginning of the 1966-67 school term in September. Committee members are Dr. Raymond D. Pruitt, chairman of the Department of Medicine; Dr. Hebbel E. Hoff, chairman of the Department of Physiology; and Dr. Shervert H. Frazier, Jr., chairman of the Department of Psychiatry.

Dr. Edwin L. Rippy of Dallas was elected to the Dallas County seat on the State Board of Education on June 4.

Dr. Rippy was chairman of TMA's Committee on School Health from 1961 and is still a member of that committee. He has also been TMA adviser to the Texas Congress of Parents and Teachers. He was a member of the Dallas School Board from 1950 to 1963, serving as president for the last seven years of that period. He has been actively involved in Texas education since 1950.

Dr. Denton A. Cooley of Houston was named a 1966 recipient of the University of Texas Distinguished Alumnus Award in May.

Dr. Cooley is professor of surgery at Baylor University College of Medicine and chief of cardiovascular surgery at St. Luke's Episcopal and Texas Children's Hospitals. He is a 1941 Phi Beta Kappa graduate of the University of Texas and a 1944 medical graduate of Johns Hopkins University School of Medicine in Baltimore.

The Distinguished Alumnus award will be presented on Oct. 14 by the UT Ex-Students' Association at the annual Homecoming banquet. Award selections are made on the basis of outstanding professional achievements and support of University of Texas and alumni programs.

Eleven Texas doctors recently inducted into the American College of Physicians are Dr. Pierre P. Poole of Brownsville; Drs. Eugene P. Frenkel and Jay P. Sanford, both of Dallas; Maj. Henry Worth Boyce, Jr., of Fort Sam Houston; Drs. Richard M. Fenno and Stuart Riggs, both of Houston; Drs. Milward W. Bayliss and Gerhard A. Meyer, both of San Antonio; and Drs. John D. Bonnet, Robert J. Carabasi, and Per H. Langsjoen, all of Temple.

Dr. Anthony A. Deep of San Antonio was installed as a fellow of the American College of Obstetricians and Gynecologists in Chicago in May.

Dr. J. Fred Mullins, chairman and professor of dermatology at the University of Texas Medical Branch, assumed duties in May as a council representative to the Residency Review Committee for Dermatology of the American Medical Association Council on Medical Education.

Dr. John D. Chase, chief of staff at the Houston Veterans Administration hospital, has been named director of the VA hospital in Oklahoma City.

Dr. Louis J. Stahl of Gonzales received his Fifty Year Pin and the Golden T certificate from the University of Tennessee in June. Dr. Stahl was graduated from the University of Tennessee College of Medicine in June, 1916.

Correction

In the June issue of *Texas Medicine* an error was made in the list of authors of the article, "Acute Hyperparathyroidism; a Surgically Correctable Cause of Coma." The author listed as William R. Berry, M.D., should have been listed as William B. Berry, M.D.

New Books In TMA Library

Aldrich, C. K.: *An Introduction to Dynamic Psychiatry*, New York, McGraw-Hill Book Co., 1966.

Ciba Foundation Symposium: *Preimplantation Stages of Pregnancy*, Boston, Little, Brown and Co., 1965.

Ciba Foundation Symposium: *Transcultural Psychiatry*, Boston, Little, Brown and Co., 1965.

Conn, H. F., et al.: *Current Diagnosis*, Philadelphia, W. B. Saunders Co., 1966.

Edwards, Jesse E. et al.: *Congenital Heart Disease*, 2 volumes, Philadelphia, W. B. Saunders Co., 1965.

Foster, W. D.: *A History of Parasitology*, London, E. & S. Livingstone Ltd., 1965.

Hildick-Smith, Gavin, et al.: *Fungus Diseases and Their Treatment*, Boston, Little, Brown and Co., 1964.

Johnson, S. A., and Greenwalt, T. J.: *Coagulation and Transfusion in Clinical Medicine*, Boston, Little, Brown and Co., 1965.

Kessler, E.: *Diabetic Acidosis; Part 1: A Programmed Course of Instruction; Part 2: Illustrated Medical Lecture Tape and Film Strip*, Los Angeles, Edlen Research, 1965.

King, J. H., and McTigue, J. W.: *First World Congress on Cornea*, Washington, D. C., Butterworth Inc., 1965.

Lawson, J. H.: *A Synopsis of Fevers and Their Treatment*, ed. 11, Chicago, Year Book Medical Publishers, Inc., 1965.

Leavell, H. R., and Clark, E. G.: *Preventive Medicine for the Doctor in His Community*, ed. 3, New York, McGraw-Hill Book Co., 1965.

McKusick, V. A.: *Heritable Disorders of Connective Tissue*, ed. 3, St. Louis, The C. V. Mosby Co., 1966.

Maegraith, B.: *Exotic Diseases in Practice*, New York, American Elsevier Publishing Co., Inc., 1965.

Nader, R.: *Unsafe at Any Speed*, New York, Grossman Publishers, 1965.

Ratliff, F.: *Mach Bands: Quantitative Studies on Neural Networks in the Retina*, San Francisco, Holden-Day, Inc., 1965.

Richter, C. P.: *Biological Clocks in Medicine and Psychiatry*, Springfield, Ill., Charles C Thomas, Publisher, 1965.

Roche Laboratories: *The Anatomy of Sleep*, Nutley, N. J., Hoffmann-LaRoche Inc., 1966.

Ruyssen, R., and Vandendriessche, L.: *Enzymes in Clinical Chemistry*, Amsterdam, Elsevier Publishing Co., 1965.

Samter, M., and Alexander, H. L.: *Immunological Diseases*, Boston, Little, Brown and Co., 1965.

Second National Conference on Cardiovascular Diseases: *The Heart and Circulation; vol. 1: Research; vol. 2: Community Services and Education*, Washington, D. C., Federation of American Societies for Experimental Biology, 1965.

Sevelius, G.: *Radioisotopes and Circulation*, Boston, Little, Brown and Co., 1965.

Stewart, G. T.: *The Penicillin Group of Drugs*, Amsterdam, Elsevier Publishing Co., 1965.

University of Texas M. D. Anderson Hospital and Tumor Institute: *Developmental and Metabolic Control Mechanisms and Neoplasia*, Baltimore, The Williams and Wilkins Co., 1965.

Walford, R. L.: *Leukocyte Antigens and Antibodies*, New York, Grune & Stratton, Inc., 1960.

Wasserman, P.: *The Librarian and the Machine*, Detroit, Gale Research Co., 1965.

Wiltse, C. M., ed., US Army in World War II, Technical Services, Medical Dept.: *Medical Service in the Mediterranean and Minor Theaters*, Washington, D. C., Government Printing Office, 1965.

Book Notes

Pharmacology and Therapeutics, edited by Arthur Grollman, ed. 6, 1181 pp., 330 illus., tables, Philadelphia, Lea & Febiger, 1965. $15.

BECAUSE OF the great numbers of new therapeutic agents physicians are constantly confronted with, there is an increasing sense of frustration in often being forced to depend on promotional literature for information and guidance. Rarely is there time to read the fine print of the package inserts which, by law, now serve as legal documents, giving complete information on uses, effectiveness, and toxicity of a preparation.

There has been an increasing need for an authoritative drug reference book which would adequately and concisely describe the actions of the many drugs now available. There are several new editions of major general texts on these subjects now available. One, Di Palma's *Drill's Pharmacology in Medicine*, is excellent, giving the latest concepts of drugs in depth but less emphasis on therapeutic usage. Another, Goodman and Gilman's *The Pharmacological Basis of Therapeutics*, is also excellent, but it now has become almost encyclopedic in size, and is thus unwieldy for quick reference.

Dr. Grollman is well known as an authority in this field. His new text shows some revision from previous editions, and contains a chapter on prescription writing and the legal aspects of drug therapy—information sorely needed. The book presents straightforward and uncomplicated knowledge of pharmacology and practical drug therapy without lengthy theoretical discussions. The data are presented from a scientific as well as from a practical background, and the book contains a concise and complete source of information on the indications, actions, dosage, toxic effects, and administration of all official drugs, including those recently introduced.

This text can be recommended as one most nearly conforming to the ideal pharmacology and therapeutic reference. It provides a wealth of quick and concise information for students, practitioners, and specialists in any field of medicine.

—L. C. Paggi, M.D., Austin

The Visual Fields, edited by David O. Harrington, 386 pp., illus., St. Louis, C. V. Mosby Co., 1964. $16.

THIS BOOK provides an excellent reference for the practicing ophthalmologist and student of ophthalmology, thoroughly exploring all aspects of perimetry, its instrumentation, its techniques, its anatomical basis, and its interpretation. *The Visual Fields* is well written though tedious to read. The comprehensive manner in which all aspects of perimetry are dealt with is a tribute to Dr. Harrington's intense interest in his subject.

The second edition is an improvement over the first, in that more illustrative cases are presented, newer instrumentation and techniques are discussed, and the section on glaucoma is almost completely rewritten. This section of the book should be of profound interest to all practicing ophthalmologists as it is most thorough and illustrative in its description of glaucoma and glaucomatous field defects.

The book is divided roughly into two portions. The first one-third deals with examination of the visual fields, and includes an extensive section on the anatomy of the visual pathway as well as routine procedures of the visual fields examination and some of the author's methods. The latter two-thirds of the book is dedicated to the interpretation of defects in the visual fields. Included are innumerable illustrations showing nearly all types of visual field defects with case presentation, field studies, and anatomical drawings of lesions encountered.

This textbook of clinical perim-

etry, though difficult to read in its entirety, serves as an excellent reference for the ophthalmologist and neurologist.

—JERRY JOHNSON, M.D., Beaumont

Caring for the Aged. Bertram B. Moss. 372 pp. Garden City, N. Y., Doubleday & Co., Inc., 1966. $4.95.

DR. MOSS, a physician who has taken special interest in the care of the aging, shares with us the basic facts, the latest findings, and the results of years of experience in his own practice. This is a book filled with specific, practical guidance for anyone who is in any way involved with an elderly person.

Among the topics Dr. Moss covers are housing for the elderly, adjustments in a household with children, nursing homes, diseases of the heart and blood vessels, digestive disorders, arthritis, cancer, impairment of vision and hearing, psychological and mental problems, quacks and frauds, terminal illness and death, legal and financial resources, and the provisions and implications of Medicare.

Controversy in Internal Medicine, edited by Franz J. Ingelfinger, Arnold S. Relman, and Maxwell Finland, 679 pp., illus., Philadelphia, W. B. Saunders Co., 1966. $14.50.

TWENTY-THREE controversial topics are each discussed by at least two experts selected to present contradictory points of view. The natures of particular controversies are indicated, and the reader is able to scrutinize arguments used to defend positions. Editorial comments, usually at the end of each section, serve to clarify further the nature of the issues. The subjects covered are of general interest and should appeal not only to internists but to all practicing physicians.

It is not too surprising that most differences of opinion exist because of inadequate data. The frequency with which conclusions are drawn on the basis of poorly controlled studies is also readily apparent. A strong case is made for the necessity of well designed and controlled prospective studies.

Of the major topics selected for discussion, six are gastroenterologic, four renal, and four cardiovascular, reflecting the specialty interests of at least two of the editors. A considerable number of additional topics can profitably be presented in similar fashion in the future. It is likely that *Controversy in Internal Medicine* will form the basis for a second volume or perhaps a new series entitled "Controversy of the Month."

—BURTON COMBES, M.D., Dallas

New Films In TMA Library

Mrs. Reynolds Needs a Nurse. Sound, black and white, 38 minutes, 1963, Dolores Little, Director.

DESCRIBING the problems involved in caring for a "difficult" patient, this film shows the importance of personalized care, the necessity for a complete nurse-patient relationship, the patient's ever-present emotional needs, and the development of a nursing plan.

Toymakers. Sound, black and white, 30 minutes, 1963.

THE LIVES of mentally retarded persons in institutions are portrayed in a thought-provoking way. The film depicts the range of retardation—from severe to mild—and shows how the institution must develop a varied program to meet the many needs of all the retarded—from those residents who need total nursing care to those who can be trained for work and life outside. The film makes the point that unless the community is prepared to receive them, many residents may be compelled to needlessly live out their lives in the institution.

Essentials of the Neurological Examination. Sound, color, 50 minutes, 1962, Dr. Francis A. Vazuka, Director.

A NONSPECIALIST in neurology performs a relatively thorough neurological examination of an actual patient, generally proceeding in steps from higher to lower levels of integration. Scenes of the examination are supplemented by animation depicting the underlying anatomy of the relevant portions of the nervous system. The examination covers cerebral function, the cranial nerves, cerebellar function, the motor system, the sensory system, and deep and superficial reflexes. The examination can take place in the physician's office, at the hospital, or at the patient's bedside.

Recent Deaths

DR. G. C. DAVIS

Dr. George Calvin Davis, 56, a Corpus Christi anesthesiologist, died April 18, 1966, in Corpus Christi.

The son of two physicians, Drs. George H. and Lillian Gregory Davis, Dr. Davis was born Nov. 8, 1909, in Kirkland, Wash.

After receiving a bachelor of arts degree from the University of Washington in Seattle, he received his M.D. degree in 1944 from St. Louis University School of Medi-

DR. DAVIS

cine in St. Louis, Mo. He held an internship at King County Hospital in Seattle and a residency at the University of Washington School of Medicine in Seattle, specializing in anesthesiology.

After maintaining a general practice in Kirkland, Wash., from 1949-1954, he specialized in anesthesiology, returning to practice in that field from 1957 to 1964 before moving to Corpus Christi where he practiced until his death.

During World War II he served as a captain in the US Army at Fort Knox, Ky.

A member of the Nueces County Medical Society, Texas Medical Association, and American Medical Association, Dr. Davis previously belonged to the King County Medical Society in Seattle and was secretary of the Eastside Medical Society in Kirkland. He also was a member of the American Society of Anesthesiologists, the Masonic Lodge, the Elks, and Lambda Chi Alpha fraternity. In Kirkland he belonged to the Rotary Club.

Surviving are his wife, the former Grace E. Toohey, and a sister, Mrs. Lilly May Davis Anderson of Kirkland, Wash.

DR. C. L. TUBB, SR.

Dr. Cullen L. Tubb, Sr., 68, died May 16, in Dallas. The cause of death was a heart attack.

He was born Nov. 22, 1897, in Valley Mills, Tex., the son of J. C. and Alma Barnett Tubb. He was graduated from Valley Mills High School and attended Trinity University, then located in Waxahachie, and the University of Texas in Austin. He received his M.D. degree from Vanderbilt University School of Medicine in 1923. After externing at Nashville Metropolitan General Hospital, he interned at Parkland Memorial Hospital in Dallas. He took postgraduate courses at Cook County Hospital in Chicago.

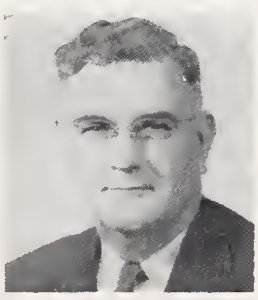
A specialist in industrial medicine, Dr. Tubb practiced in Valley Mills, Wortham, Corsicana, and Arp before moving to Dallas in 1942. He was a seaman apprentice in the US Navy before entering medical school.

Dr. Tubb was a member of Dallas County Medical Society, Texas Medical Association, American Medical Association, Texas Heart Association, Southwest Clinic Association, and an associate member of the American College of Chest Physicians. A Mason, he was a member of Phi Beta Pi medical fraternity, and the American Legion.

On June 17, 1924, he married the former Sarah Elmore Chapman in Waxahachie. She survives as do two sons, Cullen L. Tubb, Jr., and James C. Tubb, all of Dallas; a daughter, Mrs. Sarah McCaffrey of New York City, and his mother, Mrs. J. C. Tubb of Valley Mills.

Dr. Tubb was on the staffs of St. Paul's and Medical Arts Hospitals. He was on the board of directors of Reliance Life and Accident Insurance Company of America and was medical director for the Sun Oil Co., Southwest Division.

DR. TUBB



DR. E. D. SHIPMAN

Dr. Edward D. Shipman, 83, died June 1, 1966, in San Antonio, of lung abscess and generalized arteriosclerosis, particularly coronary sclerosis. He practiced in San Antonio from 1918 until his retirement in 1958.

Born in Gatesville on Nov. 12, 1882, the son of Daniel E. and Allie Grider Smith Shipman, he was a descendant of Moses Shipman, an organizer of the Austin colony of 300 pioneers who settled in Texas.

Dr. Shipman attended schools in Gatesville and San Angelo Methodist College. He received his M.D. degree from the University of Texas Medical Branch in Galveston in 1910. He practiced in a village in the state of Coahuila, Mexico, from 1910 to 1911; Del Rio, Tex., from 1911 to 1912; and Purmela, Tex., from 1912 to 1917. He served in the US Army Medical Corps as a first lieutenant in 1918. For 20 years he was on the medical staff of the Southern Pacific Co.

A former assistant city health officer in San Antonio, Dr. Shipman belonged to the Bexar County Medical Society, Texas Medical Association, American Medical Association, Texas Academy of General Practice, and the American Academy of General Practice. He also belonged to Phi Beta Pi medical fraternity.

Dr. Shipman was a member of St. Mark's Episcopal Church.

He held active memberships in the Perfect Union Masonic Lodge, Scottish Rite, Chapter and Council of York Rite, and belonged to the Alzafar Temple in San Antonio. He was a member of the San Antonio Knife and Fork Club, the Sons of the Republic of Texas, American Legion, Disabled American Veterans, and Woodmen of the World.

He is survived by his wife, Ida Frances Pfeuffer Shipman, whom he married on April 11, 1918, in Temple; a son, Edward Daniel Shipman, New Orleans; two daughters, Mrs. F. Dean Clift, and Mrs. F. P. Cunningham, both of San Antonio; two half brothers, A. Clyde Murrell, Okmulgee, Okla.; L. G. Murrell, Tulsa, Okla.; and two half sisters, Mrs. W. M. Strong, Shawnee, Okla.; and Mrs. H. K. Shelton, Dallas; and nine grandchildren.

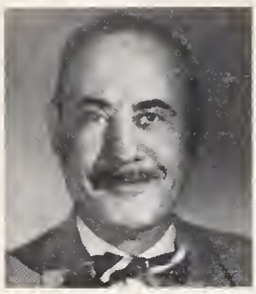
DR. SHIPMAN

DR. J. D. LANDESMAN

Corpus Christi city physician, Dr. Joseph D. Landesman, died on May 10 after a short illness. He was 64.

Born Aug. 6, 1901, in New York City, he received a degree in pharmacy from the University of Toledo, Toledo, Ohio, in 1924. He received his medical degree in 1933 from Wayne State University College of Medicine in Detroit, after interning at Toledo Hospital. His residency was served at Mt. Vernon State Hospital, Mt. Vernon, Ohio, from 1933 to 1935. He was a cardiologist with the Veterans Administration Hospitals from 1935 to 1943 serving in California, Milwaukee, Wisconsin, and Dallas.

After leaving Dallas in 1944, he moved to Corpus Christi, where he practiced until his death. He was plant physician for Pontiac Refining Corporation, serving in that capacity from 1958 until his death.



DR. LANDESMAN

He had been appointed city physician in March, 1966.

A fellow of the American College of Chest Physicians, he was a past president of the Nueces County Tuberculosis Association. He was a member of the Nueces County Medical Society, Industrial Medical Association, Texas Medical Association, and the American Medical Association.

Dr. Landesman was a past president of the Six Points Kiwanis Club, a past president of B'nai B'rith, and a past vice president of the board of Temple Beth El. He also served as chief of medicine at Memorial Hospital shortly after moving to Corpus Christi.

Survivors include his wife, the former Lillian Goldstein whom he married on Feb. 23, 1930, in Detroit; two sons, Dr. Dennis Landesman, a resident physician at the Veterans Administration Hospital in Memphis, Tenn.; and Dr. Harmon Landesman, a lieutenant in the US Navy Medical Corps, now serving at the Marine Hospital in Da Nang; a brother, Abe Landesman of Toledo, Ohio; a sister, Mrs. Rose Levine of Los Angeles; and three grandchildren.

DR. H. V. HEDGES

President-elect of the Private Clinics and Hospitals Association of Texas, Dr. Homer V. Hedges, 58, of Hico, was found dead on May 29 in his car about four miles south of Lampasas. Death was attributed to a heart attack. Dr. Hedges had been on his way to Seguin to visit his mother.

The son of Charles M. and Neta Shelton Hedges, he was born in Snyder on Aug. 28, 1907. After graduating from high school in San Antonio in 1924, he attended the University of Texas in Austin and received his bachelor of arts degree in 1930. He was graduated from the University of Texas Medical Branch in Galveston in 1933 and interned at Parkland Memorial Hospital in Dallas. He began practice in Hico in 1936, where he

DR. HEDGES

founded the Hico City Hospital in 1947.

A director of the First National Bank of Hico, he also had interests in farming and ranching. He was a former mayor of Hico, a school trustee, and a past president of the Chamber of Commerce there. Dr. Hedges was on the Heart of Texas Council of the Boy Scouts of America in Waco. A Mason, he was a member of Stephenville Commandery No. 100 K.T.; he also was a member of the Hico Civic Club.

A delegate from Erath-Hood-Somervell Counties Medical Society, he was a member of Texas Medical Association, American Medical Association, American Academy of General Practice, and a fellow of the International College of Surgeons. He belonged to Phi Chi medical fraternity.

He belonged to the Hico First Methodist Church. During World War II he served in the South Pacific as a captain in the US Army Medical Corps.

His wife, the former Emma Dee Hall of Hico whom he married Dec. 7, 1937, survives. Other survivors besides his mother and wife are a son, Alan C. Hedges, a student at the University of Texas; a daughter, Mrs. J. G. Beavan of Del Rio and San Carlos, Coahuila, Mexico; and two sisters, Mrs. V. L. Sherman of Seguin, and Mrs. A. A. Brooks of San Angelo.

DR. H. M. RAGLAND

Dr. Hugh Mayo Ragland, 58, a Gilmer general practitioner, died May 7 after a brief illness, in a hospital there.

A lifelong resident of Gilmer, he attended Gilmer public schools, Baylor University in Waco, and was graduated in 1933 from Baylor University College of Medicine, then located in Dallas. He interned at Harris Hospital in Fort Worth, and returned to Gilmer in 1934 to enter practice with his father, Dr. T. S. Ragland, and his brother, Dr. M. S. Ragland. He was an owner

DR. RAGLAND

of the Ragland-Fenlaw Clinic-Hospital.

Dr. Ragland was a past president of Upshur County Medical Society, and served several terms as secretary of the society. He had been president of the Northeast Texas Medical Society, a vice councilor of the Texas Medical Association (District 15), and a member of the American Medical Association.

A former Worshipful Master of Bethesda Lodge of Gilmer, he was a Royal Arch Mason and a member of Sharon Temple Shrine in Tyler and the Rotary Club of Gilmer. He belonged to the First Baptist Church of Gilmer.

On Dec. 23, 1933, he married the former Marie L. Salter in Dallas. She survives as do a son, Hugh M. Ragland, II; a daughter, Mrs. Mary Lou Davidson; his mother, Mrs. Mamie Denson Ragland; a brother, Dr. M. S. Ragland, all of Gilmer; two sisters, Miss Cecile Ragland, Gilmer, and Mrs. Curtis Finley, Lufkin.

DR. J. A. HOCKADAY

Dr. James Addison Hockaday, a practicing physician in Port Isabel, Tex. for 47 years and a civic leader in the Lower Rio Grande Valley, died of internal hemorrhage on May 10, 1966, in the Mercy Hospital at Brownsville, Tex.

Dr. Hockaday was born Jan. 23, 1892, at Plattsburg, Mo., the son of James Addison and Nana Elliott Hockaday. He attended schools at Holt, Mo., and the University of Missouri at Kansas City, Mo. He received his medical degree from the University Medical College of Kansas City, Mo., and, at the age of 21, interned at the University Hospital in Kansas City, Mo.

In 1914, he began practicing medicine in an area of small mining camps in Camden, Mo., and Satanta, Kan. He served about 18 months as lieutenant in the Medical Corps during World War I. After the war he moved his family

to Port Isabel, where he resided for the remainder of his life.

Dr. Hockaday married Clara Achterberg in Kansas City, Mo., in 1913. They had one son, James Achterberg. Clara died in 1915 and James was killed in a car accident near Port Isabel when he was 15. In 1917, Dr. Hockaday married Ruby Dietz, and they had two sons, Richard Sterling and William Burns. Both Ruby and William are deceased. His marriage to Buelah Lee Rankin in 1935 ended with her death in 1937.

In 1940, Dr. Hockaday and the former Jimmie Lee Jordan were married. She, their two sons, Donald Lee and Robert Edwin, his older son Richard, and six grandchildren survive.

Dr. Hockaday was a member of the Texas Medical Association through the Cameron-Willacy Counties Medical Society. He was president of the society in 1922. He also belonged to the American Medical Association. He had served with the US Public Health Service, the county and state health departments, and was physician and surgeon for the Missouri-Pacific Lines for many years. He reportedly delivered more than 5,300 babies, including 24 sets of twins and two sets of triplets.

He was a fishing enthusiast and had authored numerous articles on South Texas history and sports, especially big game fishing in the Western Hemisphere. He was founder and lifetime director of the Texas International Fishing Tournament. He was recognized for his interest in oceanography, ornichology, taxidermy, genealogy and archeology—especially with respect to the life and history of the area in which he lived. He was a member of the Texas Historical Association, Texas Historical Foundation, Cameron County Historical Survey Committee, and past president and lifetime board member of the Lower Rio Grande Valley Historical Society.

He is listed in the National Social Directory, Who's Who in the South and Southwest and has a chapter devoted to him in Leonard King's "Port of Drifting Men." He had almost completed drafts for two books which he planned to have printed when he retired—one a history of Port Isabel which dates back to 1519 and the other on Indians of the Lower Rio Grande Valley. His files of historical data will be included in a memorial library planned for Port Isabel.

Dr. Hockaday was mayor during 1946-1947 and 1956 to April, 1966.

Both Catholic and Protestant churches participated in his funeral services. He was given a military burial by the Veterans of Foreign Wars.

DR. J. V. BLAIR

Dr. John V. Blair, 74, retired Corpus Christi physician, died on April 7, 1966, in Kerrville after a long illness.

Dr. Blair was graduated from the University of Oklahoma School of Medicine in 1921 and served three years' internship at the University Hospital in Oklahoma City, where he was a resident physician in surgery.

After postgraduate work in New York he opened his medical practice in Corpus Christi in 1928, where he practiced 38 years until his retirement in January, 1965, because of ill health.

Dr. Blair was a member of the Nueces County Medical Society, Texas Medical Association, American Medical Association, and the Southern Medical Association.

He was a member of the Corpus Christi Yacht Club and a past commodore. He was a charter member of the Yachting Club of America. Before World War II he organized the US Coast Guard Auxiliary at Corpus Christi—Flotilla 71. He had been vice commodore of the 8th District Coast Guard Auxiliary. He was very much interested in churches and charitable organizations. He was a member of the Shidler Lodge of Shidler, Okla., from which he had just received his 40th year membership pin. He also belonged to the Scottish Rite Masons of Guthrie, Okla., the Alzafar Shrine Temple of San Antonio, and the American Legion.

Surviving are his wife, Margaret, and a sister, Mrs. Florence Guild of Pawhuska, Okla.

DR. V. R. LAZO

Dr. Vicente Roldan Lazo, 60, Laredo ophthalmologist, died Feb. 20, 1966, two days after suffering a stroke at his home in Laredo.

Dr. Lazo was born in San Vicente, Ilocos Sur, Philippines, on Feb. 1, 1906, to Pedro and Augustina Roldan Lazo.

After graduating from high school in Vigan, Ilocos Sur, he left the Philippines to study at Lewis Institute in Chicago. He received a bachelor of science degree from Indiana University in Bloomington and his M.D. degree in 1929 from Indiana University School of Medicine, Indianapolis. He held internships at Mercy Hospital, Bay City, Mich., and St. Francis Hospital, Grand Island, Neb.

Dr. Lazo practiced general medicine and surgery in Indiana until 1947, when he returned to the Philippines to join his brother, Dr. Marci Lazo, in opening their own hospital. In 1950 he returned to Indiana where he practiced until 1955, after which he began the study of ophthalmology, serving residencies at Philadelphia General Hospital and Metropolitan Hospital in New York City.

He opened his ophthalmology practice in Gary, Ind., in 1958, moving in 1965 to Laredo.

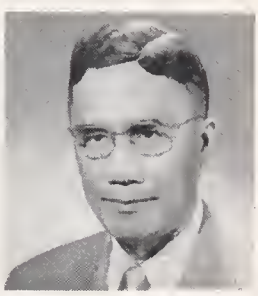
Dr. Lazo was a member of the Webb-Zapata-Jim Hogg Counties Medical Society, Texas Medical Association, and American Medical Association. He also had belonged to Sullivan County, Lake County, and Knox County (Indiana) Medical Societies, and to the Indiana State Medical Association.

He is survived by his wife, the former Alice Bennett; and three brothers, Dr. Catalino R. Lazo and Emil R. Lazo of San Vicente, and Dr. Marci R. Lazo of Mindanao, Philippines.

DR. HOCKADAY

DR. BLAIR

DR. LAZO



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COMING MEETINGS

American Medical Association, Clinical Meeting, Las Vegas, Nov. 27-Dec. 1, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Austin, Public Relations Conference, Oct. 1, 1966; Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

JULY

Postgraduate Medical Assembly of South Texas, Houston, July 18-20, 1966. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

AUGUST

American Hospital Association, Chicago, Aug. 29-Sept. 1, 1966. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

Texas Division, American Cancer Society, Austin, Aug. 2-3, 1966. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966, Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 8-10, 1966. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill., Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. G. Whittlesey, 219 E. 42nd St., New York, Sec.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Gastroenterology, Philadelphia, Oct. 23-26, 1966. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, Dr. Robert A. Peterman, 3 Craig Court, Totowa Borough, N. J., Sec.

American College of Obstetricians and Gynecologists, Houston, Oct. 24-26, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, February, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American College of Surgeons, Annual meeting, San Francisco, Oct. 10-14, 1966. Dr. Samuel P. Harbison, 230 Lothrop St., Pittsburgh, Sec.

American Congress of Physical Medicine and Rehabilitation, San Francisco, Aug. 28-Sept. 2, 1966. Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., Sec.

American Gynecological Society. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, New York City, Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Medical Women's Association, Washington, D.C., Nov. 2-5, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Philadelphia, Oct. 1-5, 1966. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Washington, D.C., Sept. 16-24, 1966. Dr. Lester H. Hoyt, Methodist Hospital, Indianapolis, Ind. 46207, Sec.

American Surgical Association. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis, Ind., Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Chicago, Nov. 4, 1966. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104 Sec.

Gastrointestinal Research Group, Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Private Clinics and Hospitals, Dallas, Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La., Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec., 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore, Md.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston, Pres.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of General Practice, Houston, Sept. 12-14, 1966. Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006. Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Heart Association, Houston, Sept. 16-18, 1966. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Fort Worth, Sept. 29-Oct. 1, 1966. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Surgical Society, Houston, Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

United States-Mexico Border Public Health Association, Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Spring, 1966. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

Blackford Memorial Lectures, Denison, Sept., 1966. Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Southwest Regional Cancer Conference, Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

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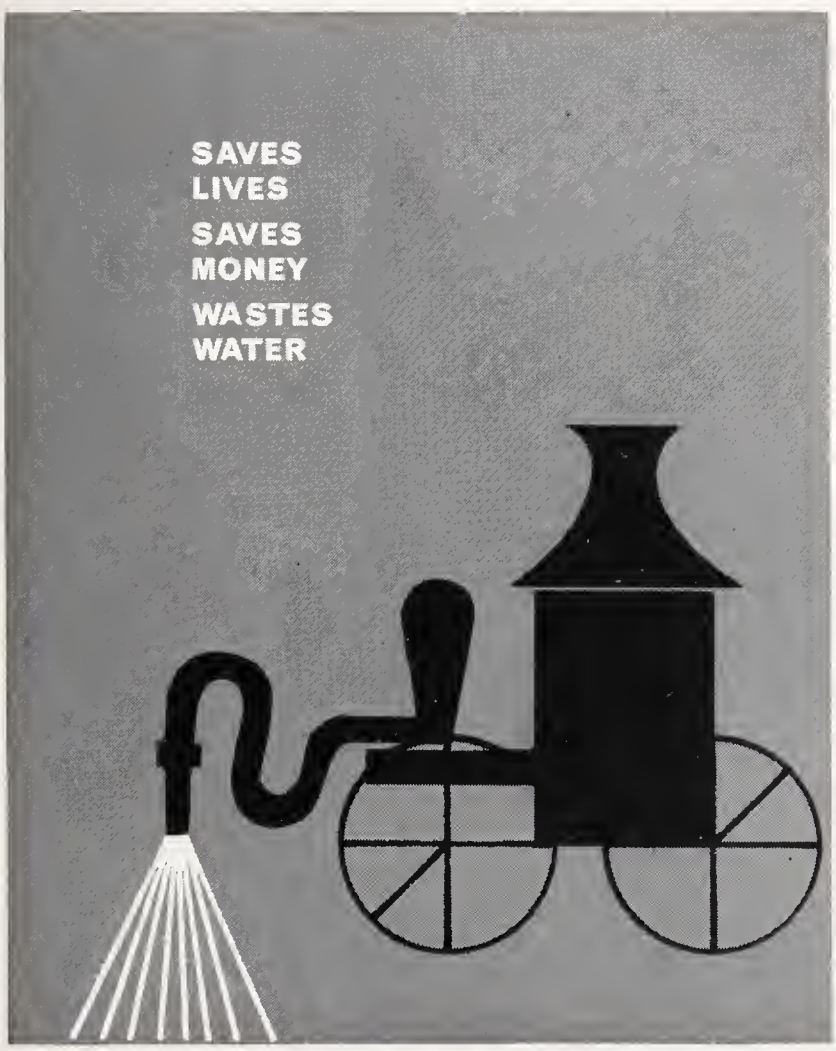

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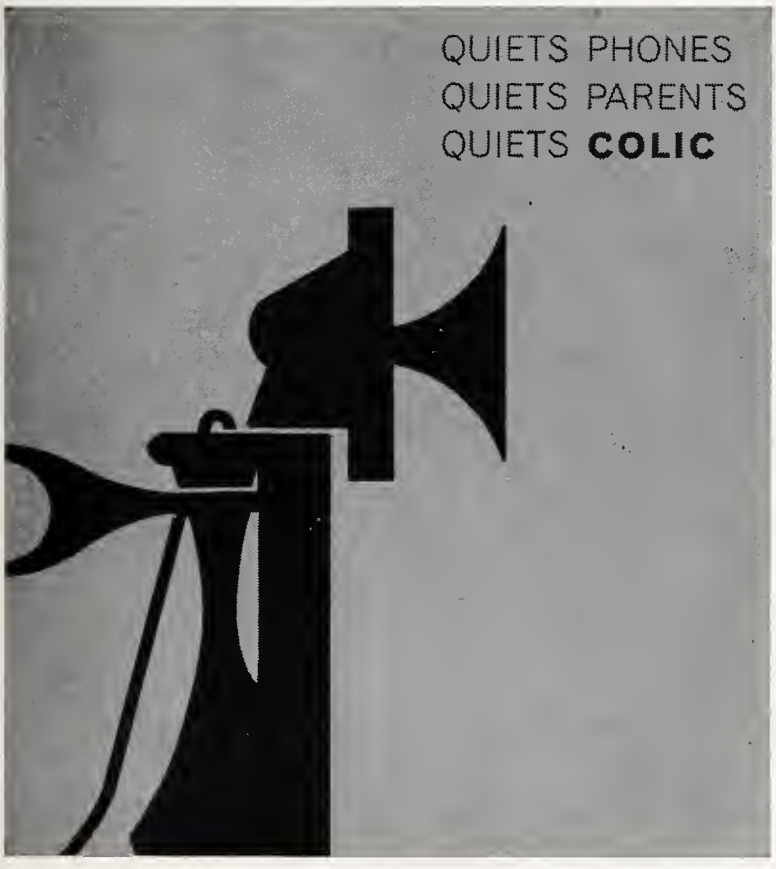

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Side effects: Drowsiness may oecur and, rarely, ataxia, usually controlled by dccreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maeulopapular rash. Acute nonthromboeytopenie purpura with peripheral edema and fever, transient leukopenia, and a single ease of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fcver, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proetitis. Treatment should be symptomatie in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: Onc or two 400 mg. tablets thrce times daily. Doses above 2400 mg. daily are not reeommended.

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
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Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

If a reaction or overgrowth of nonsusceptible organisms occurs, withdraw the drug.

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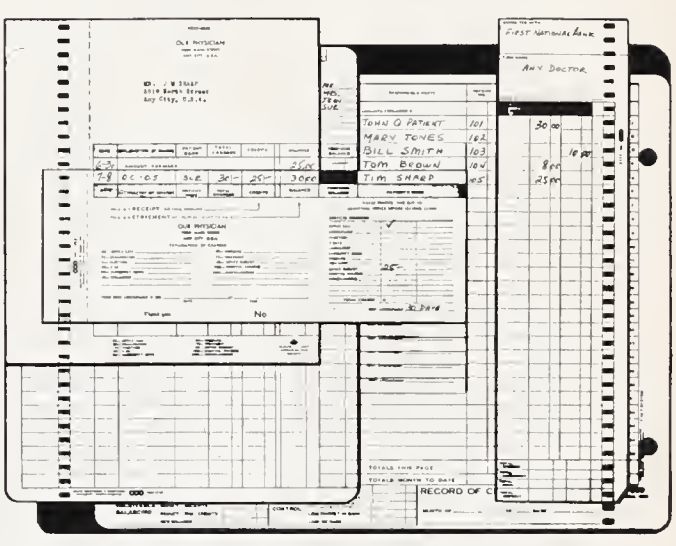

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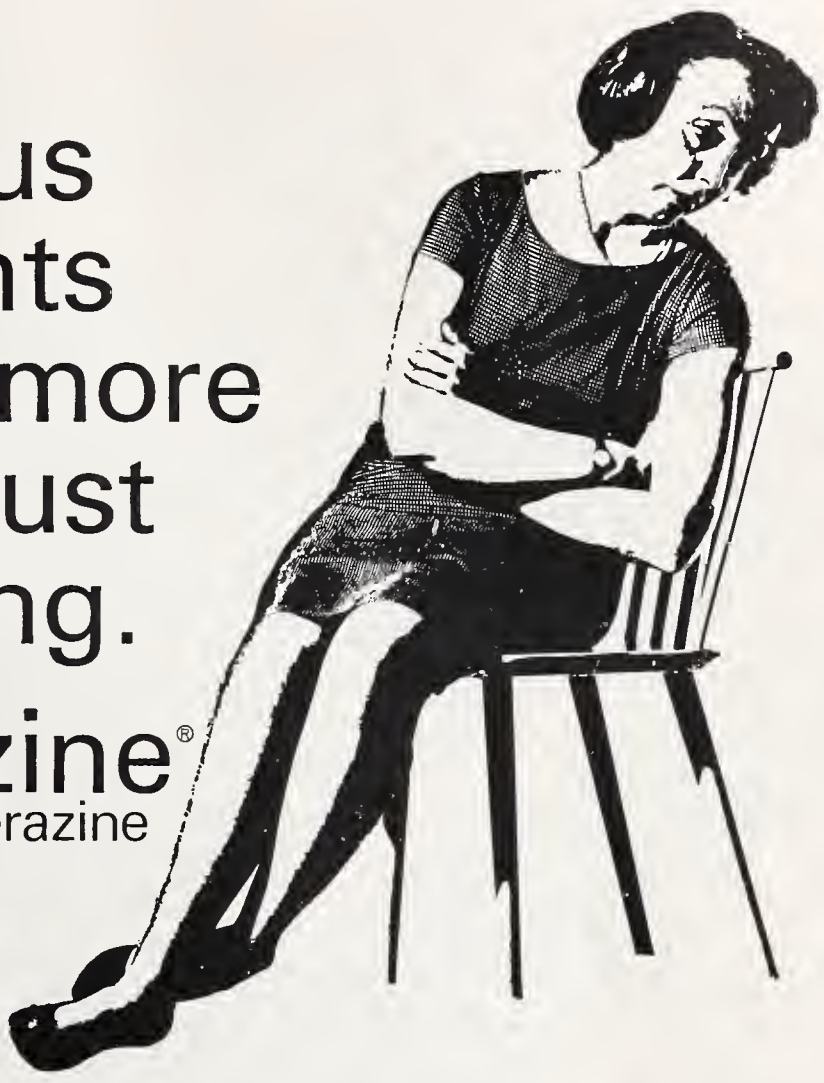

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Meprobamate: Careful supervision of dose and amounts prescribed is advised; especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of pre-existing symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dose—operation of motor vehicles, machinery or other activity requiring alertness should be avoided. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

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Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.

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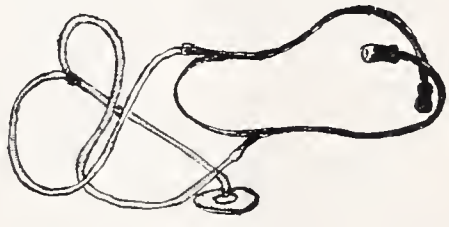

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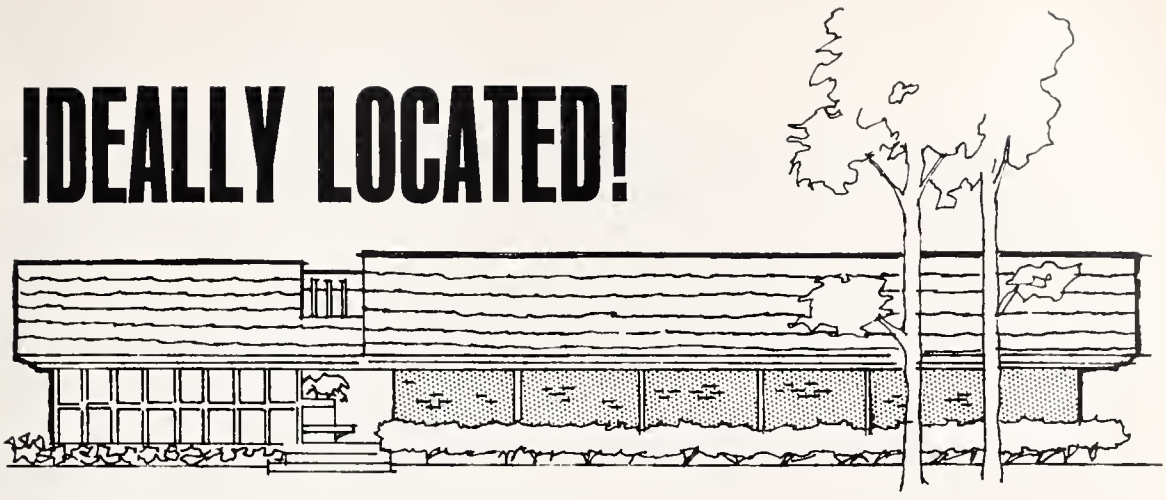

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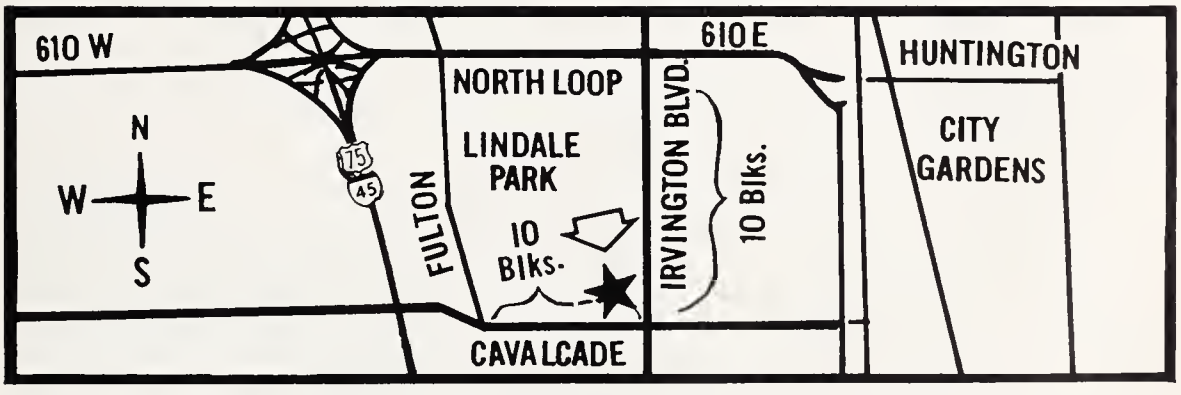

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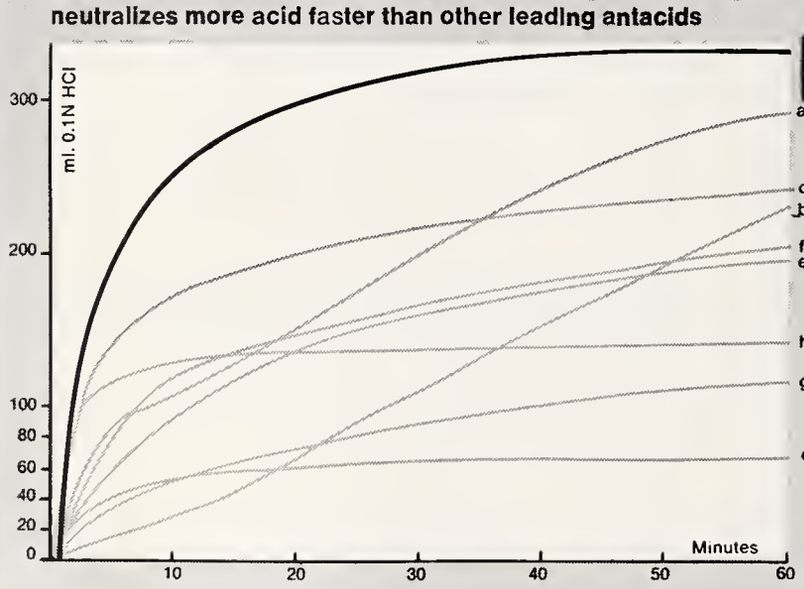

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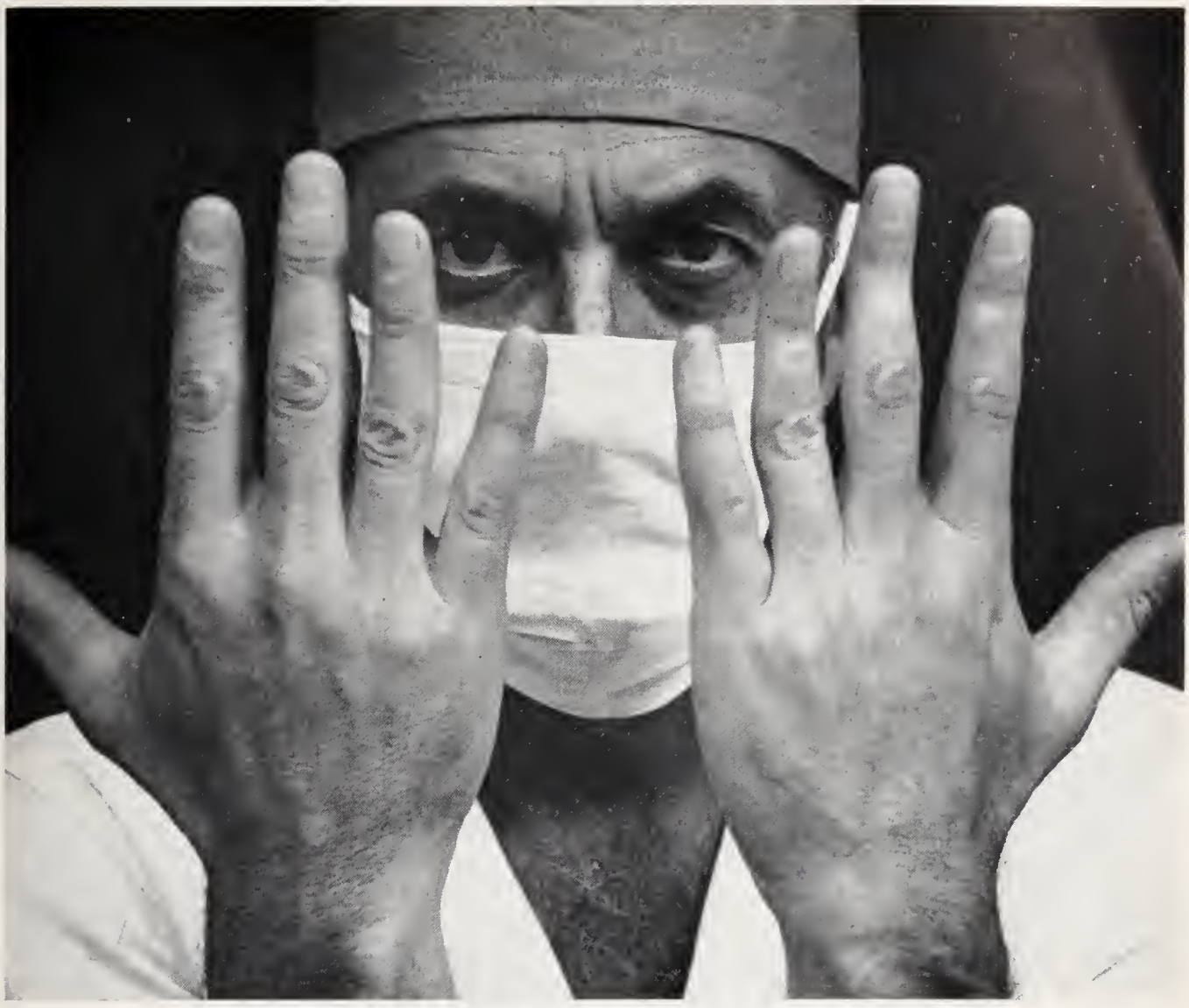

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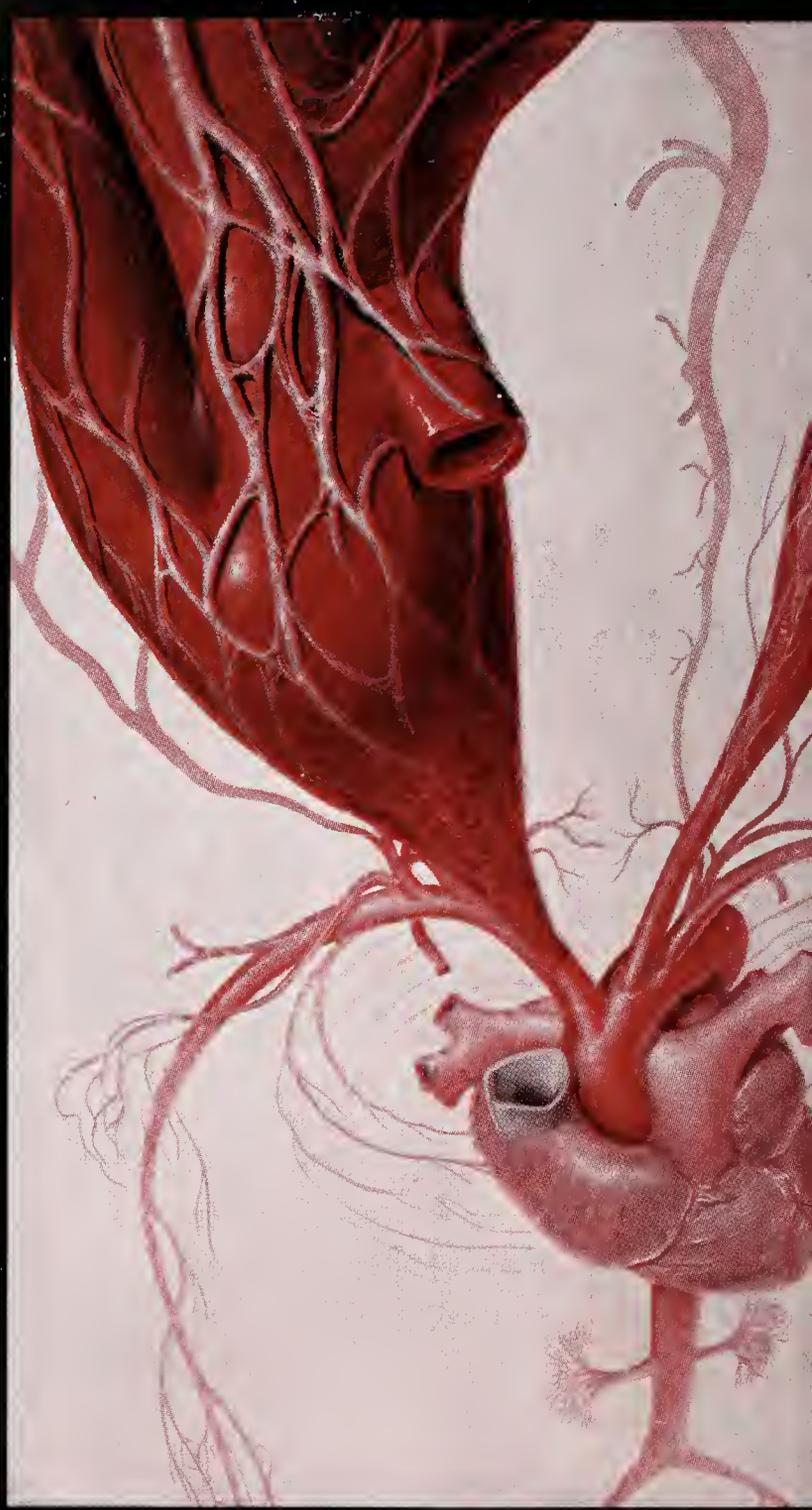

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References: 1. Editorial: J.A.M.A. *189*:691 (Aug. 31) 1964. 2. Foertsch, J. H.: J. Oklahoma Med. Assn. *57*:449 (Oct.) 1964. 3. Paul, H. E., and Paul, M. F.: The Nitrofurans—Chemotherapeutic Properties, in Schnitzer, R. J., and Hawking, F. (Eds.) Experimental Chemotherapy, Vol. 2, New York, Academic Press, 1964.

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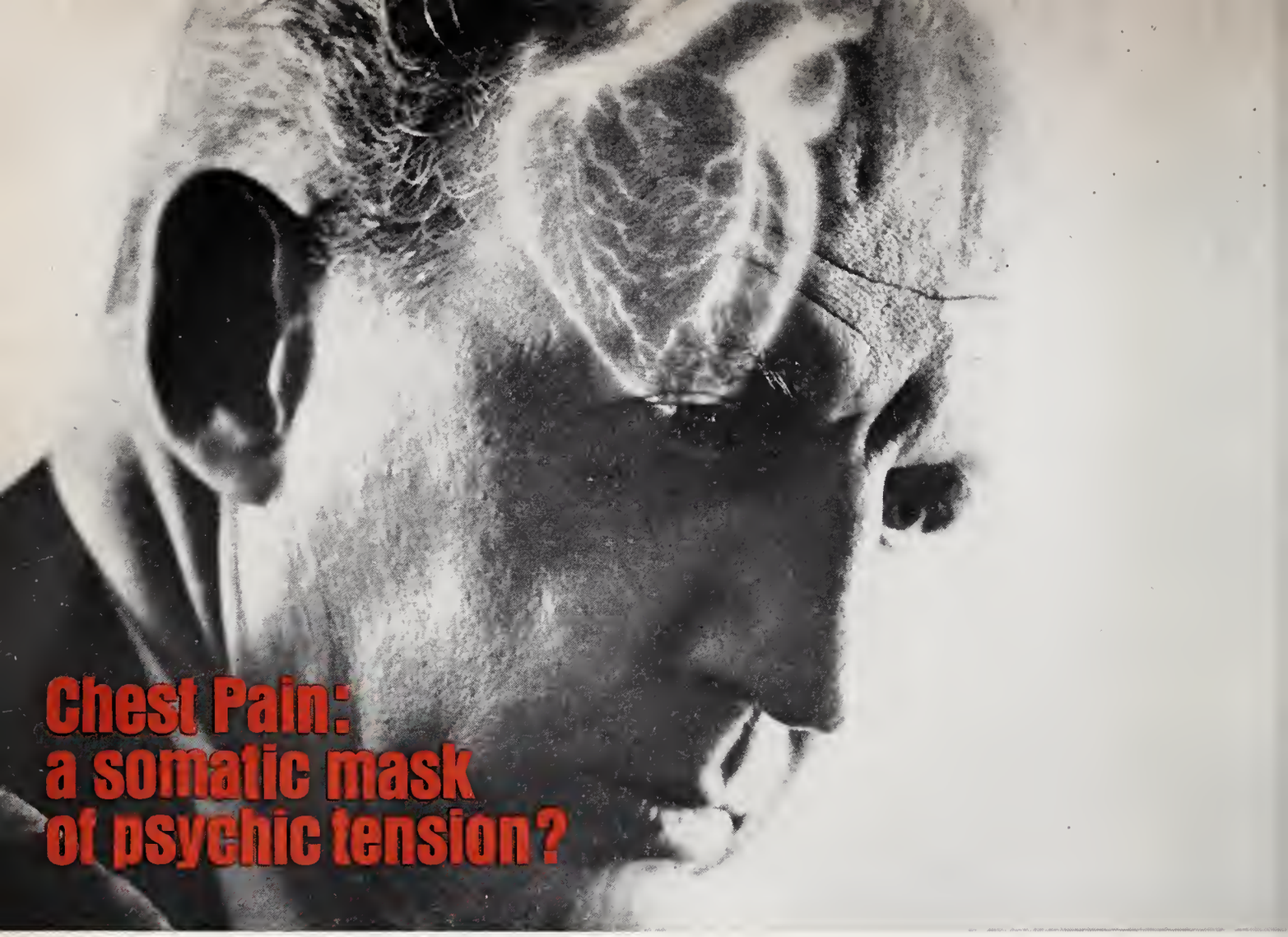

Chest Pain: a somatic mask of psychic tension?

"Heart symptoms"—chest pain, tachycardia, arrhythmia—invariably alarm and preoccupy the patient, though they may be completely without organic basis. Such symptoms often are somatic masks of psychic tension, arising from constant encounters with stressful situations.

When the problem is diagnosed as emotionally produced, consider Valium (diazepam) as adjunctive therapy. Valium (diazepam) acts rapidly to calm the patient, to reduce his psychic tension and relieve associated cardiovascular complaints.

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Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

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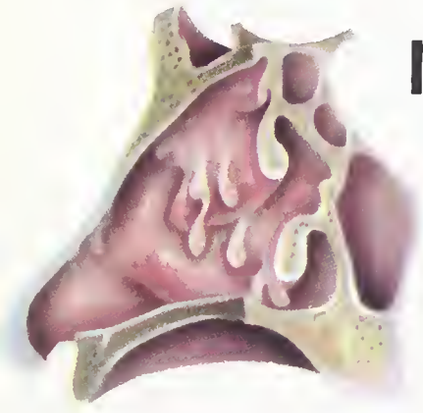
this issue: emergency anesthesia and the common cold

When emergency anesthesia is complicated by the common cold

Barry Belonsky, M.D., F.A.C.A.
Staff Anesthesiologist, Hospital of The Albert Einstein College of Medicine, New York City



Medical facilities are often presented with unfamiliar patients who have unknown health histories. This is particularly true in emergency situations that arise due to accidents or acute illnesses. These cases may need prompt care requiring anesthesia, and if they involve colds, nasal allergies or other upper respiratory infections, can account for many complications which make up a major hazard during emergency anesthesia.

Administration of general anesthesia to a patient with a cold or upper respiratory infection is a hazardous undertaking. It should be avoided if at all possible. Indeed, the presence of U.R.I. is good reason for postponement of elective surgery.[1] In emergency surgery, regional or local block should be considered, but if general anesthesia is mandatory, it should be approached with utmost caution.

Since the attitude of "emergency surgery — hurry" has been replaced by "emergency surgery — watch out",[2] a knowledge of the complications is a great help in preventing them. Here is a brief outline of the problems involved and their treatment. Prevention of the complications is discussed later.

Complications during the induction of anesthesia

Most of the complications are a direct result of secretions and some a result of accompanying secondary infection. For example, *airway obstruction* due to excessive secretions occurs very commonly and is the direct effect of the cold. Respiratory exchange may be obstructed at any time during anesthesia because of excessive secretions, but is most likely to occur during induction. Suction apparatus must be available to overcome this.[3]

Excess secretions which stimulate and irritate the epiglottis and vocal chords can cause *laryngeal stridor and obstruction*. This can lead to complete laryngeal closure with resultant anoxia and death.

Bronchospasm and laryngospasm can result from secretions penetrating the bronchi and bronchioles. In laryngospasm, there are both inspiratory and expiratory stridor and difficulty in inflating the chest; in bronchospasm there is an expiratory wheeze, but not as much difficulty in inflation, although some resistance may be felt. Stridor is due to partial or complete closure of the vocal cords in spasm and the "crowing" sound is almost pathognomonic.

Secretions obstruct the nasal airways. This produces *difficulty in ventilation* through the mouth until the patient is deep enough to place an oral airway. An intravenous agent can be given to facilitate the induction of anesthesia.

Difficulties can arise if intubation is performed to ventilate the patient. For example, teeth can be broken by too vigorous attempts at intubation, or the intubation itself may be technically difficult due to secretions obstructing the view of the glottis. The postoperative sequelae of intubation ranges from mild laryngitis to pneumonia with atelectasis, and are seen far more commonly in patients suffering from colds than in normal patients.

Successive stages of laryngospasm which produce the characteristic stridor or "crowing" sound.

Progression of bronchioles into bronchospasm.

Complications during the maintenance of anesthesia *Bronchospasm* can occur in an unintubated patient due to secretions entering the bronchial tree from above, and acting as an irritant to the bronchi and bronchioles. Secretions accumulate quickly and the patient has to be suctioned continually. The whole cycle of coughing, bucking, laryngospasm and bronchospasm may ensue. The difficult decision here is whether it is better to suction the patient continually or to use an endotracheal tube which protects the cords and bronchi but introduces the risk of attendant complications.

Postoperative complications Postoperatively, complications can be more serious than even the intra-anesthesia complications, and occur much more frequently in a patient who has been intubated.[4]

Sore throat and pharyngitis can result both from the preoperative upper respiratory infection and from the drying of the mucous membranes which occurs during anesthesia.

Tracheitis and bronchitis often result from secretions trickling down the tracheobronchial tree.

Laryngitis is frequently seen in patients with upper respiratory infections who have been intubated. There is a significant increase in the incidence of laryngitis compared to that in patients without upper respiratory infections.

Subglottic edema is a condition which occurs mainly in children who have been intubated. This pathology results from an exudate developing in the areolar tissue just below the cords. Because of the small size of the child's trachea, even a 1 mm increase in size of the mucous membrane can severely impair the air passage. Children exhibit this by severe expiratory stridor and may even become cyanotic. This may so severely embarrass the child's breathing that it must be treated vigorously. Most authorities agree on the treatment[5,6,7,8] consisting of a high oxygen concentration in the inspired air (60%), plus high humidity (close to 100%). Adequate parenteral fluid intake and slight cooling of the body temperature (by a cooled oxygen tent) also help in mild cases. In severe cases, there may be hypoxia which increases the restlessness and the oxygen demand rises. Sedation is often necessary, although concomitant depression of the respiratory center is undesirable. An antihistaminic accomplishes this purpose well, and adds sedation. Since there is always a possibility that an allergic response plays a role in edema, some relief of the respiratory distress may occur. Steroids should be used to control inflammatory and allergic phenomena and swelling. If all this fails, and the patient is still restless and hypoxic, a tracheostomy should be performed immediately.

(concluded on following page)

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Cross section of trachea showing subglottic edema and lumen reduction due to mucous membrane congestion.

Pneumonia may also follow anesthesia administered to a patient with a cold. This can be caused by accumulated secretions becoming secondarily infected and causing consolidation of the lung. *Atelectasis* of the lung can result if one of the bronchioles becomes plugged by secretions, preventing aeration of the distal part of that lung. This is seen more frequently following upper respiratory infection because dry anesthetic gases aggravate the infection, causing secretions to change from watery to thick and viscid, and consequently difficult to suction.

Prevention of complications The first rule to prevent complications, of course, is to use a regional or local anesthesia whenever possible. But when emergency surgery is a must, in spite of the presence of a cold, allergy, or upper respiratory infection, here are some ways to prevent complications.

Give nose drops preoperatively. This can help shrink the congested nasal mucous membranes and reduce secretions for better air passage. (Results of this method are sometimes unsatisfactory because of the short duration of effect or rebound congestion.) For longer effect, oral antihistamines with nasal decongestants are often given to provide and maintain a drying effect on secretions.

To clear the tracheobronchial tree, instruct the patient to cough preoperatively. Cold steam or water nebulizers effectively humidify the nasal, pharyngeal and bronchial passages and often make the patient more comfortable. Tenacious secretions become more watery under humidification, clear more thoroughly preoperatively and are more easily suctioned from the airway during anesthesia.

Give intravenous fluids to those patients who appear dehydrated due to a cold. In a well hydrated patient the respiratory tract secretions are less viscid and more watery. This is particularly true in asthmatics.

Summary: Administration of emergency anesthesia to a patient with a cold or upper respiratory infection can lead to a chain of events that may result in increased postoperative morbidity and even death. This is because of the excess secretions formed in these conditions. Preoperative measures to prevent or reduce these secretions should be undertaken and will result in smoother and safer anesthesia.

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Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

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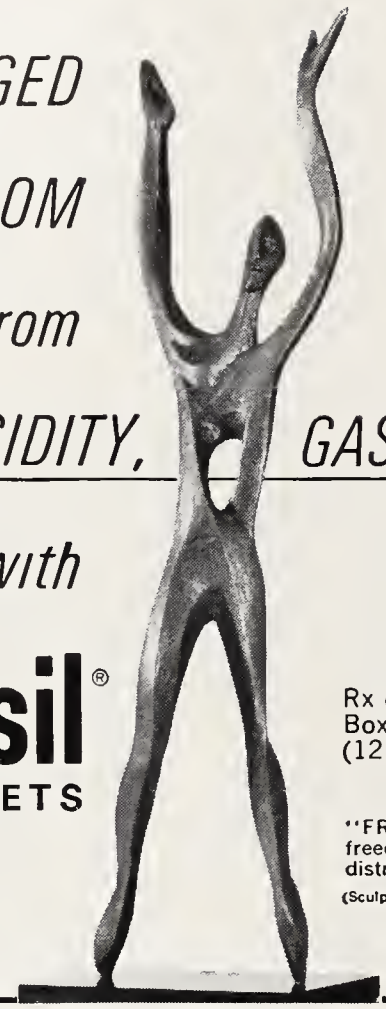

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
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Behind continued high blood pressure readings lies the possibility of organic damage[1-13]

MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.

"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

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Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

References: 1. Page, I. H., and Dustan, H. P.: The Usefulness of Drugs in the Treatment of Hypertension, in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 95. **2.** Hollander, W.: The Evaluation of Antihypertensive Therapy of Essential Hypertension in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 97. **3.** Nickerson, M.: Antihypertensive Agents and the Drug Therapy of Hypertension, in Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 3, New York, The Macmillan Co., 1965, p. 727. **4.** Berkson, D. M.: Indust. Med. & Surg. **32**:371, 1963. **5.** Cohen, B. M.: M. Times **91**:645, 1963. **6.** Lee, R. E., et al.: Am. J. Cardiol. **11**:738, 1963. **7.** Moyer, J. H.: Am. J. Cardiol. **9**:821, 1962. **8.** Moser, M.: New York J. Med. **62**:1177, 1962. **9.** Wood, J. E., and Battey, L. L.: Am. J. Cardiol. **9**:675, 1962. **10.** Moyer, J. H., and Heider, C.: Am. J. Cardiol. **9**:920, 1962. **11.** Moser, M., and Macaulay, A. I.: New York State J. Med. **60**:2679, 1960. **12.** Judson, W. E.: Nebraska M. J. **44**:305, 1959. **13.** Hodge, J. V.; McQueen, E. G., and Smirk, H.: Brit. M. J. **1**:5218, 1961. **14.** Moyer, J. H., and Brest, A. N.: Hypertension Recent Advances, Philadelphia, Lea & Febiger, 1961, p. 633. **15.** Berry, R. L., and Bray, H. P.: J. Am. Geriatrics Soc. **10**:516, 1962. **16.** Reid, W. J.: J. Am. Geriatrics Soc. **13**:365, 1965. **17.** Feldman, L. H.: North Carolina M. J. **23**:248, 1962.

Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

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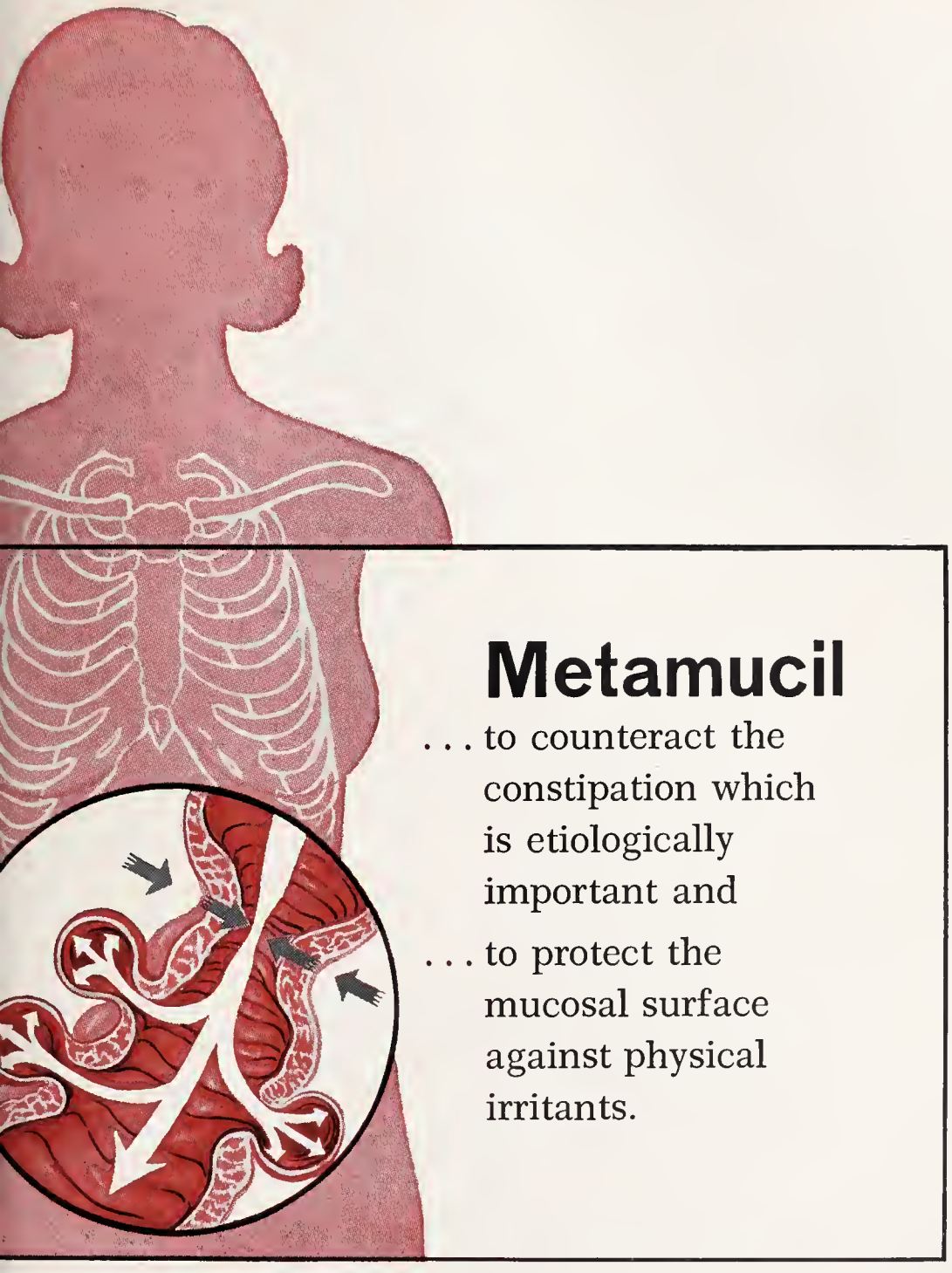

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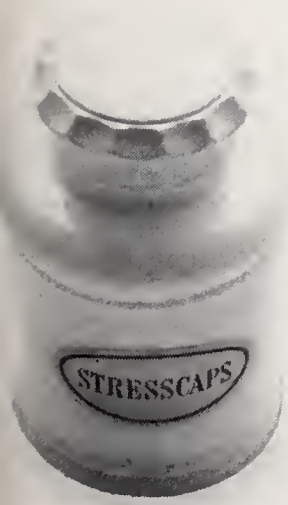

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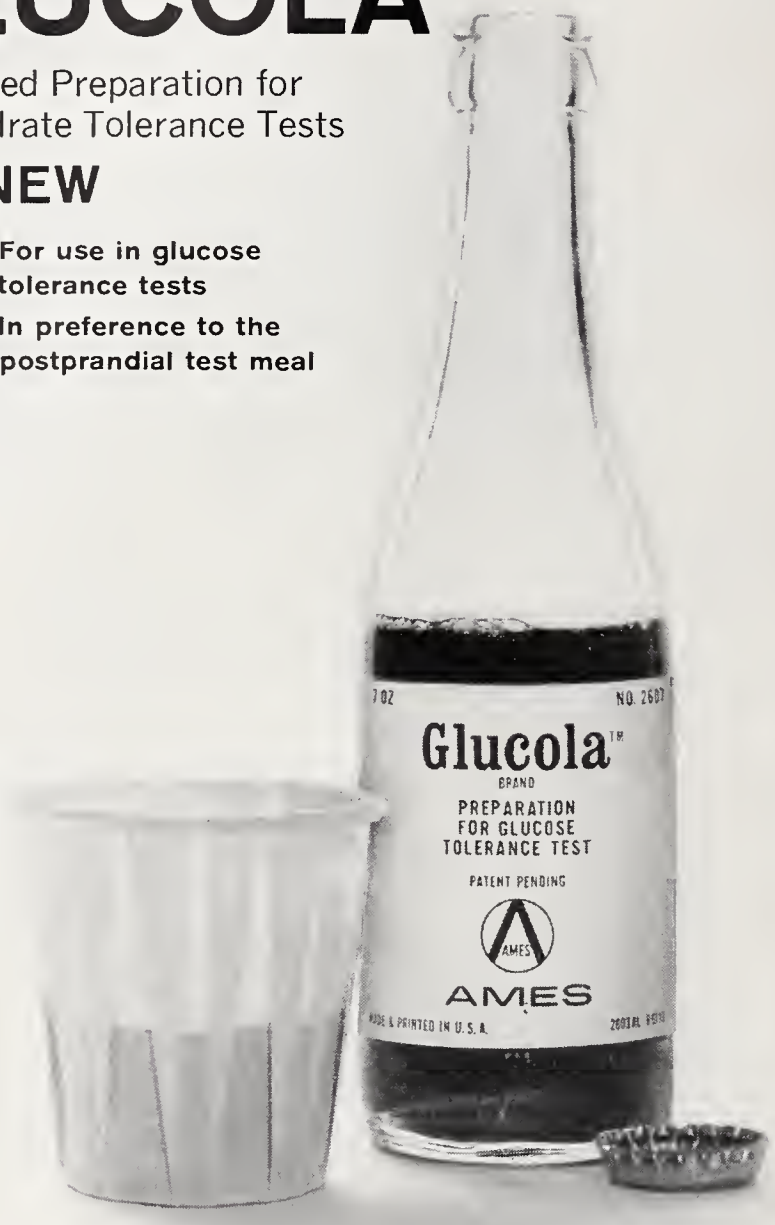

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(see previous page)

The Cancer Registry—A Vital Hospital Tool

THE INTEREST of the medical profession in the fight against cancer has been intensifying since 1895 when Roentgen discovered x-ray and foresaw its usefulness as a diagnostic aid.

In the United States, this discovery was noted so rapidly that it was reflected in medical reports and state medical society proceedings generally. Here at last was a way of determining the presence of cancer in otherwise obscure situations. The development of radium, x-ray therapy, radiation isotopes, and chemotherapy as therapeutic tools in addition to surgical excision has encouraged us to face cancer as a problem for which something can be done.

In 1913, two major events had an important effect on cancer programs throughout the United States; one was the establishment of the American College of Surgeons and the other was the activation of the American Cancer Society. By 1930, the American College of Surgeons had published a list of minimum requirements for cancer clinics. The most recent listing of cancer programs and registries by the College shows that 957 institutions in the U. S. have met these minimum standards for approval; 36 such institutions are in Texas.

Since Jan. 1, 1965, the American College of Surgeons has required the building and maintenance of a cancer registry as an essential feature for its approval of a hospital cancer program.

The term, "registry," has been used several ways. Sometimes it refers to a large medical center's cancer listing, used primarily for statistical research. It may also refer to special registries for certain tissues or it may be a registry used primarily for epidemiologic studies. The value of all types of registries is readily recognized, but the hospital cancer registry, as described in the "Manual for Cancer Programs" of the American College of Surgeons (1966), is of most value to the patient as well as the physician.

The fundamental purpose of a hospital's cancer registry is to bring to members of its

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staff information regarding their handling of cancer so they may provide the best care possible for cancer patients. This kind of care includes determining not only that the patient survived a certain number of years, but that he is also free from cancer. It also includes instituting therapy at the earliest possible moment should cancer recur.

Once this basic purpose (survival and end result) has been achieved, the use of the registry may be extended in many ways, depending on local circumstances and the desires of the medical staff. For example, a hospital may wish to compare the results of its treatment of cancer with the results obtained in other hospitals.

Other information provided by a hospital registry may be:

1. Total number of patients seen annually, categorized by site of cancer.
2. Number of primary lesions, as compared with recurrent ones.
3. Number of cases diagnosed in this hospital, compared with the number first diagnosed elsewhere.
4. Proportion of cancer cases microscopically confirmed.
5. Ratio between localized and advanced cases at time of diagnosis, and the change in this ratio year after year.
6. Results of diagnosis and treatment, expressed in survival rates.

The success of a hospital's cancer registry can be measured only by the interest of the medical staff in the results of their treatment of cancer, willingness of the medical staff to establish and support this method of bringing constant evaluation into reality, and concern of the medical staff that there be a method by which periodic reports on cancer patients can be issued to the staff. Only through medical staff involvement can a hospital cancer registry serve the purpose for which it was intended.

The Texas Medical Association's Committee on Cancer has repeatedly urged that all hospitals with a significant number of cancer patients build and maintain a cancer registry as another tool for improving patient care. Hospitals desiring assistance in establishing registries may address inquiries to the Committee on Cancer, Texas Medical Association, 1801 N. Lamar, Austin 78701.

—William S. Brumage, M.D.,
Austin, Director,
Division of Cancer and Heart Diseases,
Texas State Department of Health.

Fructrated or Frustrated

ARE YOU a frustrated physician having lately stamped out no diseases, recorded no cures, defeated no Medicare, discovered no grand panacea, Egyptian moly, or Greek elixir?

Do you deem yourself dishonorable, inadequate, ineffective, and a disgraceful failure, having replaced no kidney, heart, brain, congressman, nor embryo?

Are you ready for the day when medical science will have obtained that apex of perfection wherein it will be able to remove all the disease of man? Will you have a role when mankind, at last, reaches a point where the allotted span of life is achieved? Where will you be in this dream of universal medicine that has pervaded the nations of the great society of the earth since the days of Isis and Osiris? Will there be a niche for you when disease shall be banished from the globe in the glorious period of the millennium, or the grand catholicon, or by governmental order?

Have faith. There is hope for you. Salvation is here. The art of medicine will not be superseded by technology and the humanities of medicine will not be expunged by the impersonality of cookbook specialism. There will always be worldly birth, anxiety, injury, shame, ailments, adjustments, aging, uncertainty, loneliness, and death that will cry out for a physician with a "golden treasury" of humanism. The physician will always be fructuous as long as he "comforts always" even though he seldom "cures."

—Lee F. Scarborough, M.D., Austin.

Etiology, Symptoms Vary In Hypoglycemic States

AS OUR KNOWLEDGE of normal and pathological carbohydrate metabolism increases, we find new additions to the etiology of hypoglycemia.

Not too many years ago we were concerned mainly with the still prevalent causes, namely, overdose or over-response to exogenous insulin (oral hypoglycemic agents to a lesser extent); functional hypoglycemic reactions

in some emotional disorders; insulinomas, benign or malignant; and the so-called dumping syndrome after stomach surgery.

Since the clinical manifestations may be striking and confused with a variety of non-hypoglycemic disorders, it is timely, first, to refresh our memories of the signs and symptoms which can be produced by low blood sugar levels so that hypoglycemia may be considered in a given case, and second, to refresh or add to our knowledge the more recently discovered causes. To this end, etiologic accuracy will be increased and delay in proper therapy lessened.

By definition, hypoglycemic states exist when sugar concentration in the blood falls below 50 mg. per 100 ml. Hypoglycemia should be considered clinically whenever autonomic nervous system, central nervous system, and psychic symptoms and signs are present.

The familiar hyperinsulinism phenomena of agitation, anxiety, hunger, sweats, weakness, pallor, muscle tremor and coma need only be mentioned, but such manifestations as headache, blurred vision, spastic movements, slow reaction time and difficulty in comprehending, involuntary bowel and bladder movements, easy fatigability, palpitation, tachycardia and extrasystoles, uncontrollable yawning, depression, and transient monoplegia and hemiplegia must also alert us to consider hypoglycemia. If hypoglycemia is severe and prolonged, the tendon reflexes may be lost, Babinski reflexes may be present, bizarre neurologic signs may appear, and finally cerebral damage ensues.

Of the more recent entities, the earliest described and most likely to produce hypoglycemia are the fibrosarcomas and other mesenchymal tumors. Other types include epithelial tumors of the liver, adenoma and carcinoma of the adrenal glands, carcinoma of the cecum and bronchus, and Wilms' tumor.

The pathogenesis of low blood sugar levels in these conditions probably varies with the type. A reported case of hepatoma suggests a mechanism in which the tumor consumed more glucose than was released. Several other reports indicate that the tumor may secrete insulin or an insulin-like material, or may produce increased sensitivity to endogenous insulin.

In rare instances alcohol appears to be a primary exogenous cause of hypoglycemia. A recent review states that neither alcoholism nor chronic malnutrition is necessary for alcohol-induced hypoglycemia in a susceptible person, whereas in a normal individual, a fast of two or three days is necessary before alcohol can cause hypoglycemia; this indicates depletion of liver glycogen since glucagon did not correct the condition.

Other exogenous agents causing low levels of blood sugar are salicylates, manganese, and monoamine oxidase inhibitors, probably by causing catecholamine depletion and thus upsetting normal homeostatic mechanisms.

Parenchymatous diseases of the liver of whatever form, including extensive metastatic cancer, can create hypoglycemia when impaired gluconeogenesis or defective glycogen storage inhibit or prevent release of glucose into the blood stream.

Idiopathic hypoglycemia in infants is rare, but must be recognized early if cerebral damage is to be avoided. About one-third of these cases are caused by abnormal leucine sensitivity; this is familial, although the exact mode of inheritance is not known. Large amounts of c-leucine will make most people show hypoglycemia. In addition, some patients with islet cell adenomas show unusual leucine sensitivity which disappears with removal of the adenoma. Studies on the mechanisms of this sensitivity phenomenon in general suggest pancreatic and extrapancreatic activity of an unknown process.

Fructose intolerance may also cause hypoglycemia, despite normal total blood sugar levels, because the *glucose* level may be as low as 10 mg. per 100 ml. with the classic picture of hypoglycemia. This situation appears to result from a deficiency of liver aldolase.

Congenital galactosemia, glycogen storage disease, and maternal diabetes are additional rare causes of idiopathic hypoglycemia in infancy.

Finally, there have been recorded rare cases of factitious hypoglycemia, usually in psychotic or psychopathic individuals, and insulin overdose for suicide and homicide has been reported.

—David R. Sacks, M.D., San Antonio.

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Tetanus Prevention Belongs In Every Physician's Practice

MANY PEOPLE ARE NOT immunized against tetanus. While school children and young adults who have been in military service are usually protected, older persons, especially women and agricultural workers, may not have had their shots. Each year hundreds of people contract tetanus and about half of them die in spite of all we can do.

Tetanus is preventable. Tetanus toxoid, if given soon enough, produces excellent protection; one of the best vaccines, it is 99.9 percent effective when properly used. To rely on equine antitoxin is folly. The protection is inadequate. To many who are sensitive to horse serum, or become sensitized to it, the antitoxin is poison. Skin testing does not reveal all sensitivity and is not itself without danger of anaphylaxis.

Human immune tetanus globulin is relatively quite safe and effective. No skin test is needed. It is also rather expensive. Recently it has been scarce, and sometimes it is unobtainable.

Everyone who practices medicine, no matter what his specialty interests may be, should feel free to prevent tetanus by giving toxoid. At the same time, we should encourage the production of adequate amounts of human immune tetanus globulin from any available source. Hyperimmunization of donors requires six months' preparation of each donor. Participation in such a program is laudable and should be rewarded when possible. Penal institutions can provide ideal donor sources.

—HARRY A. TUBBS, M.D., Post.

[*Editor's Note:* A leaflet, "Immunize with Tetanus Toxoid? Yes! These Are the Reasons . . ." is available on request from the Texas Medical Association, 1801 N. Lamar Blvd., Austin 78701. Prepared by the Committee on Disaster Medical Care, it is suitable for bulletin boards, distribution to patients, and as an educational aid in industrial, community, and school health programs.]

letters to TEXAS MEDICINE

Possible Diagnosis: Leprosy

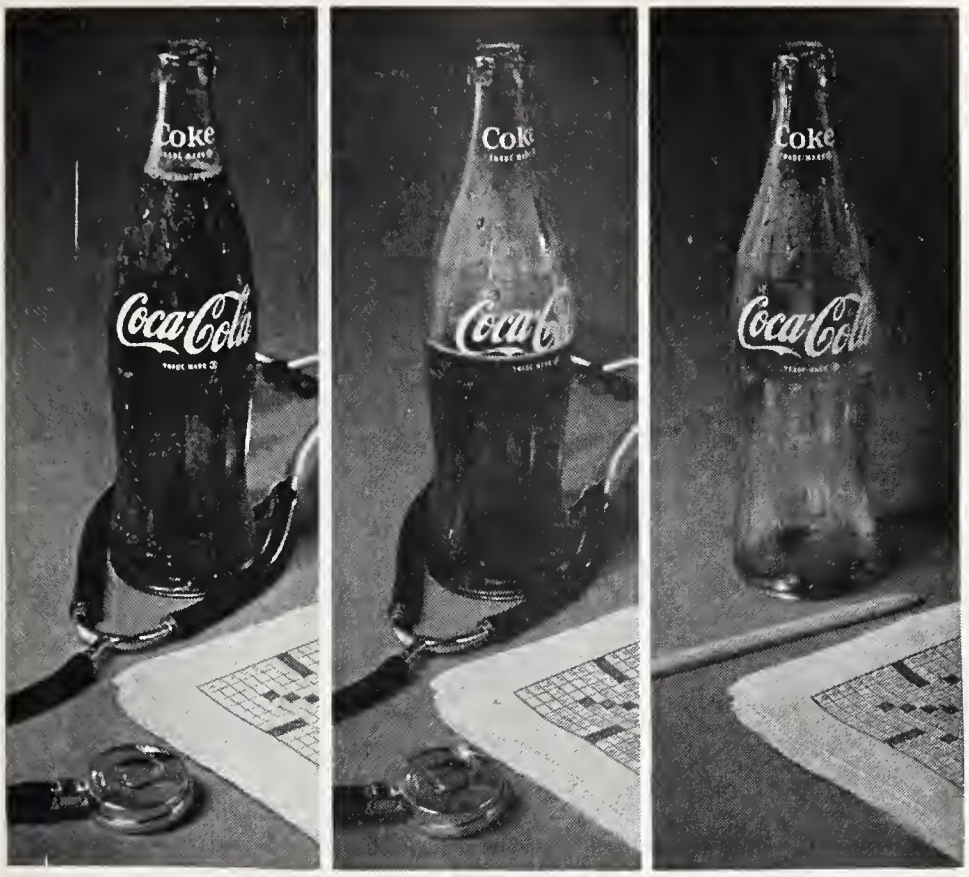
The editorial on contemporary leprosy* by Lt. Col. Robert H. Moser, El Paso, was quite timely, but I feel that he left the impression that leprosy is a rare disease except in some patients from Mexico and other foreign countries. It has been estimated by one of the dermatologists in San Antonio that there are perhaps 500 cases of leprosy in that city.

In my practice, 10 or 11 years ago, a native-born Anglo civil service employee who had never been out of the country for more than several days had active leprosy. He had been examined by a number of physicians, and also at two teaching institutions in the East, yet his condition remained undiagnosed for approximately two years because no one thought of leprosy as occurring in a native Texan who had never spent any time out of the country.

This illustrates the truth of the adage, "In medicine, if you don't think of the diagnosis you cannot make it."

—DAN A. RUSSELL, JR., M.D. San Antonio.

*"An Essay on Contemporary Leprosy," June, 1966 issue.



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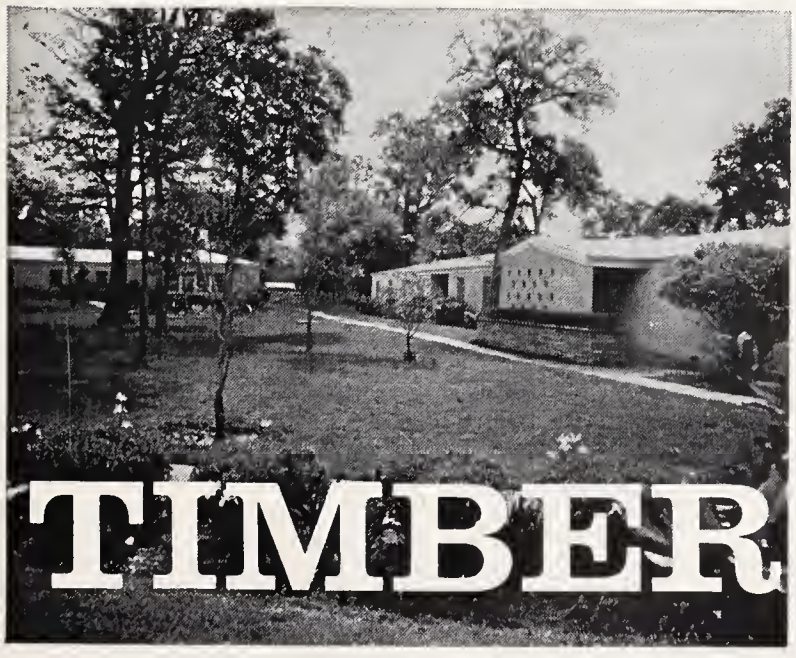

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JULY MEETINGS ADVANCE CURRENT MEDICAL PROGRAMS

For Texas physicians, the weekend of July 30-31 was indeed a busy and productive one.

The special committee to study the implementation of Title XIX devoted preliminary consideration to drafting guiding principles which it is hoped will be incorporated in Texas legislation next year. For purposes of benefits under Title XIX, the medically indigent patient was defined, the principles of reasonable and customary fees, the patient's right to choose his own physician and the physician's right to select the most beneficial therapy were endorsed. It was agreed to recommend that the program begin with the five basic benefits stipulated in Public Law 89-97 and expand as the need becomes apparent.

The TMA Committee on Liaison with the Texas Hospital Association had fruitful discussions on Medicare, nurse recruiting, hospital employees' salaries, and the hospital aspect of physicians' direct billing. Mutual respect and cooperation in all areas was evident.

The Committee on Liaison with Blue Shield, with the help of officers and representatives of the Texas Radiological Association and Blue Cross-Blue Shield officials, spent some six hours studying in depth the history, philosophy, and problems that "hospital-based" specialists have had in separating their professional fees from hospital charges. It is evident that insurance carriers will incur additional costs and that present contracts must be amended to include separate billing.

The Texas Medical Association is backing radiologists and pathologists in their endeavors for separate and direct billing and agrees that insurance carriers do not have the right to contract for the services of radiologists and pathologists under basic hospital services. Physicians believe that hospitals should charge for the technical services which they render and that radiologists and pathologists should bill patients separately for their professional services.

Sincerely,

James D. Murphy M.D.

James D. Murphy, President,
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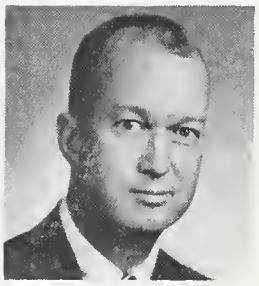
meet the authors

D. FRANK KALTREIDER is chief gynecologist and obstetrician at Baltimore City Hospitals, Baltimore. He reviews **Changing Attitudes Toward Abortion, Sterilization, and Contraception, p. 40.**

ALAN L. KAPLAN is assistant professor of obstetrics and gynecology, Baylor University College of Medicine; his coauthor, **JOHN A. WALL** practices gynecology in Houston. **Pelvic Exenteration: Appraisal, p. 46.**

E. B. MENDEL, Dallas, is an obstetrician and gynecologist. **Rupture of the Gravid Uterus; Analysis of 56 Cases in 15 Years, p. 49.**



WILLIAM KLINGENSMITH, JR., and **PATRICK OLES** practice surgery in Amarillo. Their evaluation of 150 patients with duodenal ulcer is reported in **Vagotomy and Pyloroplasty in 150 Duodenal Ulcer Patients, p. 64.**

J. ALAN ALEXANDER and **ROBERT R. FRANKLIN,** are with the Department of Obstetrics and Gynecology, Baylor University College of Medicine, Houston. **Effects of Sparteine Sulfate on Uterine Activity, p. 54.**

ED WINGA, second year resident in internal medicine, Southwestern Medical School, is a graduate of State University of Iowa College of Medicine; his internship was at the University of Utah College of Medicine. **Clinicopathologic Conference, p. 70.**

ROBERT E. HARRIS and **CHARLES E. GIBBS** report cases from Wilford Hall USAF Hospital, Lackland Air Force Base. **Pregnancy and Systemic Lupus Erythematosus, p. 57.**

A. B. GOLDSTON, Amarillo surgeon, reviews **Surgical Management of Primary Breast Cancer, p. 60.**

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Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

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Changing Attitudes Toward Abortion, Sterilization, and Contraception

REVIEW ARTICLES

THE PRESSURES confronting the political organizations of this planet—wide variations in standards of living, lack of industrial abilities to compete with wealthy countries for luxuries or necessities, lack of adequate living space and expanding population, insecurity in this age of the infinitesimal atom—are changing our political, social, and moral behavior. Our attitudes toward abortion, sterilization, and contraception are likewise changing rapidly, but there is urgent need for more widespread discussion and reevaluation, not only by the medical profession, but by social, governmental, and religious groups as well.

Abortion

Of the three procedures included in this discussion the one that has the narrowest application, in the sense of numbers involved, but which may have the gravest medical and emotional impact is therapeutic abortion. In the United States, we are fortunate that we do not need this method to control population growth. It apparently has been necessary in Scandinavian areas, Japan, Czechoslovakia, and other places. There can be no doubt that abortion is an adequate means for population control. In this country we are more concerned with the restrictions that are imposed on medical personnel, and the use of abortion as a medical therapeutic procedure rather than as a contraceptive one. Its use is forbidden in Louisiana. All other states have a variety of laws. Maryland has a fortunate law which states, "in the production of abortion a regular practitioner of medicine shall be satisfied that the fetus is dead, or that no other method will secure the safety of the mother." This law has not been tested legally, but it seems to me that "to secure the safety of the mother" would allow wide application of its use. I do not think that this law would forbid therapeutic abortion for German measles, for psychiatric reasons, for rape, or for any other reason involving the safety of the mother physically, mentally, or emotionally. The AMA has shown interest in those states which may have inadequate laws. A preliminary report from an AMA committee suggested that the AMA should go on record favoring enactment of legislation regarding abortion which provides that:

"1. A licensed physician can terminate a pregnancy if he can reasonably establish that:

a. There is substantial risk that continuance of the pregnancy would gravely impair the physical or mental health of the mother, or

b. There is substantial risk that the child will be born with grave physical or mental defects, or

c. The pregnancy resulted from legally established statutory or forcible rape or incest.

2. Abortions shall be performed only in licensed hospitals.

3. An abortion shall not be performed unless two licensed physicians, neither of whom may be performing the abortion, shall have certified in writing the circumstances which justify the abortion."

Presented to the TMA Section on Obstetrics and Gynecology, April 16, 1966.

Economic security, mortality, mother and child welfare, and the population explosion present physicians with difficult decisions on birth control. The author provokes thought on a wide range of urgent problems.

D. FRANK KALTREIDER, M.D.

What would the impact of such changes be on the medical profession? For one thing, they would legalize what many physicians are now doing. They would certainly not reduce the birth rate or control the population. In fact, the use of therapeutic abortion as a method of population control seems unrealistic when contraceptive or preventive measures are now so useful. Is it desirable to use therapeutic abortion as the starting point to decrease the number of criminal abortions in our society? Kummer of University of California at Los Angeles and Leavy[2] of the Los Angeles County District Attorney's office believe that liberalization of the therapeutic abortion law would be desirable. They recognize that sex education and an ideal contraceptive would be most effective. For therapeutic abortion to be effective in the reduction of criminal abortion, indications must be liberalized to include "the tired mother syndrome"[2] and social and economic indications. Liberalization for the health of the mother would not be sufficient. If rape, incest, and suspected defective fetuses were included among indications, the number of criminal abortions would be only slightly reduced. Contraceptive methods can effectively reduce the number of criminal abortions. At Baltimore City Hospitals the number of patients with obvious criminal abortions was drastically reduced when contraceptive methods became available to everyone. I do not believe that we should rely on broadened indications for therapeutic abortion to reduce the number of criminal abortions. Wider basis could lower the cost of abortions but I fear they would not materially reduce abortion deaths. Contraception should be a more effective method of reducing deaths. In Baltimore this has been proved.

The efforts by Kummer and Leavy[2] are, however, worthwhile because they are directed toward achieving a more realistic legal method for dealing with the problems of medical abortion—the physical and mental health of the mother, probable abnormalities of the infant, as from German measles and thalidomide, and in cases of rape, incest, and other criminal acts.

With sterilization there are more varied problems—indications, methods, and legality. We have abused the standard indication, "multiparity." When Eastman[3] showed the dangers of high multiparity—eight or more—these dangers were real. Justification for multiparity at this level today is medically unsound. All of us can be justly proud of the safety of pregnancy, particularly when criminal abortion deaths are removed from statistics. Danger of multiparity does justify us to proceed with sterilization. The danger is not justified for patients having their fourth and fifth children. On the other hand, other laudable indications for abortion are socioeconomic handicaps, the prevention of the unwanted child, and the desire not to bring one or more mouth into an impoverished environment. Socioeconomic reasons have been acceptable and used for a long time; we have merely forgotten to label them as such.

Are there any strictly medical indications, where in all honesty we can say that a patient must not have any more children, and that if she does she will die? I am really hard pressed to think of one. Where health and

welfare are involved there are many indications—hypertension, Rh problem, heart disease, and psychiatric problems. But in number these are insignificant when compared with the socioeconomic reasons.

Sterilization

For methods of sterilization, attitudes are changing. Male sterilization has become more acceptable in the middle class population. Perhaps masculinity has been proved in this group of men so that they do not feel that manhood is lost after vasoligation. Of more interest, however, is the removal of the uterus, a most effective method of preventing pregnancy. How widely accepted this method is I really do not know, but it is becoming well known among possible candidates. Occasionally I have been requested, at one fell swoop, to remove all possibilities of tubal ligation failure ("my friend got pregnant after her tubes were tied"). My own attitude is not fully crystallized. There is not sufficient data to indicate how much psychologic trauma has been committed when the patient knows she has absolutely no chance of ever becoming pregnant again, even though she may consciously be sure that this is her desire. How many women request and accept tubal ligation because they unconsciously know reversal is easy—easy for them; they do not know that it is not easy for the physician.

That psychiatric problems follow sterilization procedures is not open to question. Psychiatric problems follow almost any act or even non-act. It is difficult to ascertain whether or not there is an increase or decrease of psychiatric problems following sterilization. I have not found any acceptable data that have allowed definite conclusions. Ellison[4] reviewed 25 psychiatric patients in whom he felt that sterilization was the cause of the psychiatric problem. Most of the women who had had hysterectomies had depressions. After tubal ligation, a paranoid-depressive state was common. This is an unfortunate study because the material was gathered from the psychiatric institution and was not a prospective study beginning with the operative procedure. This does not, however, negate its value; even though the conscious desire for the patient may be to have no more children, the method whereby this is accomplished may have serious and varied effects. Barnes and Zuspan[5] reported that when patients initiated the events leading to tubal ligation, 90 percent were happy or at least did not express any regret, whereas only 70 percent persuaded by the physician showed no regret. Curiously enough where there was a bona fide indication for sterilization, such as a repeated cesarean section, there was more subsequent regret than when the reasons were socioeconomic or high parity. Some method of psychologic testing must be developed that can satisfactorily predict which patient will or will not have psychologic problems following the operative procedure. This will be more important as sterilization by hysterectomy becomes more popular. We must know how psychological problems following hysterectomy differ from those after tubal ligation. Are we causing serious marital sexual problems that are leading to divorce? There can be no doubt that hysterectomy, whether performed for medical reasons or for sterilization, initiates psychologic, sexual, psychiatric, and psychosomatic problems. After sitting with a review committee for hospital utilization, I have been impressed with the number of women admitted to hospitals for psychosomatic symptoms after hysterectomy. These questions need answering. As an individual it seems reasonable from a preventive viewpoint for a doctor to prefer hysterectomy; it is never again necessary to deliver the patient's child. It can be embarrassing when one has to deliver a baby after another contraceptive procedure fails.

Do we have a legal problem? Laws do not always protect. Virginia requires a 30-day "thinking over" period for voluntary sterilization. What can happen to the physician who sterilizes on an emergency basis is not clear to me. I feel safer under no law in these circumstances.

At the AMA meeting in November, 1965, the House of Delegates[1] returned a preliminary proposal on sterilization to the committee for study in depth. The report recommended that:

"1. An adult requesting the procedure must request it within a reasonable time prior to the time of the performance of the procedure;

2. If married, his or her spouse must join in cosigning the written request;

3. Prior to or at the time the written request is made, the physician must make a full and reasonable medical explanation to

the patient and the spouse of the meaning and consequences of the procedure."

The first recommendation concerning time is reasonable under most circumstances; in others, rather unreasonable, possibly making two operative procedures necessary. There may not be a reasonable time to discuss the problem with the patient, or the husband may be unobtainable. This requirement can cause problems.

The request that the husband is required for cosigning may be an invasion of human rights under the Constitution of the United States. If there is an urgent need for sterilization with an excellent medical reason, and the patient, having been made aware of the consequences of another pregnancy, agrees that sterilization should be done, can a husband legally refuse the sterilization procedure regardless of his reason? It seems unreasonable that an adult, being faced with a medical decision that requires consent by that adult, can be restrained by another adult. Why cannot the wife make this decision alone? Otherwise, could this not be an effective method of committing uxorcide legally? Even if the decision for sterilization is for socioeconomic reasons alone, why should one adult have the right over another (even though married) to determine the extent of her childbearing? It seems to me that if there is any relative right or wrong involved, the one who must endure the pregnancy and labor should have the primary right. This is a two-way street. If the husband desires vasoligation why should a wife deprive him of this decision? Further, about 30 percent of my clientele requesting sterilization are either not married, or are divorced or separated, or have no idea where their husbands are. Should I refuse sterilization on socioeconomic grounds because the husband cannot be found? It seems unrealistic to me. The report returned to the AMA committee called upon the House of Delegates to endorse these principles as ethical and to endorse enactment of legislation which would make it "lawful for a physician, acting in collaboration or consultation with at least one or more other physicians, to perform a sterilizing procedure upon either sex for therapeutic or non-therapeutic reasons." This sort of recommendation merely asks for trouble. If a state does not have a law nor a court decision, its people are fortunate. This decision can be made privately and is being made privately continually. If it becomes public—as with a law—the family, the wife, the husband, the deprived children, and the physician will have a chance of being handicapped. Hopefully when the committee reports again, this still further invasion of one's freedom will be put aside.

Contraception

It seems to me that the drive toward preventing a population explosion has been more explosive than the explosion itself. The Gynecology-Obstetrics Guide abstracted 175 articles on contraception during 1963 and 1964, whereas in 1965 there were 180 in one year—twice as many. This points to the interest being generated about this challenging problem.

The pills have become so well entrenched in the habit of millions of women in such an amazingly short time that one wonders why physicians have not been aware earlier of this deep feeling for family limitation. Obstetrician-gynecologists are overwhelmed with the office practice of contraception, and health department clinics are dispensing progestins and inserting contraceptive rings by the thousands. The request for information became so frequent in my own postpartum wards that it seemed more efficient to routinely interview each patient and to aid her according to her desires. This is being done by women volunteers.

I am concerned with some of the problems either actually or allegedly associated with the pill, such as thrombophlebitis, minimal to maximal strokes with all types of neurologic sequelae, headaches, personality changes, weight gain, and many other minor but annoying problems. It is unfortunate that the nation's press dramatizes these serious but infrequent complications. The serious complications are most often without prodromal symptoms or signs, while the minor ones can be eliminated almost immediately by the cessation of the progestin. Unreliable evidence suggests that the incidence of thrombophlebitis is 1 to 5 per million. Let us assume that all persons taking the pill die. If these million women were not using any type of contraception, 840,000 would become pregnant in one year. Of these 840,000 women, some 300 would die during pregnancy from obstetrical causes alone. Some 200 more would die from related causes, infections, heart disease, or other causes. If, of the one million women, all were using a diaphragm or their partners a condom, some 140,000 women would become pregnant in

one year and there would be 50 potential maternal deaths. If an intrauterine device were used in these one million women, some 30,000 would become pregnant with some 11 potential maternal deaths. It is not too difficult to draw conclusions as to which is the more desirable.

What about the sublethal complications of stroke? No definitive data are available on its incidence. With present, probably inaccurate, knowledge, the rate of minor to major strokes is no greater than the rate for occurrence of thrombophlebitis. Pregnancy increases risks of hypertension, diabetes, various thyroid diseases, as well as other illnesses. One must be realistic in prescribing the pill when patients desire it for contraception. It seems realistic that when a patient has symptoms of some moment, such as personality change and headache, the drug must be stopped immediately and a substitute method used. On the other hand this nightmare of thrombophlebitis and stroke is not as serious as the risk of pregnancy itself, particularly if the patient does not desire to become pregnant.

The complications with intrauterine contraceptive devices are probably just as momentous as those with the pill. Although I know of no deaths attributed to their use, at least three women have had perforation of the uterus with gangrene of a loop of bowel and required laparotomy with resection. Ectopic pregnancy still occurs. Patients have excessive bleeding, as well as intermenstrual spotting. I'm not making a plea for either contraceptive method. Problems occur with all types of contraceptives, but so do they with aspirin. I do not believe contraceptives should be halted.

Family Insecurity

Let us take a look at another aspect of contraception. Do we as physicians have any moral, ethical, or social obligation to society, to the ever increasing number of unwanted children resulting from illegitimate pregnancies? The deterioration of family life is increasing, and with it a large number of children are being born illegitimately. Parental security, training, and development of sexual responsibility are decreasing in our society. The demand of a high standard of living is in many ways responsible—the need for both parents to work in order to keep up with the Joneses. This increasing lack of responsibility is a major concern; vacuums left by the family have a habit of being filled by the state. As long as there is a good and decent result without jeopardy of the individual's freedom, I am delighted when the state does take over and does a good job. On the other hand, the reasonable way to fill this vacuum is reeducation of future parents and this must be done in the home and the schools. I am a firm believer in the thesis that the philosophy of family life first promulgated by the Hebrew nation some 4,000 years ago is a firm preventer of social disorders. I see no solution to our increase in abnormal social behavior until the generation after next, and it is necessary to regress to the Judaic family principle now, as well as to make some attempt now to reduce illegitimacy. Patterns established early in life are difficult to change. For this reason two generations does not seem to be too long a time to make any change in patterns of behavior. In order to reverse these undesirable trends in our culture, a reversion to family life must begin now, and this requires an immediate reduction of illegitimacy in order for family life to be available to newborns now being delivered.

If a young woman has established a sexual pattern that results in pregnancy and if at interview following pregnancy she intimates that this pattern will continue, is the withholding of contraceptive advice moral? If moral beliefs of the physician result in unwanted pregnancies, increased adoption loads, children raised without a father, adults who have not experienced the advantage of family life and who become a major social problem, can they still be considered moral? It is my opinion that they would be immoral. I am not condoning the lack of responsibility of the unmarried boy or girl who insists on sexual intercourse; I am merely being practical. I would like to prevent the unwanted child who has no emotional support and security from becoming the juvenile delinquent who lacks social responsibility. It is certainly more desirable to develop responsibility.

Sex in College Students

An increase in sexual freedom is also apparent among college students. The press reflects more often than not the attitude and opinions of the large middle-class population, from which most college students come. Let us quote headlines cited in an article in

the Nov. 21, 1965, issue of the *New York Times* entitled, "The Pill and Morality,"[6] the article recalls, in 1926, "College Looseness Blamed on Co-eds"; in 1930, "Seniors Held Unholy for Attacking the Moral Code"; in 1945, "Peale Deplores Immorality"; in 1950, "Moral Lapse Laid to Kinsey Report"; in 1960, "Survey Deplores Decay of Values"; in 1965, "Girls Entering College Have Asked About Birth Control." All young generations are sure that they are different and all older generations agree that all younger generations are different; the former think they are better; the latter think they are worse. I suspect that all young generations fit the aphorism—the more Paris changes, the more it remains the same. The headlines seem to indicate that the present younger generation is following the pattern of the one preceding it in many respects. Today the attitudes toward sex are open and blatant, rather than Victorian and closed, but there really is little basic difference.

A fine review of the college problem, published in late 1965 by the Group for the Advancement of Psychiatry, presents thoughtful analysis of the development of sexuality, campus sex issues, campus policies and guidelines on sexuality. The guidelines suggested for colleges and their relationships to contraception seem to be particularly well thought out:

> "Most campus health services make available to the student, information and medication for various physical conditions. The provision for contraceptive information or materials, however, is not usually considered within the scope of the University Health Service.
>
> The broad, over-the-counter availability of contraceptive pills, diaphragms, condoms, and foams indicates the need for reexamination of this position and suggests possible modification to the extent that information be made available through the health service. To those who might advocate dispensing contraceptive materials we would say that this cannot be done routinely or casually. Prescription of contraceptive devices requires as much judgment as does another medical decision. It should again be noted that many students will interpret the dispensation of contraceptive devices as sanction for their use.
>
> We believe, however, that silence is not the only alternate to dispensing diaphragms and pills. Providing contraceptive information in the college setting seems to us tenable and appropriate, either on an individual basis in response to request or in the context of Sex Education. It is also proper that such resources provide information about the public health aspects of venereal disease. A decision to dispense contraceptive information but not contraceptive devices would be consistent with aspect for the autonomy of the student and would place the responsibility for the use of the information in his own hands."

The development of responsibility is a most important recommendation. Responsibility must be developed. It is not an innate characteristic of the human. Sex education should be given to the college student as a continuation of sex education that the adolescent or child has received in the home, the junior and senior high schools, and the grade schools. I suspect that the child in kindergarten is not too young to learn about the sex life of the tomato or the rose.

The woman who becomes illegitimately pregnant because of lack of responsibility is too often inadequately advised and supervised. Gynecologist-obstetricians are not advising or supervising well. We have done a great deal toward changing the pregnant woman's attitude on abortion, persuading her to allow the pregnancy to continue, but we have neglected the social and learning phases which should be involved. There are inadequate social environments where an atmosphere of acceptance by the community is the norm, and where a framework of usefulness can be developed while awaiting the long time required for pregnancy to come to term. We must become active in this area. We must develop environments where the illegitimately pregnant woman can live a normal life during pregnancy, where she may be made to feel useful to society by creating work activities. Most important are learning sessions with a physician or a psychologist which lead to appropriate sexual attitudes compatible with sexual responsibility, not only to herself but to her offspring.

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Pelvic Exenteration: Appraisal

REVIEW ARTICLES

IN 1947, Alexander Brunschwig, New York, began a series of ultraradical operations for the removal from the pelvis of advanced carcinoma.[1] He selected patients who had local pelvic disease which had failed to respond to conventional treatment by irradiation or surgery and which, although advanced, was still technically removable. The operation was based on the observation that in many women who died from carcinoma of the cervix, the growth had not spread beyond the pelvis.[2]

Brunschwig originally undertook the removal of these large masses of tumor with the affected viscera as palliation, and the early attempts successfully demonstrated the feasibility of the operation and its palliative possibilities. Later the possibility of five-year survival was realized, so that today exenteration is advised only when there is a reasonable chance for cure.

By the end of 1958, Brunschwig and his team had completed 592 exenteration operations with an operative mortality (deaths within 30 days) of 17 percent and a five-year survival rate from 388 cases of 17 percent.[2] Brunschwig's experience showed that all the pelvic viscera could be safely removed, and with the exception of the external iliac artery, all the pelvic blood vessels could be tied on one or both sides without the risk of gangrene of the lower limb. It showed also that it was not necessary to cover the pelvic floor with peritoneum, although these large raw areas certainly increased the incidence of postoperative obstruction of the small intestine.

This type of surgery is highly specialized and carries in its wake complications which need expert care. The operation preferably should be performed in centers where a team of workers experienced in dealing with advanced cancer is available.

Definition and Indications

The term "pelvic exenteration" denotes total removal of the upper genitalia, contiguous fatty tissue and lymph nodes in the pelvis, plus the bladder (anterior exenteration) or the rectum (posterior exenteration) or both (total exenteration). Usually, exenteration operations are accomplished via an abdominal approach alone. The tissues are mobilized from above down as far as the levator ani muscles and then the urethra, vagina, and rectum are transected at this level. However, when cancer extends low in any of these structures or is primary in the urethra, vulva, vagina, rectum, or anus, it is advisable to do a perineal excision in continuity as well.

The accepted indications for pelvic exenteration fall into three categories: (1) lesions that persist or recur following primary treatment by radiation; (2) advanced primary lesions in which the disease has progressed beyond the scope of less radical measures; (3) lesions such as sarcoma botryoides or advanced vulva carcinoma in which irradiation is known to be ineffective.

Although the term "persistence" is generally used synonymously with the term "recurrence," we believe that persistence is the more accurate term. Persistent disease is

When pelvic cancer is still technically removable but is unresponsive to, or beyond the scope of, conventional therapy, pelvic exenteration may be considered. The patient must be physically and psychologically able to withstand formidable surgery; mortality is high, and major complications may ensue. However, exenteration can be successful when done by an experienced team.

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defined as carcinoma that continues in spite of therapy. The recurrent case is defined as a tumor that apparently heals under therapy and then returns after a specific period of time. At our institution this time interval is five years.

Selection of Patients

In selecting patients for the operation, one must consider both the lesion itself and the patient as a whole, with special reference to her mental attitudes toward the problem and her determination to overcome grave disabilities. This means that she must appreciate the nature of her disease, the prognosis if she does not have the operation, and the precise significance of what the operation is proposed to do. Without such information she cannot be in a position to decide whether or not she wants to go through with the surgical procedure.

Once the decision to proceed is made, the patient is admitted to the hospital for complete medical evaluation. Our only contraindications to exploratory surgery are distant metastases and medical fragility. Surgical exploration is an acceptable diagnostic procedure and our philosophy is to be extremely liberal in its use, but extremely critical and conservative in proceeding with resection. We do not recognize any absolute signs or symptoms such as leg edema, back pain, or ureteral obstruction as indications of nonresectability. We have found fibrosis to be the offender in many of these cases.[4] A lesion cannot be deemed inoperable unless operation is done.

Proceeding with resection is a different matter, however. The goal of this operation is cure, not palliation, and it should be done only after extensive evaluation in the operating room. Contraindications to be noted during exploratory surgery are intraperitoneal disease, involvement of para-aortic lymph nodes, and local extension from the parametrial tissues into the fascia overlying the obturator internus muscle or sciatic nerve. If the lesion is resectable, our urinary diversion of choice is the ileal or sigmoid conduit.

Case Reports

Case 1.—A 37-year-old Negro, gravida 7, para 7, was seen initially in February, 1964, with a stage 2a squamous cell carcinoma of the cervix. The first radium application was loaded with 20 mg. in tandem and 20 mg. in each of two vaginal ovoids. This remained in place for 72 hours. A second identical application was given 17 days later. External irradiation was then given to the parametria and pelvic nodes with a 2 m.e.v. Van de Graaff machine and totaled 3,500 r. tumor dose in three weeks. A 4 cm. central lead shield was used.

Following completion of therapy the patient did well until September, 1964, when a mass was noted in the pelvis. Intravenous pyelograms showed no excretion of dye on the right side and minimal dilatation on the left. Biopsy of the cervix and upper vagina failed to reveal carcinoma. At exploratory laparotomy, extensive carcinoma involving the right cardinal and uterosacral ligaments was found. A total pelvic exenteration with sigmoid conduit and sigmoid colostomy was done. The pathologic examination showed good surgical margins. The patient's postoperative course was normal and at present she is doing well.

Case 2.—A 41-year-old Negro, gravida 0, was seen initially in December, 1964, with an advanced

squamous cell carcinoma of the vulva. On Jan. 25, 1965, a total pelvic exenteration with perineal phase and vulvectomy and bilateral inguinal and iliac lymphadenectomies were performed. A sigmoid conduit and sigmoid colostomy were made. Three days postoperatively the patient had a split-thickness skin graft to the lower abdomen and upper perineum, and seven days later a second split-thickness skin graft was placed over the area of the left thigh. The postoperative course was normal otherwise. The pathologic examination showed good surgical margins; however, the node of Cloquet and lower external iliac nodes on the left side contained squamous cell carcinoma. Because of this, a 3,000 r. tumor dose to the pelvis was given with a 2 m.e.v. Van de Graaff machine. Subsequently the patient has done well and shows no evidence of persistent carcinoma.

Complications

This is an ultraradical surgical procedure and major postoperative complications are to be expected. The incidence of immediate major complications in most larger series is approximately 35-40 percent.[5,7] About 10-15 percent of the patients will require reoperation for complications occurring in the early postoperative period. The major problems are intestinal obstruction, difficulties with the urinary diversion, urinary or intestinal perineal fistulae, hemorrhage, fluid and electrolyte imbalance and wound infection.

Once the immediate postoperative period is over, one must still anticipate late problems. Approximately 30 percent of these patients will require surgery for later complications[5,7] which are related mainly to the urinary diversion, the external stomata, and the large pelvic defect which predisposes to fistulae and intestinal obstruction.

Results

The overall results with this procedure show a five-year survival rate of 25-30 percent. The immediate operative mortality rate is 15-20 percent and the incidence of late deaths somewhere between 50 and 60 percent. Over 85 percent of these late deaths are due to persistent disease and occur within the first two years following operation.[5,7] With rigid criteria for selection, operative experience, and refined preoperative and postoperative care, we should see an increase in the five-year survival rate with a decrease in the immediate operative mortality. This has been the trend since the procedure was first done in 1947. Brunschwig's operative mortality during the first several years was in excess of 30 percent. Bricker reported 13 percent* in 1956,[5] Silva 15 percent† in 1959,[7] and Rutledge 17 percent† in 1965.[8]

Summary

It seems clear that there is a place, even though a limited one, for exenteration in the treatment of pelvic malignant disease. Proper selection of cases on both physical and psychological grounds is vital for success. The operation is reserved for those in whom primary treatment has failed or for those whose lesion is too far advanced for treatment in a more routine fashion.

The philosophy in our clinic is one of liberalism concerning exploratory surgery but extreme conservatism regarding resection. This is a formidable procedure and carries with it a high mortality and major complications rate. The operation should be performed only in special centers where a team of workers skilled in dealing with advanced cancer is available.

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*Deaths occurring before hospital discharge.
†Deaths occurring within 30 days.

Rupture of the Gravid Uterus

Analysis of 56 Cases in 15 Years

E. B. Mendel, M.D., and R. D. Eward, M.D.

Prior uterine surgery, is the most common predisposing factor in rupture. Recently there has been an increase in cases associated with oxytocic drugs, and a decrease in cases resulting from forceps and breech extractions. Repeat cesarean sections at 38 weeks, and early recognition and treatment prevents high mortality.

REVIEW ARTICLES

"RUPTURE OF the uterus" denotes perforation from any cause after fetal viability has been established.[1] This obstetrical accident has a reported incidence from 1 in 137 to 1 in 4,460 deliveries.[2-4]

Some authors have stated that the incidence of rupture of the gravid uterus is rising because oxytocics are frequently used for stimulation and induction of labor.[5] An analysis of the problem as seen at Baylor University Medical Center during the past 15 years is presented.

From Jan. 1, 1950, through Dec. 31, 1965, there were 103,996 deliveries on the obstetrical service of the hospital, with 56 ruptures of the uterus. This incidence of 1:1,857 falls well within the reported range of numerous other studies (Table 1). The total incidence is slightly higher than the usually accepted average of 1:2,000 deliveries.[7] There is debate as to whether or not the incidence has increased in the past ten years.[5,6,8] As seen in Table 1 some of the most recent reports vary between 1:1,034 and 1:2,886. The variation is so great that incidence is difficult to analyze.

However, if one could classify the number of ruptured uteri in which specific factors such as oxytocics, version with extraction, or difficult midforceps rotation were utilized, one could more correctly determine whether or not an increase in incidence is present.

TABLE 1.—*Reported Ratios of Uterine Ruptures to Total Deliveries.*

Author	Year	Ratio
Brierton[12]	1950	1:1,961
Jacobs[8]	1962	1:2,402
Frudenfeld[15]	1962	1:1,500
Waters[13]	1962	1:2,086
O'Rourke[9]	1963	1:2,886
Keifer[5]	1964	1:2,665
Donnelly[16]	1964	1:2,679
Garnet[11]	1964	1:1,034
Cavanagh[10]	1965	1:1,216
Present Study	1966	1:1,857

Presented before the TMA Section on Obstetrics and Gynecology April 15, 1966, in Austin.

Classification

The classification of rupture of the uterus has been relatively standardized. Utilizing this general classification, the distribution of cases in this study is outlined in Table 2.

Of the 56 cases, 34 were rupture of previous cesarean section scars, an incidence of 61

TABLE 2.—*Classification and Distribution.*

I. Spontaneous Rupture of Undamaged Uterus:			
A. Traumatic		12	
B. Spontaneous		9	
Total			21
II. Spontaneous Rupture of Damaged Uterus:			
A. Post-cesarean		34	
1. Spontaneous	(16)		
2. Traumatic	(0)		
3. Silent	(18)		
B. Other Prior Insults		1	
1. Cornual resection	(1)		
Total			35

percent. In 18 of those cases, a silent rupture in the old scar was found; 15 were acute and symptomatic. One patient had a rupture of the right cornu of the uterus at 37 weeks' gestation, but it was unrelated to a previous transverse cesarean scar.

Eight cases of ruptured uteri without any known insult to the uterus and no known induction or stimulation of labor are worth mentioning. Two of these cases were found after forceps failed. In five cases no apparent reason was found other than "pushing" pressure during the second stage of labor in multiparous patients, or external abdominal pressure at the time of delivery.

Case 1.—In a 19-year-old patient, gravida 1, para 0, leiomyoma of the uterus was detected by examination at 32 weeks' gestation. At 40 weeks' gestation she was admitted in spontaneous labor and was delivered uneventfully. A normal postpartum course followed. Six weeks post partum she complained of cramping and vaginal bleeding; a dilatation and curettage was performed and a leiomyoma was delivered vaginally. At that time a tear in the left lateral side of the uterus was noted and repaired at laparotomy. The tear did not have any relationship to the leiomyoma implantation site on the right side.

Case 2.—A 39-year-old white woman, gravida 4, para 0-1-4-0, had a previous resection of the right cornu. At 20 weeks' gestation she had a Shirodkar procedure with a normal course until 28 weeks' gestation, at which time pain in the left lower quadrant and shock developed. Exploratory surgery revealed rupture of the left cornu through a placenta accreta site.

Incidence

The incidence of ruptured uteri due to oxytocic induction has been determined in some reports. Jacobs and associates reported 6 ruptures in 41 cases in which Pitocin was utilized.[8] O'Rourke and Nichols,[9] reported that 4 of 19 ruptures were due to Pitocin induction, and Cavanagh and others[10] reported 6 of 41 cases as resulting from oxytocics. In Garnet's analysis[11] of 133 patients in a combined survey in the Northeastern portion of the United States, there were 30 cases of ruptured uteri in which oxytocics were a contributing factor. In our study there were 12 such cases out of 56. As seen in Figs. 1 and 2, there is an increase not only in the number of cases in which rupture occurred, but also in the number of cases in which oxytocics were implicated.

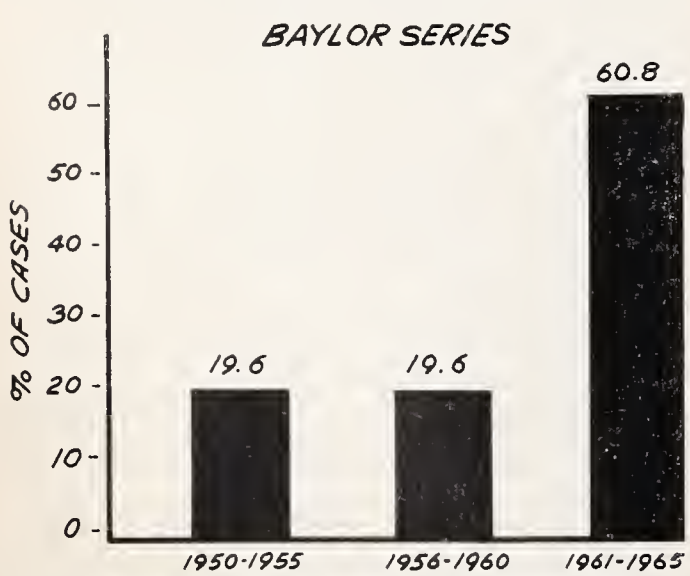

Fig. 1. Distribution of 56 cases of rupture occurring in a 15-year period.

Age

One-half (27) of the patients experiencing uterine rupture were at least 30 years of age. Eight of these patients had no known previous insult to the uterus. In toto, 44.6 percent of the ruptures occurred in the 15-29 age group, 55.4 percent in the 30-50 age group (Table 3). Our findings are in agreement

TABLE 3.—*Age Incidence, Baylor Series.*

Age	No. Patients	%
15-19	2	3.6
20-29	23	41.0
30-39	27	48.2
40-50	4	7.2
Total	56	100.0

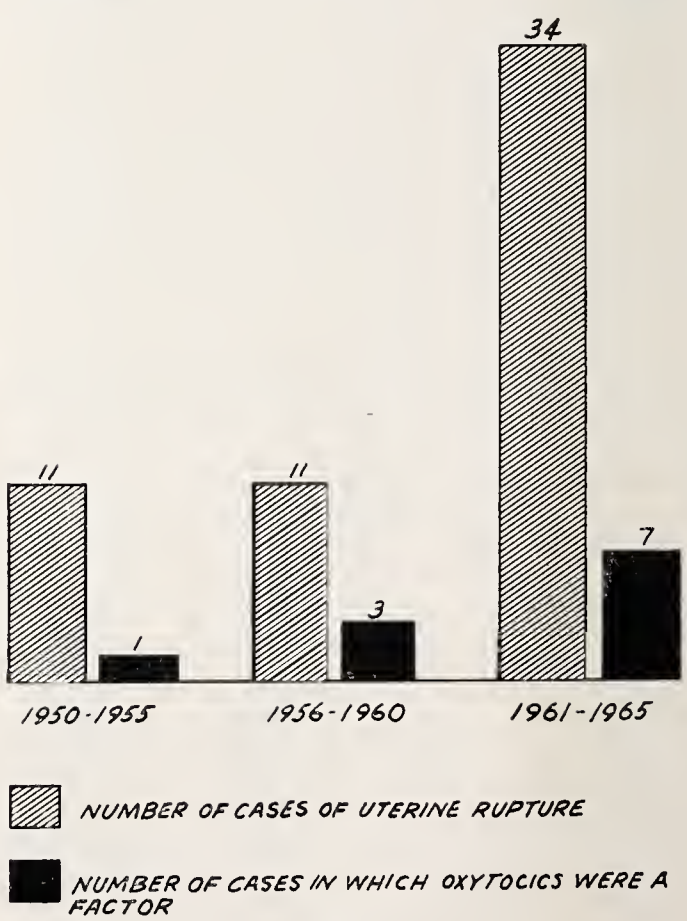

Fig. 2.

with other authors' findings in that there is minimal evidence that age is a contributing factor in rupture of the uterus.[8,11]

Parity

Increasing parity is a predisposing factor in spontaneous or traumatic rupture of the gravid uterus. Of the 56 patients in our study, 55 were multiparous and 18 had four or more pregnancies. Twelve of the 18 patients had no previous insult to the uterus, i.e. 67 percent of the patients had a noncesarean rupture. Seven of the 12 that had a noncesarean rupture had oxytocic stimulation. As has been noted, oxytocin induction or stimulation is a hazard in multiparas.

Time of Rupture

The majority of the uterine ruptures occurred at or near term. In 51 patients the gestation was 36 weeks or longer. Three ruptures occurred between 30 and 35 weeks' gestation. One, at 20 weeks, was through an old low cervical scar, and occurred during premature labor.

There were 34 cases of dehiscence, or silent rupture of an old uterine scar. Only one case presented a problem of surgical intervention prior to 35 weeks' gestation:

Case 3.—A 25-year-old patient, gravida 2, para 1-0-0-1, had a history of abdominal pain, ruptured membranes, and slight vaginal bleeding. The diagnosis was rupture of the old scar, and a uterine repair was performed along with tubal ligation.

Others have reported a peak incidence of rupture of the uterus through previous scars at one to two weeks prior to term, while in the intact uterus it was primarily at term.[8,11,12] It has been the impression that two-thirds of the ruptures of the previous uterine scar can be prevented by performing elective sections at 38-39 weeks' gestation.

Type of Rupture

It is impossible to draw any conclusion concerning the preponderance of either a low transverse or longitudinal uterine scar rupturing more frequently, due to the lack of knowledge concerning the total number of each type of uterine incision.

In the group of acute spontaneous ruptures two were cornual. The first followed a Shirodkar procedure in the accreta implantation site, and the second, for no apparent reason, involved the left cornu even though a previous low transverse scar was present.

Case 4.—Rupture of the fundus of the uterus occurred in a 33-year-old white woman, gravida 4, para 3-0-0-3. Labor was induced because of prolonged rupture of the membranes. Two rupture sites, one in the fundus, and one in the left lateral side of the uterus, were found. Treatment was subtotal hysterectomy.

Case 5.—A 41-year-old white woman, gravida 5, para 4-0-0-4, had normal labor and spontaneous delivery of a 7 lb., 2½ oz. infant following elective induction at 40 weeks' gestation. The uterus was inspected at delivery, but 36 hours post partum, tenderness developed in the right lower quadrant, and progressive shock ensued. A large, retroperitoneal hematoma of the right gutter was found, and a hysterectomy was performed. The pathologic report was rupture of an aneurysm of the right uterine artery. The uterus was intact.

Case 6.—A 37-year-old white woman, gravida 4, para 3-0-0-3, was admitted for induction of labor at 38 weeks' gestation. Her uterus ruptured around a 10 cm. myoma.

Two of the spontaneous ruptures included in this study were previously reported by Mendel and Bone as partial ruptures of the uterus found during tubal ligation. The ruptures included the serosa and part of the muscle of the uterus with an intact endometrium.[14]

Factors

The majority of uterine ruptures, whether they be traumatic or spontaneous, occur in the distended and weakened lower segment. This lower segment is either weakened and scarred from multiple pregnancies, or traumatically ruptured by numerous factors. The latter has been reported most commonly with breech extractions, midforceps delivery, and oxytocic stimulation. However, low forceps, fundal pressure, and manual dilatation of the cervix are also common causative agents in traumatic rupture. A recent article noted a decrease in ruptured uteri associated with version and extraction due to the trend toward less frequent use of this procedure.[10] Cavanagh[10] found the chief etiological factors in noncesarean ruptures to be oxytocic stimulation, breech extraction and fundal pressure, previous uterine perforation, spontaneous rupture, and internal version and breech delivery. In 1950, Brierton found that version and breech extraction or both were the most common agents in traumatic ruptures.[12] There were no reported cases of oxytocin-induced uterine ruptures. Publications since then have reported many cases attributed to the use of oxytocics.[8,9,11,13]

In one case of uterine rupture after oxytocin administration, there was also coexist-

ent failure of forceps delivery. The rupture was from the midline to the left broad ligament in the lower uterine segment, and could have been attributed to either cause. In the other cases, no other etiological agent except multiparity (9 cases) could be implicated.

Case 7.—A 35-year-old gravida 9, para 7-0-1-7, was known to have a dead fetus, and was given injections of Pitocin. The next day, she was admitted to the hospital with abdominal pain and shock. A right anterolateral tear was found.

Treatment

The majority of the defects were repaired (Table 4). Tubal ligation in association with repair was performed in multiparas, and most commonly following repeat cesarean sections. Three of the cesarean hysterectomies were performed because of severe rupture not associated with previous scarring of the uterus.

There has been a definite trend toward total abdominal hysterectomy instead of subtotal hysterectomy in the past 15 years, and also an increase in the number of cesarean hysterectomies (Table 5).

TABLE 4.—*Treatment of Ruptures Uteri, Baylor Series.*

Repair of defect	24
Repair of defect, tubal	6
Cesarean hysterectomy	9
Total hysterectomy	7
Subtotal hysterectomy	9
Subtotal hysterectomy & hypogastric ligation	1
	56

TABLE 5.—*Treatment, Baylor Series.*

	Hysterectomy		
	Subtotal	Total	Cesarean
1951-55	4	0	0
1956-60	4	2	1
1961-65	2	14	8

Of the 12 traumatic ruptures, only 2 were repaired per vagina. The severity of the other cases dictated hysterectomy. This finding is in close agreement with Garnet's findings in 1964.[11]

Fetal Mortality

Reported fetal mortality ranges from 27.8 percent to 85 percent.[9,12] The highest mortality was in Brierton's study in 1950 with traumatic rupture of the uterus.[12]

The fetal mortality in our series was approximately 16 percent (9 stillborns, 47 live births). Two of the stillbirths were at 20 and 28 weeks of gestation. In the past five years there have been two fetal deaths, one attributable to uterine rupture at 28 weeks' gestation, and one to uterine rupture secondary to Pitocin stimulation in a term pregnancy.

Maternal Mortality

There were no maternal deaths or serious postoperative complications in our series. One patient had a septic condition for three days postoperatively, but it subsided without complication.

Comment

About 32 percent of the uterine ruptures have been silent ones in previously damaged uteri. Previous scarring has emerged as the greatest single predisposing factor in uterine rupture with an incidence of 62 percent in this study. Elective repeat cesarean section at 38 weeks' gestation would prevent many of these ruptures.

If one allows a patient who has had a previous cesarean section to labor, then a close watch by the physician is mandatory. Few such patients have been allowed to progress to labor at this hospital. Those electively treated in this manner have been women who were admitted in active labor and were 5 to 10 cm. dilated, with no signs of rupture and without cephalopelvic disproportion as an indication for the previous section; and women already in active premature labor with no signs or symptoms of impending rupture. In these few cases, there have been no maternal or fetal deaths, nor ruptures of the uteri that required laparotomy following delivery.

As noted, there is an increasing trend to stimulate labor with oxytocic drugs. The actual number of inductions at Baylor University Medical Center cannot be tabulated, but lies well between 45-60 percent of all deliveries. Along with the increased use of oxytocic drugs is an increase in the percentage of ruptured uteri that can be associated with this medication. The percentage of ruptured uteri secondary to Pitocin in our study was 21.4 percent over the past 15 years. Sixty-one percent of these occurred during the past five years.

When Pitocin induction is chosen, then a close observation of labor is mandatory. The adequacy of the pelvis should be known before the stimulant is administered. Parity is

also a consideration, since a weakened or scarred lower uterine segment is more frequent in multiparous patients. Also, myomas may weaken the uterine muscular wall and create a potential site of rupture.

The absence of maternal deaths in our series is attributed to the high index of suspicion, fast surgical preparation and techniques; administration of adequate parenteral fluids and blood is also a factor. Often, repair of the rupture can be performed if the patient is desirous of more children. Otherwise a trend toward total hysterectomy as the choice of treatment is seen in this study.

Perinatal loss has decreased. In the past five years, one fetal death was attributable to utrine rupture secondary to Pitocin stimulation, and one to prematurity and uterine rupture.

Summary and Conclusions

Review of 15 years' experience at one private institution has been presented.

The greatest single factor predisposing to uterine rupture is prior surgical insult to the uterus.

An increase in ruptured uteri due to use of oxytocic drugs is found. A decrease of cases resulting from hard forceps deliveries and breech extractions has occurred because these procedures are being performed less frequently.

The speed of recognition, the use of repeat cesarean sections electively at 38 weeks' gestation, and the early treatment of uterine rupture has prevented the severe maternal and perinatal mortality previously seen.

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Speed Is Biggest Killer

Exceeding the speed limit was the driver error which caused 41.1 percent (17,100) of all deaths on the nation's streets and highways last year. More than 1.5 million people were injured as a result of excessive speeding—enough to populate Abilene, Kingsville, Austin, El Paso, Tyler, Brownsville, Amarillo, Midland, Paris, Wichita Falls, Texas City, and Waco, with about 36,000 left over for your town.

In 1964, speeding caused 35 percent of the highway fatalities; thus, in one year, the percent of deaths directly attributable to exceeding the speed limit has risen about six points.

Effects of Sparteine Sulfate On Uterine Activity

RESEARCH & INVESTIGATION

SPARTEINE SULFATE has been available since 1873, when it was first used for the treatment of cardiac irregularities. Because of its oxytocic action, it has been studied by obstetricians since 1939. Plentl and associates have had the most experience with the obstetrical use of this drug, having reported its use in nearly 2,000 patients.[1] The investigators were favorably impressed, their data having demonstrated a reduction in the length of labor with a high success rate in both the acceleration of and induction of labor. Most importantly, they thought that the drug had no demonstrable detrimental effect on either mother or baby. However, other authors have implicated this drug as a cause of uterine rupture as well as uterine tetany with fetal distress. It was our purpose to do a double-blind clinical study of the efficacy and safety of sparteine sulfate in addition to a study of its effect on uterine activity by using continuous intrauterine pressure recordings.

Materials and Methods

Identically appearing ampules of the placebo which contained 1 cc. of normal saline and the active drug which contained 1 cc. or 150 mg. of sparteine sulfate (Actospar by Sandoz Pharmaceuticals) were coded and intermixed so that the obstetricians did not know whether the solution administered intramuscularly was the placebo or the active drug. Two groups of patients were chosen at random for the clinical study. One group consisted of 120 patients whose labor was induced and the second group included 57 patients whose labor was stimulated or accelerated. In the first group, labor was induced because of premature rupture of the membranes, and the patients were not in labor. There were no elective inductions included in this group. The test solution was given intramuscularly in 1 cc. doses at hourly intervals until there was a satisfactory result—that is, sufficient enhancement of uterine activity to effect delivery—or until three doses of the solution had been given. Data sheets obtained on every patient contained cervical dilatation time curves and detailed information on past and present obstetrical and medical history, with detailed recording of events throughout labor and delivery.

Results

Results are outlined in Tables 1-7.

The one case of uterine tetany with fetal bradycardia was a 34-year-old gravida 5, para 4 patient at term who had secondary uterine inertia. She was in the acceleration group. Twelve minutes after the first dose

TABLE 1.—*Success-Failure Figures in Acceleration of Labor.*

Type of Ampule Given	No. of Successes	No. of Failures	No. of Cases
Active	21	8	29
Placebo	15	13	28
Total	36	21	57

TABLE 2.—*Success-Failure Figures in Induction of Labor.*

Type of Ampule	No. of Successes	No. of Failures	No. of Cases
Active	26	37	63
Placebo	12	45	57
Total	38	82	120

Using the double-blind study technique, the authors have researched the value of sparteine sulfate as an oxytocic. One group of patients was monitored by continuous pressure recordings, revealing a difference of results in incidence of uterine tetany.

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of the active drug, she had a contraction which lasted for three minutes, and during the last minute, the fetal heart rate slowed to 80 beats per minute. Following the end of the contraction, the fetal heart rate returned to normal and there were no further episodes of fetal distress; the patient delivered an infant with an Apgar 7 score approximately three hours and 45 minutes after one dose of the active drug. The one case of cervical laceration was in a 39-year-old gravida 5, para 4 patient who was given a series of three intramuscular injections of the active drug which resulted in effective labor that ended three and one-half hours after the last injection; an infant with an Apgar score of 10 was delivered. A 3 cm. cervical laceration noticed following delivery was easily repaired. The one case of nausea and vomiting started precipitously approximately one to two minutes after the intramuscular injection and was associated with generalized numbness and tingling but all of the symptoms disappeared in approximately three minutes.

TABLE 3.—*Number of Doses Required in Successful Acceleration.*

Doses	Number of Active Cases	Number of Placebo Cases
1	2	2
2	12	4
3	7	9
Total	21	15

TABLE 4.—*Number of Doses Required in Successful Induction.*

Doses	Number of Active Cases	Number of Placebo Cases
1	5	0
2	7	6
3	14	6
Total	26	12

In the evaluation of the active drug, continuous tracings of uterine activity and fetal heart rate were performed for ten patients. These tracings were analyzed in detail with graphs being made of the fetal heart rate, cervical dilatation, and uterine tonus and activity. In seven of the ten patients, there was no apparent increase in uterine tonus following the injection of the active drug, but there was a noticeable increase in the frequency and intensity of the contractions.

However, in three of the ten patients, there were moderate fluctuating increases in the uterine tonus. This effect was probably due to the irregular absorption at the site of the intramuscular injection, resulting in an unpredictable fluctuation in blood titers of the oxytocic agent. This was noted in three patients. Stander[3] has reported on 25 patients who had continuous monitoring of the intrauterine pressures and he has also noted elevated tonus or uterine tetany in approximately one-third of his cases.

TABLE 5.—*Apgar Mean Scores in Acceleration Group.**

	Mean Apgar Score
Active Successes	8.3
Active Failures	8.6
Placebo Successes	7.5
Placebo Failures	7.6

TABLE 6.—*Apgar Mean Scores in Induction Group.**

	Mean Apgar Score
Active Successes	7.6
Active Failures	8.3
Placebo Successes	7.5
Placebo Failures	7.5

*There were no low scores in any of the four categories.

Comment

The incidence of uterine tetany following the use of the active drug has been noted to be much greater in patients who had continuous monitoring of the amniotic fluid pressures as compared to those who were not monitored. This is evidenced by the one case of uterine tetany seen in the unmonitored group of 92 patients receiving the active drug, compared to three of ten in the monitored group. Stander mentions two reasons for this apparent discrepancy between the clinical appraisal and the evaluation by continuous recording of intrauterine pressures: (1) Intermittent manual palpation of the abdomen may not detect transient periods of elevated tonus, and (2) mild degrees of elevation of intrauterine pressure may be recorded by sensitive devices but remain imperceptible to the hand of the examiner, particularly in an obese patient. The incidence of hypertonicity or uterine tetany must be much greater than recognized in our clinical studies on unmonitored patients. There is no doubt but that the safest way to administer an oxytocic agent is by titrating it intravenously. There is better predictability in response due to the irregular uncontrolled absorption which results from intramuscular administration.

TABLE 7.—*Side Effects with Active Drugs.**

	No. of Cases
Uterine Tetany with Fetal Bradycardia	1
Cervical Laceration	1
Retained Placenta	1
Nausea and Vomiting	1

*There were no side effects with the placebo.

Summary

The results of a double-blind study of the efficacy and safety of sparteine sulfate has been presented. In this study, the drug was found to be an effective oxytocic agent without any associated seriously detrimental effects to mother or baby. Mention is made of the discrepancy in incidence of uterine tetany from this drug in patients undergoing scientific monitoring of uterine activity as compared to unmonitored patients. Mention is made of better predictability in response to intravenously administered oxytocics.

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COMMENTS

Our brief experience with use of sparteine sulfate as an agent to augment or induce labor is limited. Our first case performed with monitoring of uterine pressures and fetal heart rate almost ended in disaster. There was sustained tetanic uterine contraction, severe fetal bradycardia, and signs of fetal distress with delivery of an extremely depressed infant. The same objections that have been stated against the use of intramuscular or subcutaneous oxytocin may be said about sparteine sulfate—perhaps to a lesser degree—but any agent given intramuscularly that may cause a tetanic contraction and cannot be immediately discontinued, as with an intravenous oxytocin drip, is potentially dangerous. The authors' study showing that tetany and increased uterine tone occurs three to four times more frequently than could be observed from gross clinical monitoring reinforces this point.

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The average patient's hospital stay is nearly half as long today as it was 30 years ago. The average stay in 1935 was 14 days and today it is 7.7 days. The decline is attributed by the American Hospital Association to better medicines, new equipment and improved treatment.

Pregnancy and Systemic Lupus Erythematosus; Seven Cases

Capt. Robert E. Harris, MC, USAF, and Col. Charles E. Gibbs, MC, USAF

Seven cases illustrate many of the complications that may occur when pregnancy and systemic lupus erythematosus coexist. The incidence of abortions, premature labor, stillbirth, and complications of the labor, delivery, and postpartum periods is increased. Exacerbation of the lupus is common during this period.

CASE REPORTS

SYSTEMIC LUPUS erythematosus is a connective tissue disease of exacerbations and remissions that predominantly affects young women.[1,2] The disease does not impair fertility,[3,4] and therefore, pregnancy and systemic lupus erythematosus occasionally coexist. Seven pregnant patients with systemic lupus erythematosus recently treated at Wilford Hall USAF Hospital illustrate complications that may be encountered.

Case Reports

Case 1.—A 28-year-old white woman (para 4-0-1-4) had the onset of systemic lupus erythematosus in 1954 at 17 years of age; it was manifested by arthralgia, skin rash, and seizures. Her first pregnancy, at age 21, terminated at 13 weeks with an incomplete abortion. She had three grand mal seizures during the first post-abortal day, but did well otherwise. Full-term infants were delivered after four uncompleted pregnancies when the patient was 23, 24, 27, and 28 years of age, respectively, and she had no postpartum complications. She shows no evidence of renal disease but has arthralgia and intermittent skin rashes.

Case 2.—A 20-year-old white woman had the onset of systemic lupus erythematosus manifested by rash, arthralgia, pleuritic pain, persistent fever, and microscopic hematuria in 1958. The next year, during the 18th week of her first pregnancy, a severe exacerbation of the lupus necessitated an increase in steroid dosage. Another exacerbation occurred at 32 weeks while the patient was receiving 80 mg. of methylprednisolone daily. At 33½ weeks, with superimposed preeclampsia (i.e., blood pressure 140/120 mm. of mercury), she was delivered of a viable 4½ lb. infant. Her postpartum course was one of steady improvement for six weeks, when seizures occurred. She subsequently manifested lupus psychoses.

Case 3.—A 26-year-old white woman (para 2-0-0-2) had no problems with two previous pregnancies. At 33 weeks' gestation of her third pregnancy, painless hematuria developed. Her blood pressure was 148/100 mm. of mercury and she had a butterfly rash on her face. Results of laboratory studies showed urine protein, 2+ with microscopic hematuria; on several occasions the 24-hour urine protein level was 1.3 to 2 gm. Creatinine clearance increased to 233 ml. per min. and phenolsulfonphthalein excretion was only 9 percent in the first 15 minutes. The blood urea nitrogen level rose from 18 to 28 mg. per 100 ml. in three weeks. Results of bone marrow aspirate examination were positive for lupus erythematosus.

At 37 weeks, premature labor began, and a viable 5 lb., 7½ oz. infant was delivered. Post partum renal biopsy specimen revealed the characteristic lesion of lupus nephritis. During the year after delivery, the renal functions returned to normal, but the blood pressure remained elevated.

Case 4.—A 37-year-old white woman (para 4-0-1-4) was admitted at 32 weeks' gestation because of a weight increase of 15 lb. in one week with subsequent generalized edema. Her blood pressure during a previous clinic visit had been 100/60 mm. of mercury, but on the day of admission it had risen to 160/100. The patient had no change in fundal height of her uterus during the previous two months. She had pitting edema (4+) and 4+ reflexes and was considered to have preeclampsia. The laboratory data revealed microscopic hematuria, albuminuria (4+), urinary granular casts, phenolsulfonphthalein excretion of 7.25 percent at 15 minutes, 24-hour urine protein of 2.3 gm. rising to 4.75 gm., serum blood urea nitrogen of 25 mg. per 100 ml., and serum albumin of 1.7 and globulin of 3.0 mg. per 100 ml. Although the patient was treated for preeclampsia, albuminuria and hypertension persisted and even became worse. Labor was induced, and a stillborn 3 lb., 3 oz. infant was delivered. Postpartum study of aspirated bone marrow was positive for lupus erythematosus, and the characteristic lesion of lupus nephritis was found in a renal biopsy specimen. The patient slowly improved with diuretics alone, but the blood pressure remained at levels of 140/90. Six months post partum, the lupus remained in remission.

Case 5.—A 22-year-old white woman (para 0-0-0-0) had an uncomplicated pregnancy until the 30th week of gestation, when proteinuria and edema

(1+), but no elevation of blood pressure developed. She was admitted with the diagnosis of possible early preeclampsia. She had gained no weight recently and only 15 lb. during the entire pregnancy. On admission the values for blood urea nitrogen and creatinine were normal, but the 24-hour urinary proteins were 2.1 and 4.5 gm. Even though the patient was treated with bed rest and diuretics for the next five weeks, the blood urea nitrogen and creatinine levels rose, serum albumin decreased, and the 24-hour urinary protein level rose to 8.5 gm. The fundal height of the uterus did not change during these five weeks, and the blood pressure had risen to 150/100. Bone marrow aspirate showed positive signs of lupus erythematosus, and the patient was considered to have systemic lupus erythematosus with secondary nephrotic syndrome. Because of her worsening renal status, labor was induced at 37 weeks, and the 3 lb., 12½ oz. infant did extremely well. A postpartum renal biopsy examination was not satisfactory because of renal bleeding. Although the patient continued to have albuminuria (0.3 gm. per 24 hours) and slight elevation of blood pressure at two months post partum, she did well.

Case 6.—A 25-year-old white woman (para 0-2-0-2) had the onset of systemic lupus erythematosus at age 15 and was treated with steroids. Her first pregnancy in 1962, when she was 22, was complicated by persistent hypertension (140/110) and hypersplenism. The latter was associated with a hypoplastic bone marrow, anemia, and thrombocytopenia, believed to be secondary to an unknown agent (possibly a hair dye). She received two units of blood for anemia during the second trimester. The lupus exacerbated mildly during the second trimester, and at 37 weeks she gave birth to a 5 lb., 10½ oz. infant who did well. One month post partum she had a splenectomy for hypersplenism.

The lupus remained in remission until the second pregnancy began in 1965, although the interval laboratory data was consistent with lupus nephritis with proteinuria, abnormal creatinine clearances, and phenolsulfonphthalein excretion. At 18 weeks' gestation an exacerbation of lupus necessitated an increase in steroids for control. Blood pressure ranged from 140 to 160 systolic over 90 to 100 diastolic, and the laboratory data revealed albuminuria (2+) with fine granular casts and a creatinine clearance of 56 ml. per min. Her condition remained clinically stable until 30 weeks' gestation when an upper respiratory infection and gastroenteritis developed. This was complicated by sinusitis with early exacerbation of the lupus with arthralgia and skin rash. The sinusitis improved with increased therapy. However, at 33 weeks the patient became acutely as well as seriously ill, and severe upper mid-abdominal penetrating pain developed, accompanied by an increase in amylase from 360 to 630 units. She was considered to have acute pancreatitis secondary to lupus. With therapy for systemic lupus supplemented by specific therapy for pancreatitis, i.e., methantheline bromide, nasogastric suction, and intravenous fluids, the patient improved slightly. However, uterine contractions ensued and were followed by a grand mal seizure. No change occurred in blood pressure or in albuminuria. She was treated for eclampsia for 24 hours, and then labor was induced. A 3 lb., 15¾ oz. infant was delivered and did well. Post partum, she slowly improved and at six months the lupus was still in remission.

Case 7.—A 28-year-old white woman (para 1-1-1-1) had a skin rash, hemoglobin of 9.5 gm. per 100 ml., albuminuria (2+), and microscopic hematuria in the 20th week of pregnancy. During the next two weeks the levels of hemoglobin fell to 6.5 gm. per 100 ml., urine albumin rose to 3+ and 4+, and serum blood urea nitrogen rose from 30 to 37 mg. Bone marrow aspirate was positive for lupus erythematosus. She was given packed red cells, which elevated the hemoglobin to 11.6 gm. per 100 ml., and prednisone therapy was started. Anemia persisted during the pregnancy, requiring six more units of packed red blood cells to maintain the hemoglobin above 10 gm. per 100 ml. She went into premature labor at 35 weeks, and the 5 lb. infant did well. A postpartum renal biopsy specimen revealed the characteristic changes of lupus nephritis. The patient continued her progressive, fulminant, downhill course with renal failure, and died two months post partum.

Discussion

These seven patients with lupus erythematosus had a total of 12 pregnancies and, with the exception of the four normal pregnancies in case 1, all were complicated. Exacerbations of lupus occurred during the second trimester in three pregnancies, during the third trimester in five pregnancies, and post partum in one pregnancy.

Four patients had the initial episode of systemic lupus erythematosus during pregnancy. Three patients had preeclampsia, three had nephrotic syndrome, and five had lupus nephritis. Three patients had seizures, ante partum or post partum, all unassociated with preeclampsia. The pregnancies resulted in one abortion, six premature living infants, one stillbirth, and four normal term infants.

The complications in these patients vary from those reported previously. Pregnancy has been stated to increase the incidence of exacerbation of lupus three times in the first trimester and seven times in the postpartum period.[4,5] The patients in this group had exacerbations during the second and third trimesters primarily. As in the reports of other investigators,[2,4,5] no congenital abnormalities of the fetus occurred in this group.

Most of the patients in this series had severe manifestations of systemic lupus erythematosus; lupus nephritis in five patients, nephrotic syndrome in three patients, and neurologial involvement with seizures in three patients. This severity is probably a reflection of the referral of more severe cases of systemic lupus erythematosus to this

facility from other United States Air Force hospitals. Therefore, the results of these pregnancies do not reflect those reported in other series. Preeclampsia occurred in three patients compared to the overall reported rate of 18-19 percent.[3,6,7] Although the rate of abortions was reported to be 17-31 percent,[2-9] only one occurred in this group, whereas the incidence of premature deliveries was 50 percent compared to the reported 15-22 percent.[4,6,7]

The differential diagnosis of eclampsia and central nervous system involvement with lupus may be quite difficult, especially if there is underlying renal disease (lupus nephritis).[10,11] Case 1 is similar to a previously reported case, in which seizures first began after abortion.[10] Encephalitis (i.e., convulsions, psychosis, or neurologic manifestations) occurs in 20 percent of patients with systemic lupus erythematosus.[2] Case 2 illustrates the possible end-result of central nervous system involvement by lupus—progressing into lupus psychoses, even with therapy. The management of these seizures during the third trimester should be the same as that for eclampsia.

Lupus nephritis is a poor prognostic sign in systemic lupus erythematosus patients.[2,5] The only maternal death in this group (case 7) occurred in the patient with the most severe renal involvement.

Antepartum care in systemic lupus erythematosus patients is directed toward close observation and vigorous therapy for exacerbation of the disease process. Manifestations may be renal involvement, anemia, joint pain and swelling, or skin eruptions. Minor infections, such as gastroenteritis or sinusitis (case 6), may be enough to trigger the exacerbation. More rarely, acute pancreatitis (case 6) may be a manifestation of a lupus exacerbation. Adequate rest and nutrition, analgesics, antibiotics for specific infections, correction of anemia, and increased steroid dosage are all indicated therapy for exacerbations.[1-3,5]

These patients do not tolerate the normal stresses of labor well.[3] Therefore, the principles of labor and delivery include minimizing trauma, shock, and hemorrhage.[3] Conduction anesthesia is preferable, and an increase in steroid therapy is required.[3] Postpartum complications include hemorrhage, possible adrenal failure (inadequate steroid substitution), hyponatremia, poor wound healing, infections, and convulsions.[3,10]

Because of the increased incidence of exacerbation of lupus associated with pregnancy and the resultant poor prognosis for fetal survival, some investigators have recommended that systemic lupus erythematosus patients not become pregnant.[3,8,12] However, therapeutic abortion is contraindicated, for maternal mortality has been extremely high in these patients.[3,4,13]

Summary

Seven pregnant patients with systemic lupus erythematosus represent the entire spectrum of the disease, from mild involvement to maternal mortality. Exacerbations were common in this type for they exhibited a high degree of severe involvement with lupus. The incidence of abortions, premature labor, and stillbirth was increased. Prompt, vigorous therapy is indicated for exacerbation during pregnancy or post partum. The labor, delivery, and postpartum periods are characterized by an increase in the number of complications, and these must be prevented if possible, or treated promptly if they occur.

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Surgical Management Of Primary Breast Cancer

REVIEW ARTICLES

THE TREATMENT of breast cancer is one of the most controversial issues in medicine today. Surgical measures have formed the principal basis of therapy. The adoption of ionizing radiation, the use of hormones, the abolition of natural sources of hormones, and, finally, chemotherapeutic agents as supplemental aids, gradually have appeared. Seventy-two years ago the nearly simultaneous reports of radical mastectomy by Halsted[1] and Meyer[2] culminated what had been evolutionary steps in the preceding 30 years. These had progressed from local excision to simple mastectomy, followed by removal of axillary lymph nodes and pectoral fascia. In undertaking curative treatment, the Halsted and the Meyer operations occupied almost impregnable positions for several decades. Minimum technical modifications were made in the early years after the initial reports, and the radical operation, as it was and has been performed, gradually has evolved more toward the Meyer procedure than the Halsted mastectomy. For many years, to question radical mastectomy in the approcah to curative therapy was almost tantamount to heresy.

However, soon after World War II, two opposing trends arose, one toward conservatism and the other toward more radicalism. The revival of interest in extending the radical operation was reflected in the reports of Margottini,[3] Wangensteen and associates,[4] Dahl-Iversen and Tobiassen,[5] and Urban.[6] The approach of Wangensteen and his coworkers to super-radical treatment, with the inclusion of supraclavicular, internal mammary, and mediastinal lymph node dissections, proved to be unattractive, with nearly prohibitive mortality. A trend in the direction of a more aggressive approach became evident in the increased interest in the super-radical or extended radical mastectomy in treatment of lesions of the medial and central sections of the breast. The work of Urban continues to attract considerable attention. His results suggest that the total increment of salvage in applicable instances approximates 10 percent. During the past decade, several surgeons have demonstrated the significance of internal mammary lymph node metastasis in human breast cancer. Handley[7] reported a 33 percent incidence of internal mammary lymph node metastasis in 300 patients treated by radical mastectomy with removal of the internal mammary nodes. Medial and central lesions showed 43 percent involvement as compared with 20 percent for lateral lesions. When the axillary nodes were involved, 60 percent of the inner quadrant lesions showed internal mammary spread, 30 percent of outer-half lesions showed this involvement. Extension of the radical mastectomy procedure to include excision of the internal mammary lymph node chain is based upon accepted anatomic and clinical findings and should result in increased control of primary operable breast cancer. Urban[8] reports five-year follow-up data on 265 patients. In this group, 33 percent had internal mammary involvement, and 47.5 percent had axillary involvement. The five-year survival rate is 68 percent; 60 percent are clinically free of disease after five years; and the local recurrence rate is 7.9 percent. Urban reported no increase in operative mortality. Of 480 patients who had radical mastectomy with continuity resection of the internal mammary chain, two patients died, one from perforated ulcer and one from myocardial infarction.

Returning to the conservative end of the mastectomy spectrum, the work of McWhirter[9] with simple mastectomy used in conjunction with postoperative ionizing radiation (or, one might put the matter in converse terms as well) led to widespread interest and friendly controversy. Ackerman[10] presented some of the possible questions raised by McWhirter's results and

Extended radical mastectomy should be the treatment of choice when facilities and personnel are available; otherwise, standard radical mastectomy and radiation therapy should be used. The most important factor is early detection and treatment.

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interpretations with suggestion of better use of more precise methods in data-gathering and the sophisticated tools of statistical analysis.

McWhirter presented a report on 1,882 patients who had undergone simple mastectomy and super-radiation treatment from 1941 to 1947. He had traced 100 percent of his cases, and five-year and ten-year survival rates were computed. It is to be regretted that in this bold and well-executed experiment McWhirter did not take advantage of the opportunity to carry out a controlled experiment comparing the results of simple mastectomy and irradiation with those of radical mastectomy. Since he did not do that, it is of interest to compare his series with that of Clagett,[11] who performed radical mastectomy on 1,900 patients during the same period at the Mayo Clinic, and had a 99 percent follow-up on his cases. The two series are of almost equal size. They cover the same period. Both have practically 100 percent follow-up. In computing survival rates, all deaths are attributed to carcinoma. The average ages were approximately the same. McWhirter reported that about 15 percent of his patients had distant metastases when first seen. In Clagett's series, 16 percent had distant metastases. In both series, a standard policy of treatment was maintained, and an essentially uniform technical procedure was used. The five-year survival rate for the patients treated by simple mastectomy and irradiation was 48.1 percent while the rate for patients treated by radical mastectomy was 59.3 percent. It is apparent that the comparison is favorable to radical mastectomy by more than 10 patients per 100 operated on, or an increase of more than 20 percent over survival achieved by simple mastectomy.

Another factor involved in the policy of simple mastectomy and irradiation that is not reflected in the survival rate, which is important in so far as the patient is concerned, is the radiation morbidity that it incurs. Ackerman[10] reviewed 719 of the cases treated by McWhirter. There were 47 cases of serious radiation damage with multiple fractures of ribs, clavicle or humerus or both, or fixation of the shoulder joint, and three cases in which amputation of the arm was required. Radiation fibrosis of the lungs and other lesser manifestations of radiation effects are difficult to assess, but are not to be disregarded. Radiation therapy as practiced by McWhirter is not a conservative procedure. Results similar to Clagett's supporting conventional radical mastectomy, have been reported by Ryan and associates[12] and by Watson.[13]

Auchincloss[14] and other surgeons have said that the conventional radical mastectomy is unnecessarily radical. "The more thorough axillary dissection which it affords accomplishes nothing for the survival rate of patients with breast cancer and axillary metastases that a more limited operation would not do equally well." They do what is called a modified radical mastectomy without removing the pectoral muscles. These surgeons believe the only women surviving breast cancer are those in whom the involved nodes are few and confined to the central or lower axillary vein groups, and therefore very extensive axillary dissection which can be achieved when the pectoral muscles are removed is unnecessary because it adds nothing to the salvage rate of the patients with breast cancer.

Crile[15] advocates simple mastectomy, and even local excision of cancer of the breast, under certain circumstances, over the conventional radical mastectomy. He believes that removing the axillary nodes, or irradiating the axillary nodes, lowers the host's resistance to cancer. He recommends

a simple mastectomy without irradiation for clinical stage 1 cancer, or in cancer of the breast in which there are no palpable axillary nodes. However, it has been repeatedly shown that there is approximately a 50 percent error in diagnosing clinical stage 1 cancer. Humphrey and Swerdlow[16] reported that of 160 patients who were thought to have affected axillary nodes at clinical examination, microscopic examination showed that in 27 patients the axillary nodes were not involved —an error of 16.9 percent. When axillary nodes were described as negative clinically in 162 patients, microscopic study showed that 82 had positive nodes, an error of over 50 percent. Should nodes develop after the simple mastectomy is done without irradiation therapy, then removal of these nodes is advised by Crile.[17] He reports a small series of 135 operable cases seen at the Cleveland Clinic between 1955 and 1957. He reports that in seven years 52 percent of patients treated by simple mastectomy or by local excision of cancer are living and apparently free of cancer as compared with 40 percent of those treated by either conventional radical mastectomy or modified radical operations, in which the pectoral muscles were preserved.

We are indebted to all these men for having stimulated a great deal of controversy about the problem of carcinoma of the breast. Unfortunately, their efforts have not provided a more effective treatment. There is no valid evidence to support the concept that simple mastectomy or simple mastectomy plus irradiation is as effective as classical radical mastectomy. Radical mastectomy with en bloc removal of internal mammary nodes probably is an improvement, and as this operation is done in more medical centers, and more follow-up reports are made, its value for general use will be decided.

Relationship of Tumor Size To Rate of Cure

The size of the tumor is also a determining factor in curability. Lewison[18] noted that the five-year survival rate for women with tumors less than 2.5 cm. in diameter was 74 percent; whereas, for patients with tumors of more than 5 cm. in diameter, it was only 53 percent following the same treatment. In general the earlier the detection and the smaller the tumor at the time of treatment, the better will be the results, This statement is further substantiated by results from the University of Minnesota Cancer Detection Center[19] where a study of 25 women whose asymptomatic cancer of the breast was detected at the center showed an 88 percent five-year survival rate after treatment. Since the usual overall five-year survival figure is about 50 percent, this demonstrates the benefit of early detection and treatment. Haagensen[20] showed in a series of 668 cases of primary breast carcinoma (between 1935 and 1942) that the mean diameter of the primary tumor was 5 cm. At surgery 61.6 percent of these patients had axillary metastases. In another series of patients at the same hospital, in which the carcinomas averaged only 1.1 cm., only 44.5 percent had axillary metastases.[20] Haagensen reports that his five-year cure rate in patients without axillary metastases is 90 percent.

Humphrey and Swerdlow[16] state that as the size of the tumor increases, the chance of the patient's surviving five years decreases. Of patients with lesions measuring 0 to 2 cm., 67 percent survived five years compared to 23.8 percent of those with lesions greater than 8 cm. Of primary lesions measuring 0 to 2 cm., 42.9 percent had axillary metastases. In lesions over 8 cm., the percent of axillary node involvement was 88.1 percent.

Criteria of Operability

Haagensen and Stout have outlined their criteria of operability.[21] I have been inclined to be less conservative, having been impressed by the criteria of operability as expressed by Adair, who stated simply, "We consider any case of operability in which the disease is localized in the breast or the breast axilla, and in which we consider there is a chance of cure, no matter how small that chance. To disregard the well recognized criteria of inoperability, however, is to invite disaster and is a foolhardy gesture unworthy of a conscientious surgeon. On the contrary, to deny a chance for prolonged survival and possible cure to a single patient with an operable tumor is unforgivable and portrays the surgeon as believing himself to be an infallible judge. Our responsibility extends to all patients, not just to those with easily curable lesions."[11]

Report of Series

Between 1946 and 1961, I have performed conventional or standard radical mastectomy

in 100 consecutive cases. All were private patients, and the operations were performed in a community hospital.

Operative Procedure.—Operative principles include wide excision of tumor, at least 5 to 7 cm. from the lesion in all directions, in one of the three types of skin incisions, thin skin flaps, sharp dissection of the axilla, and removal en bloc of the axillary contents, pectoralis major and minor muscles, with the breast. After hemostasis, the wound is irrigated well with 2 mg. per 100 ml. nitrogen mustard solution. Then, operating personnel change gloves and instruments, and the skin flaps are closed without tension, and usually using a skin graft. Two catheters with suction are used to remove the drainage from the wound.

Mortality and Morbidity.—The average size of these tumors was 3.6 cm., and 48 patients had metastases to the axillary lymph nodes. There was no mortality from surgery. The immediate morbidity included four wound infections, two minor and two major, and one pneumothorax. Delayed morbidity included: moderate swelling of arm, 49; severe swelling of arm, 1; restricted function of arm, 1. In 95 cases, patients retained full function and use of the arm.

Adjunct Treatment.—All patients with axillary node involvement and patients with medially located tumors had radiation therapy to internal mammary nodes consisting of 5,000 r. (cobalt, 300 r. per day, with two-thirds of the dose given anteriorly and one-third posteriorly, using a 4 by 10 cm. portal). Ten patients in the menstruating age who had more than three positive axillary nodes had surgical castration before leaving the hospital.

Follow-up.—Four cases were lost to follow-up. Of the remaining 96, 50 are living and without clinical evidence of disease; 30 died from cancer; 12 died from other causes (no clinical evidence of cancer); 4 are living with cancer. None have had local recurrence of tumor, but 18 patients have already developed cancer in the other breast or another organ.

Conclusion

A cancer operation was described many years ago as an operation which consists of removing the involved organ and its contiguous structures and the regional lymphatics and nodes. I do not believe that the principle of cancer surgery has changed. Simple mastectomy does not fulfill this definition of a cancer operation nor does the conventional radical mastectomy entirely do so. The extended radical mastectomy, such as is being done by Urban, better fulfills this definition, and it would seem that if other surgeons can duplicate his results of low morbidity and mortality and better salvage ratio, that this operation should at least be done in teaching centers. Statistics indicate that those of us who do not do the extended radical mastectomy but do the standard radical mastectomy should treat the internal mammary nodes with radiation. What is probably more important than the method with which we treat carcinoma of the breast is the size of the tumor that is being excised. This means we have a duty to perform, namely, to educate these patients to seek regular examinations to assure early detection and surgery.

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Vagotomy and Pyloroplasty In 150 Duodenal Ulcer Patients

William Klingensmith, Jr., M.D., and Patrick Oles, M.D.

Treatment of 150 peptic ulcer patients with vagotomy and pyloroplasty has resulted in a low complications rate with no reported mortality. The procedure apparently was safe and effective for more than 90 percent of them.

CLINICAL MEDICINE

PROPER SELECTION of a surgical procedure for the treatment of peptic ulcer remains difficult, but the trend toward conservative resection and vagotomy, or vagotomy with a drainage procedure, has increased and in many major surgical clinics the radical gastric resection has been abandoned. Recently, much attention and publicity have been given to treatment of duodenal ulceration with gastric freezing, but actual usefulness of the procedure is doubtful and detailed studies of the acid secretion of individuals after freezing shows no alteration.[1] If our knowledge of gastric physiology is correct, then gastric freezing will not offer any long-term improvement in most patients with true duodenal ulcer disease. It seems safe to assume that gastric freezing will not stand the test of time. Certain patients with peptic ulcer still will face open surgical procedures.

High gastric resection is a relatively safe operation and gives good results insofar as control of the ulcer is concerned (Fig. 1); however, it is associated with a definite mortality, and in some individuals, the aftereffects of high resection are devastating. All surgeons are familiar with the postgastrectomy patient who is underweight and frequently nauseated after eating. Anyone with much experience with resection has patients who have been crippled and permanently incapacitated by the procedure; this is especially true for women, and many surgeons have been unwilling to submit the thin, emotional woman ulcer patient to resection unless driven to it by unavoidable complications of bleeding or obstruction.

Following the pioneer work in gastric physiology by Dragstedt[2] and others, it became apparent that all patients with duodenal ulcer had an overproduction of the acid pepsin component of gastric juice. All methods of therapy, whether medical or surgical, had as their objective the reduction of this acidity with food or acid-neutralizing compounds, the removal of the acid-secreting portion of the stomach, or the abolition of the cephalic phase of gastric secretion by removal of the vagi. Others[3] have advocated the removal of the antrum as well.

The great bulk of acid secretion is brought about by vagal stimulation and can be completely abolished if a total resection of the vagus nerves is performed. Stimulation of the antrum by stasis of food, distention, or the presence of alkali, produces a secondary secretion of acid by the elaboration of the hormone gastrin which in turn stimulates the acid producing cells of the stomach. Since vagotomy reduces the tone of the gas-

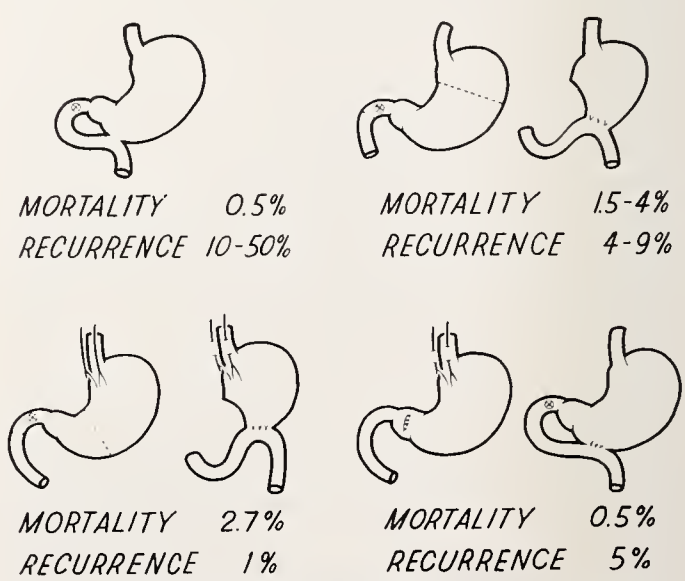

Fig. 1. Various types of surgical procedures in use for the treatment of duodenal ulcer, and the mortality and recurrence rates for each, are shown.

tric pouch and delays gastric emptying, the antrum is stimulated to form gastrin. Vagotomy must always be accompanied by a drainage procedure—either a pyloroplasty or a properly placed gastrojejunostomy. If this is not done, paradoxical gastric ulceration can and does occur. Dragstedt has shown that duodenal ulceration is primarily the result of cephalic stimulation and hyperacidity through the vagus nerves, whereas gastric ulceration is the result of antral stasis with stimulation of the antrum. This is substantiated clinically by the patient with chronic duodenal ulcer and obstruction of the pylorus who develops a secondary gastric ulcer when the stomach does not empty. This is directly comparable to the gastric ulcer which developed in the early days of vagotomy, before the necessity of a concomitant drainage procedure was established.

In some institutions, vagotomy has been combined with resection of the antrum or "low gastric resection." This has the advantage of not only removing the cephalic phase of acid secretion but also removing the gastric antrum so that the production of gastrin is impossible. Statistically and physiologically, the procedure gives excellent protection from the possibility of further development of peptic ulceration and it is usually followed by a mortality rate lower than that of high gastric resection. However, this procedure carries with it the dangers of the duodenal stump, the weakness of a long gastric closure, and a definite incidence of postoperative dumping; except for a lower mortality rate, it varies little from the complications of a high classical gastric resection.

Case Reviews

During the past six and one-half years we have performed vagotomy with an associated drainage procedure on 150 consecutive patients, and we have not done a gastric resection for duodenal ulcer during this time. All of these patients had duodenal ulcer without concomitant gastric ulcer and without previous gastric surgery, except for a previous closure of an acute perforation of the duodenum in some cases. The longest follow-up has been 76 months and the average length of follow-up for all 150 patients has been 24.2 months.

The indications for surgery in these 150 patients are listed in Table 1, and are the generally accepted reasons for surgery in duodenal ulcer disease. Most of our patients have had surgery because of intractable pain or pain associated with bouts of bleeding. Fifteen consecutive patients with acute, massive bleeding are included and all have done well.

Primarily, we have performed vagotomy and pyloroplasty, using this procedure in 129 patients. It is a simple, effective procedure that leaves the antrum in the acid stream and effectively permits proper gastric emptying. In 21 patients, however, we have utilized gastroenterostomy with vagotomy. We believe that if pyloroplasty is technically difficult because of inflammation or scarring, then a properly placed gastroenterostomy should be used.

The age of this group of individuals has varied greatly and is shown in Table 2. Our youngest patient was 17 years old and the oldest was 82; both had repeated episodes of bleeding. This more conservative operation has great appeal at both extremes of age.

These 150 surgical procedures have been accompanied by a complication rate of 7.3 percent, and these are listed in Table 3. Pulmonary embolism occured in one patient and probably in a second. Wound abscess was a problem in three patients, all of whom had surgery for massive bleeding while the stomach was filled with clots; we now take special precautions to protect the wound when surgery is necessary under these conditions. Two patients, one with a nonfunction-

TABLE 1. *Indications for Vagotomy and Pyloroplasty.*

Type of Indication	No. of Patients	%
Pain	67	(44.7)
Pain and bleeding	40	(26.7)
Obstruction	23	(15.3)
Massive bleeding	15	(10)
Acute perforation	5	(3)
Total	150	

TABLE 2. *Age of Patients.*

Age	No. of Patients	%
Teens	3	(2.0)
20's	8	(5.3)
30's	24	(16.0)
40's	40	(26.7)
50's	38	(25.3)
60's	26	(17.3)
70's	10	(6.7)
80's	1	(0.7)
Total	150	

TABLE 3. *Complications (7.3%).*

Partial small bowel obstruction	1
Pulmonary embolism	2
Wound abscess	3
Postoperative bleeding	2
Obstructed gastroenterostomy	1
Obstructed pyloroplasty	1
Dumping	1
Total	11
Mortality	0

ing pyloroplasty and one with a nonfunctioning gastroenterostomy, required a secondary operation. There have been no deaths.

Evaluation of Results

In preparing this report, we have resorted to a questionnaire sent out to all patients whose surgery had been completed for five or more months. This is not the best way to evaluate results, but in a private practice, it is not always possible to do elaborate postoperative studies.

In all, 127 questionnaires were sent to patients whose surgery had been completed for five months or more. The average length of follow-up was 27 months. Four patients were no longer living, and four questionnaires were returned undelivered. If these are excluded, then the response from 119 patients who received the questions was 88 percent. Patients were questioned regarding weight, bowel habits, eating habits, postoperative ulcer symptoms, necessity for further medical therapy, and their appraisal of the overall result obtained (Table 4). One very interesting and perhaps significant question was the presence of further bleeding following the vagotomy. Of 54 patients responding who had had significant bleeding as a preoperative indication for surgery, only three had experienced any further bleeding and two had insignificant bleeding. Evaluation of the entire questionnaire is difficult, because some patients who stated that they had great difficulty in eating appraised the operation as a complete success. Although 13 percent of those responding stated that they had predominantly loose stools, only one had troubling diarrhea. It is our opinion, from evaluation of the entire questionnaire, that more than 90 percent of those responding had complete or almost complete relief of their ulcer symptoms with no serious side effects, and that those who had bled were well protected following surgery.

TABLE 4. *Information Obtained from Questionnaire (101 Respondents).*

No. of Patients	Aftereffects
	Postoperative Weight Change
46	Same
35	Increased
20	Decreased
	Postoperative Bowel Habits
69	Regular
18	Constipated
13	Loose
1	Diarrhea
	Food Eaten
60	Anything
32	Most anything
2	Bland foods
7	With great difficulty
	Postoperative Ulcer Symptoms
74	Completely disappeared
10	Almost disappeared
16	Occasional
1	No improvement
	Further Medical Treatment Required
88	None
13	Some
	Patients' Appraisal of Results
82	Complete success
6	Partial success
10	Of some help
3	Failure
	Bleeding
54	Before surgery
3	After surgery

Summary

During the past six and one-half years, we have performed vagotomy and a drainage procedure, preferably pyloroplasty, on 150 consecutive private patients with duodenal ulcer. There has been no mortality in this group, but the complications rate was 7.3 percent. An attempt has been made to appraise results by means of a questionnaire sent to those at least five months postoperatively. Eight-eight percent responded to this questionnaire, and from it and our own appraisal it is felt that the procedure is safe and effective in more than 90 percent of patients; protection from further bleeding episodes has been dramatic, and troublesome sequelae have been minimal.

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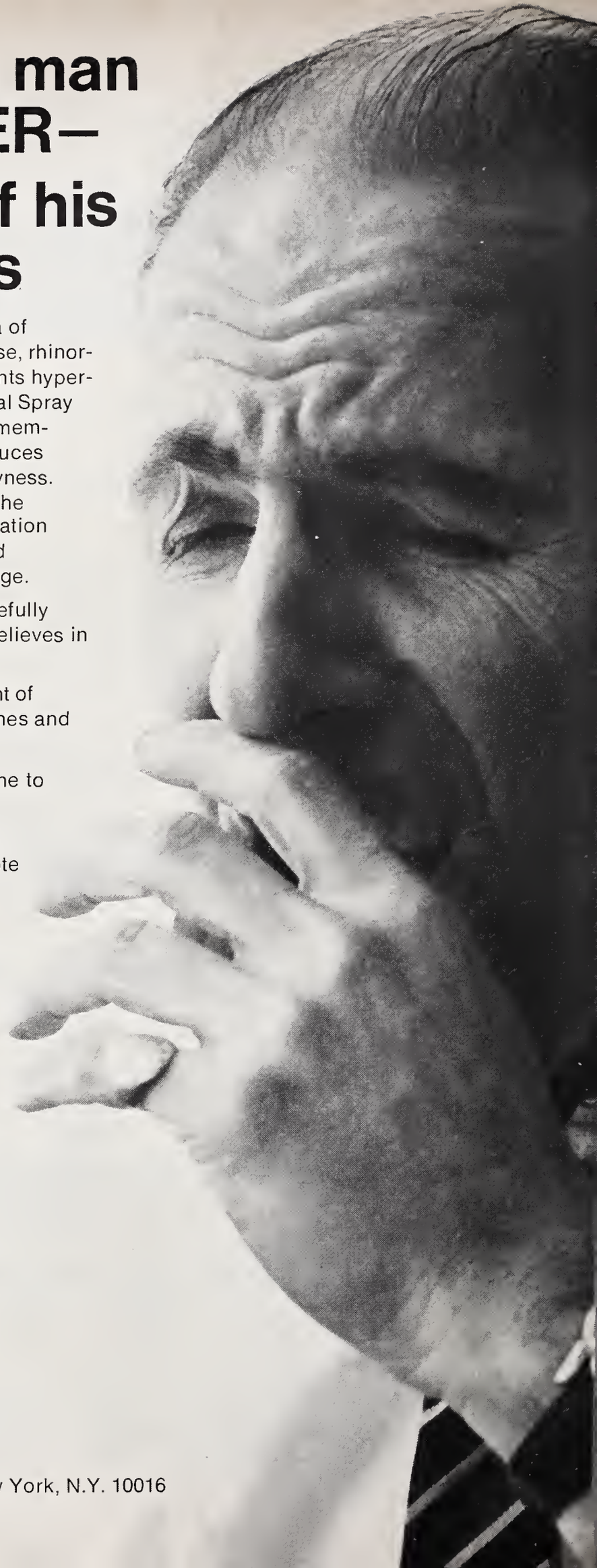

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LX

Clinicopathologic Conference

Ed Winga, M.D.; Edward Christensen, M.D.; Burton Combes, M.D.;
John S. Fordtran, M.D.; and C. T. Ashworth, M.D.

A woman, 56, had anorexia, nausea, vomiting without hematemesis, abdominal fluid distention, weight loss, and intermittent diarrhea and constipation. Although she was a chronic alcoholic, results of liver function tests were relatively normal. Hematemesis developed, and the patient died seven weeks after admission.

DR. WINGA: A 56-year-old white woman was first seen in the Parkland Memorial Hospital Outpatient Clinic in 1959. She had been an alcoholic for ten years, and had spider angiomas and hepatosplenomegaly. Abnormal laboratory values included hemoglobin, of 9.8 gm. per 100 ml. and bromsulphalein excretion of 9 percent.

She was hospitalized in June, 1960, for barbiturate intoxication following a suicide attempt. Recovery was uncomplicated. Her second admission was in February, 1964; while in the county jail she began having grand mal seizures, and on arrival at Parkland she had hallucinations. The liver was palpated one fingerbreadth below the right costal margin, but the spleen was not felt. Her cerebrospinal fluid was normal, and she was thought to have alcoholic withdrawal symptoms. She responded well to sedation.

Her third admission was on July 5, 1965, with a three-week history of anorexia, nausea, and vomiting without hematemesis. She had noted progressive abdominal swelling, which she attributed to a blow in the xiphoid area about four months before admission. This was followed by persistent nonspecific upper abdominal pains. She had lost about 20 lb. in three months, and had intermittent diarrhea and constipation without melena. Questionable fever and chills were also noted. She was taking an unknown antibiotic for a urinary tract infection characterized by nocturia and possibly hematuria, but no dysuria.

Her past history included acute rheumatic fever at age 13, with polyarthritis and a heart murmur; hemorrhoidectomy in 1953; and cobalt and radium therapy for squamous cell carcinoma of the cervix (stage 2) in 1956 with no evidence of recurrence on follow-up examinations. In 1959, she was evaluated for arthritic complaints, but all studies were within normal limits. In spite of many prior urinary tract infections, an intravenous pyelogram in 1959 showed no abnormalities. One of her brothers had tuberculosis, and another had died of tuberculosis at age 27.

Physical examination on admission revealed blood pressure, 150/90 mm. of mercury; pulse, 96; respirations, 24; and temperature, 98.8 F. She was emaciated and appeared older than her stated age, and was in moderate respiratory distress because of a distended abdomen. Spider angiomas were present on the chest, and palmar and facial

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This is the third in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School, Dallas. Guest editor for the series is Dr. Bruce D. Fallis, professor of pathology. Participants this month are Drs. Ed Winga, resident in internal medicine; Edward Christensen, assistant professor of radiology; Burton Combes, associate professor of internal medicine; John S. Fordtran, assistant professor of internal medicine; and C. T. Ashworth, professor and chairman of the Department of Pathology.

TABLE 1. *Laboratory Determinations.*

Date	7-5	7-17	7-28	8-12	8-17
Hemoglobin	11.8	11.2	7.1	5.5	7.7
Hematocrit	36	35	23.5	18.5	
White blood cells	5,500	19,750	13,750	9,000	10,000
Platelets	317,500	Adequate	Adequate		
Blood urea nitrogen	11	12	17	24	25
Creatinine		0.6	1.3	2.2	2.0
Fasting blood sugar	118				
Carbon dioxide	25	22	20	15	18
Chlorides	96	95	100	106	98
Sodium	135	129	131	131	124
Potassium	3.5	3.6	3.1	3.4	4.1
Albumin/globulin	4.0/3.5	3.6/2.9	3.2/2.7	2.8/3.4	3.2/2.9
Bilirubin:					
Direct		2.4	1.6	1.0	
Indirect	0.9	3.6	2.8	1.5	
Serum glutamic oxaloacetic transaminase	40		62	76	
Thymol turbidity	4.5	4.0	3.3	8.1	
Alkaline phosphatase	13	11.5	9.9	11.2	

TABLE 2.—*Ascitic Fluid Examinations.*

Paracentesis	7-6	7-10	7-28
Gross appearance	Bloody	Bloody	Minute blood
Red blood cells	285,000	Hgb.: 2.1 gm. %	11,140
White blood cells	3,100	1,700 (73 % polys)	550 (65 % polys)
Protein	2.7 gm. %	2.4 gm. %	
Cell block	Neg	Neg	Neg
Culture	Neg	Neg	Neg

erythema was present. No icterus was seen. The abdomen showed fluid distention and dilated superficial veins. The liver was palpable 4 to 5 cm. below the right costal margin with a smooth, tender margin. The spleen was not palpable. Bowel sounds were active. The upper vagina was stenotic with a friable mucosa. There was no fixation to the lateral pelvic walls. Neurologic examination revealed only generalized weakness without asterixis. Other results of the examination were within normal limits.

Initial laboratory data (July 5) are shown in Table 1. In addition, serum amylase was less than 320 units. Urinalysis revealed specific gravity 1.030, pH 5.5, trace albumin, moderate acetone, 8-15 red blood cells per h.p.f., and 19-24 white blood cells per h.p.f. The cerebrospinal fluid was normal. Repeated urine cultures produced no significant growth. An intermediate strength tuberculin (PPD) skin test was nonreactive.

Abdominal paracentesis the day after admission produced bloody fluid. Paracentesis was repeated twice, and the results of laboratory studies on the ascitic fluid are shown in Table 2.

Initial treatment included restricted fluid intake and vitamins. Because of increasing lethargy and confusion, fluids, low protein diet, and neomycin were begun on July 14. The stomach aspirate was guaiac negative, but the stools remained guaiac positive. INH and pyridoxine were begun at this time. On the evening of July 16, the patient began vomiting coffee-ground material from bleeding esophageal varices, which were seen on esophagogastroscopy the following day. A tracheostomy was done. Because of continued hematemesis, blood transfusions were given, and a Blakemore tube was inserted July 19 and remained until July 21.

Following this the patient continued to have intermittent bloody diarrhea. On July 23 a stool culture grew coagulase-positive *Staphylococcus aureus*, which was treated with oral vancomycin. At repeated consultations, no surgical shunting procedure was recommended because of the patient's wasted condition. On Aug. 12 another episode of severe hematemesis occurred, with a drop in hemoglobin to 5.5 gm. per 100 ml. Treatment was with blood transfusions, ice water lavage, insertion of a Blakemore tube, and Pitressin. The bleeding ceased in a few days, but the patient's condition slowly deteriorated with development of asterixis, hypotension, and coma. She died quietly on Aug. 23.

Radiological Findings

DR. CHRISTENSEN: The first chest x-ray was done on July 17, and the last on

July 29. The diaphragm was abnormally high, especially on the right side, probably because of ascites. The lung fields were clear, and the heart was not enlarged.

On July 12 an upper gastrointestinal series showed definite varices in the lower third of the esophagus. The stomach and duodenum were normal. The upper small bowel was moderately dilated, and there was coarsening of the mucosal pattern. The transit time was slow, and the barium flocculated and segmented a good deal. The radiographic appearance was characteristic of malabsorption syndrome. A barium enema examination showed no abnormalities.

Clinical Discussion

DR. COMBES: I shall discuss only the diagnosis during the final admission. Obviously the patient was a chronic alcoholic, and portal cirrhosis was probably present. Was death the consequence of portal cirrhosis complicated by portal hypertension and hepatic coma, or did the patient have a superimposed process such as tumor or tuberculosis?

Evidence for cirrhosis includes a history of chronic alcoholism and obvious malnutrition. Physical findings consistent with chronic liver disease were spider angiomas, an enlarged and tender liver, ascites, and dilated superficial veins. Esophageal varices were seen on roentgenograms. The protein concentration of her ascitic fluid was in the range commonly found in cirrhotic patients with portal hypertension.

There were, however, several findings unusual for a patient with cirrhosis alone. I saw this patient once and was impressed by her profound emaciation. This cachexia was the major reason the surgeons were unwilling to perform a portacaval shunt even though she had bled, presumably from varices. Despite striking emaciation, her plasma protein level was 7.5 gm. per 100 ml. with serum albumin of 4 gm. per 100 ml. Even after she had been in the hospital for 12 days and had been adequately hydrated, the serum albumin level was still 3.6. She also had a fairly good hemoglobin level, and the serum bilirubin and thymol turbidity values were normal. It pays then to ask how often one sees significant cirrhosis clinically with relatively normal liver function tests? We see about two or three such cases each year. Nevertheless, the combination of ascites and normal serum albumin in a cirrhotic patient with normal liver function tests must be uncommon.

In view of the unusual features, one must consider the possibility of some other process superimposed on the cirrhosis. On the basis of hepatic vein thrombosis, could this complicating process be a Budd-Chiari syndrome? The patient had abdominal pain for several months, and her liver was large and tender. She also had ascites with a protein concentration of 2.7 gm. per 100 ml. Although it is frequently stated in the literature that the protein concentration in ascitic fluid in patients with the Budd-Chiari syndrome is high, careful review of the available data in the literature and of our own cases reveals that the protein is almost invariably less than 3 gm. per 100 ml. The composition of her ascitic fluid and the relatively normal liver function tests are quite compatible with those of Budd-Chiari syndrome. Causes of hepatic vein thrombosis and the Budd-Chiari syndrome in the adult include trauma; certain hematologic disorders, particularly polycythemia; tumor in the liver, either primary or metastatic; inflammatory processes in the liver; and idiopathic endophlebitis of the hepatic veins. Hypernephroma may extend into the renal vein and inferior vena cava and block the hepatic veins. Polycythemia was absent in our patient. There were no renal findings suggestive of hypernephroma nor were pulmonary metastases noted. Hypernephroma is a possibility, but one for which we have no definite evidence. A recent intravenous pyelogram would have been helpful. Of the various possible causes of hepatic vein thrombosis, the severe cachexia in our patient would favor neoplasm. Furthermore, the roentgenograms show a high right hemidiaphragm. I wonder whether this elevation was essentially confined to the anterior dome of the liver or not? Do we have a lateral projection?

DR. CHRISTENSEN: There was a smooth elevation of the diaphragm from front to back.

DR. COMBES: All of the findings are compatible with a tumor in the dome of the liver, either primary or metastatic, causing the Budd-Chiari syndrome.

Could this woman have had a malignant hepatoma? Hepatoma is suggested by certain clinical features such as polycythemia,

hypercalcemia, or relentless hypoglycemia.[1-4] Hypercholesterolemia and unexplained fever may also occur with hepatoma. However, relatively few patients with hepatoma exhibit these findings. Much more commonly, such patients present with progressive deterioration of hepatic function, often indistinguishable from the deterioration seen in terminal cirrhosis. Unless such deterioration occurs in a previously well-compensated cirrhotic patient, without the intervention of known precipitating factors such as recent hemorrhage or persistent alcoholic intake, it is difficult to make the diagnosis of hepatoma. In the present case none of the unusual manifestations of hepatoma was present, and the rapid deterioration of liver function was not seen; indeed, hepatic function was relatively good.

As hepatomas grow, they tend to invade the portal venous system extensively and produce severe portal hypertension. Bleeding from varices is often the first evidence of a hepatoma. Bleeding may also occur from the congested intestinal mucosa. Perhaps the abnormal small bowel pattern in our patient is the result of severe venous congestion of the small intestine secondary to portal hypertension caused by a hepatoma. In addition, hepatomas may bleed from the liver surface into the peritoneal cavity, and bloody ascites may occur. Although this is a relatively uncommon finding, such bloody fluid was present in our patient. Another way in which hepatoma may present is with nonspecific manifestations of neoplasm, that is, with a mass, weight loss, and local pain. In our patient, the combination of severe portal hypertension and manifestations of tumor such as cachexia and abdominal pain are so striking that I think that one could strongly suspect the presence of a hepatoma.

The ascitic fluid, in cases of hepatoma, resembles that seen in uncomplicated cirrhosis with a protein concentration usually less than 3 gm. per 100 ml. Only rarely do peritoneal metastases occur, and one rarely finds malignant cells in the ascitic fluid. The white cell count in the ascitic fluid of our patient was relatively high. In uncomplicated cirrhosis, ascitic fluid rarely contains more than 200 cells per cu. mm. Eleven of our patients with hepatoma had ascitic fluid analyses. In two, white blood cell counts were in excess of 1,000. The elevated white cell count in the ascitic fluid of the present patient, in the absence of any positive evidence of infection, is consistent with hepatoma.

Many of the things I have attributed to hepatoma could apply just as well to a metastatic tumor of the liver. Although it has been said that patients with cirrhosis rarely have metastases in their livers, there is now significant evidence that a cirrhotic liver will take up metastases just as well as a noncirrhotic liver, and will provide fertile soil for tumor growth.[5] In our case, we have no direct evidence for the existence of a primary tumor outside the liver. Study of the gastrointestinal tract was negative. Cervical carcinoma, although present in the past, was well treated, and there was no evidence of local recurrence. Peritoneal metastases were not demonstrated by cell block, and the ascitic fluid did not contain a high concentration of protein. Nevertheless, it is important to emphasize that ascites can occur with metastatic tumors to the liver, in the absence of peritoneal carcinomatosis, if sufficiently high portal venous pressure and decreased oncotic pressure of the plasma are produced. Thus, it is impossible to exclude a metastatic liver tumor in this patient.

Finally, in an analysis of portal hypertension which was obviously present in our patient, we should consider the possibility of portal vein thrombosis. The causes of portal vein thrombosis include omphalitis in the newborn, cirrhosis with or without hepatoma, extrahepatic tumors, especially pancreatic neoplasm, trauma, and a variety of less common conditions. Chronic portal vein thrombosis by itself will rarely cause ascites. Only when the portal hypertension of portal vein thrombosis is accompanied by a decrease in plasma albumin or renal salt and water retention (for example, after major hemorrhage) does ascites occur. In our patient, portal vein thrombosis, perhaps complicating a hepatoma, could well be a contributing factor to ascites, but I do not know of any way to prove its existence with the data we have. After considering all the features of portal hypertension in the present case, I believe the most likely diagnostic possibility is Budd-Chiari syndrome secondary to a primary or secondary liver tumor, superimposed on cirrhosis. Perhaps portal vein thrombosis is also present.

Finally, we must also consider the possibility of tuberculosis, especially tuberculous peritonitis. The patient had a strong family history; two brothers had tuberculosis, and one died with the disease. Malnourished cir-

rhotic patients are susceptible to tuberculosis. In view of her cachectic state, a negative tuberculin test is not significant.[6] A normal chest x-ray without evidence of pulmonary tuberculosis is not uncommon in patients with tuberculous peritonitis.[6] Certainly her fever and abdominal pain are consistent with tuberculous peritonitis. We expect most patients with tuberculous peritonitis to have high protein levels in their ascitic fluid, but in the setting of cirrhosis the protein may be watered down, so to speak.[7] However, bloody fluid is quite uncommon in tuberculous peritonitis, and the high proportion of neutrophils in the ascitic fluid would be unusual. Nevertheless, of the various diagnostic possibilities in this patient, tuberculosis is a treatable one. Even though no acid-fast bacilli were seen in peritoneal fluid, cultures take a long time, and the patient was too sick for laparotomy. The alternative was antituberculosis therapy, and this was given.

DR. FORDTRAN: I agree that this patient had cirrhosis. The rapid development of severe ascites in the face of an almost normal serum albumin level suggests the possibility of portal vein thrombosis, but I know of no way to prove this diagnosis with the information at hand.

The most important problem was repeated upper gastrointestinal bleeding. When cirrhotic patients have upper gastrointestinal bleeding, it is not necessarily from esophageal varices, even if varices are known to be present. Unless the varices are observed to bleed at esophagoscopy, the bleeding site remains uncertain. Varices were seen at esophagoscopy, but even this finding requires qualification because it has recently been shown that observer difference between two experienced endoscopists is high.[8] In this patient, we not only saw varices but identified a bleeding point, and I feel fairly certain that this was the major source of hemorrhage.

Management of variceal bleeding in cirrhotic patients is difficult. Our standard therapy, after the diagnosis has been established, is first to use iced saline gastric lavage. This serves several purposes: it removes blood from the gastrointestinal tract, which decreases the tendency to develop hepatic coma; it may help stop the bleeding, presumably by cooling the gastric and esophageal mucosa and thereby decreasing blood flow to these areas; and it serves as an index to the rate of bleeding. If bleeding persists after cold saline lavage, we then try intravenous Pitressin, 20 units in 200 cc. of isotonic saline solution administered in 20 minutes. This has been shown to decrease bleeding from varices, but the effect is usually temporary. Recently, it has been suggested that repeated injections of Pitressin, 20 units intravenously every two hours, can be used with more lasting benefit.[9]

If these measures are unsuccessful, we use a Blakemore tube. There are many dangers inherent in its use, including aspiration pneumonia, esophageal necrosis, and asphyxiation. Furthermore, the Blakemore tube is not a good diagnostic procedure, because variceal bleeding is not controlled by esophageal tamponade in 25 to 50 percent of cases and, on the other hand, upper gastrointestinal bleeding from other sources may stop spontaneously and give the false impression that bleeding stopped because of esophageal tamponade.[10]

For this patient, none of these methods was effective in a lasting sense. The remaining therapeutic possibility was a portacaval shunt, but this was contraindicated because of her severe ascites, persistent hyperbilirubinemia, and cachexia. Considering these clinical points, and because it has never been clearly established by double-blind clinical study that protacaval shunt is beneficial to patients with esophageal varices, I think it was wise to forego a shunt in this case.

Concerning the small intestinal abnormality shown by x-ray, it should be pointed out that transit times with barium are almost meaningless. The time it takes for barium given by mouth to reach the cecum normally may vary from 15-20 minutes to 4-6 hours. Barium is certainly not food, and it is wrong to infer that the transit time observed with barium has anything to do with the rate of transit of a meal through the small intestine. Furthermore, I think the modest dilatation was probably related to malnutrition, and I do not believe the patient had a malabsorption syndrome.

Pathologist's Report

DR. ASHWORTH: At necropsy, the patient was wasted but not jaundiced. The abdomen was distended by 6 liters of clear, serous ascitic fluid.

The liver weighed 1,530 gm. and had the characteristic gross and microscopic appear-

ance of Laennec's, or portal, cirrhosis (Figs. 1 and 2). The regenerating cirrhotic nodules measured 3 to 4 mm. in diameter. There were many larger nodules of soft, yellowish-gray tumor tissue in the liver, especially in the right lobe. In the center of the right lobe was a tumor mass 10 cm. in diameter. It was composed of similar soft, yellowish-gray tissue and represented the primary mass of a hepatocellular carcinoma, from which the satellite tumor nodules had spread. This large tumor encircled the porta hepatis (Fig. 3) and the portal vein was completely filled with tumor tissue and thrombus. Thus, as was predicted in the clinical discussion, complete portal vein obstruction had occurred.

Histologic study showed the tumor nodules to retain many features of hepatic tissue (Figs. 4 and 5), but the neoplastic cells showed nuclear hypertrophy and hyperchromatism, nucleolar enlargement, frequent mitoses, and an increase in nuclear-cytoplasmic ratio. The tumor cells formed tra-

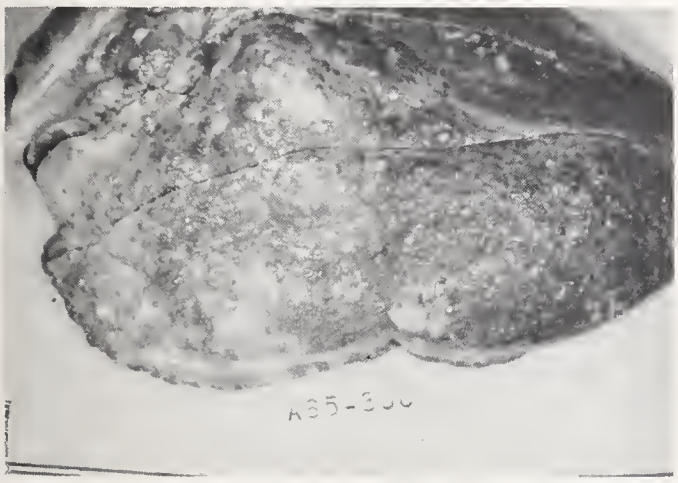

Fig. 1. Gross photograph of liver shows finely nodular appearance characteristic of portal cirrhasis. Several larger, gray nodular areas are sites of neoplastic tissue.

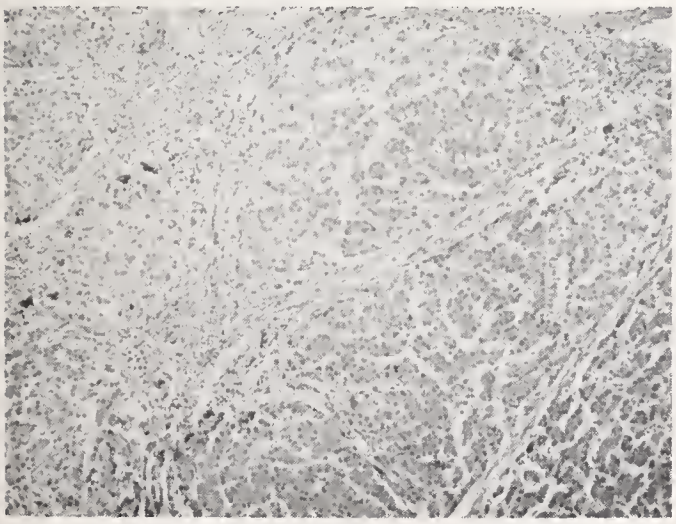

Fig. 2. Photamicragraph of liver shows discrete regenerating nodules in which the hepatic labular architecture is missing. The appearance is characteristic of portal cirrhosis. X 80.

Fig. 3. Grass phatagraph of cut section of liver. There is a large, gray mass of carcinama encircling the partal vein which is accluded by tumar thrombus (arraw).

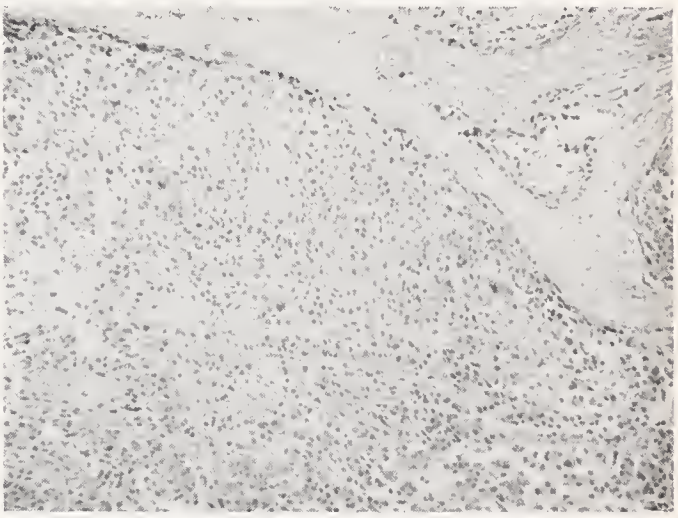

Fig. 4. Phatomicragraph of a partian of the hepatacellular carcinama. X 80.

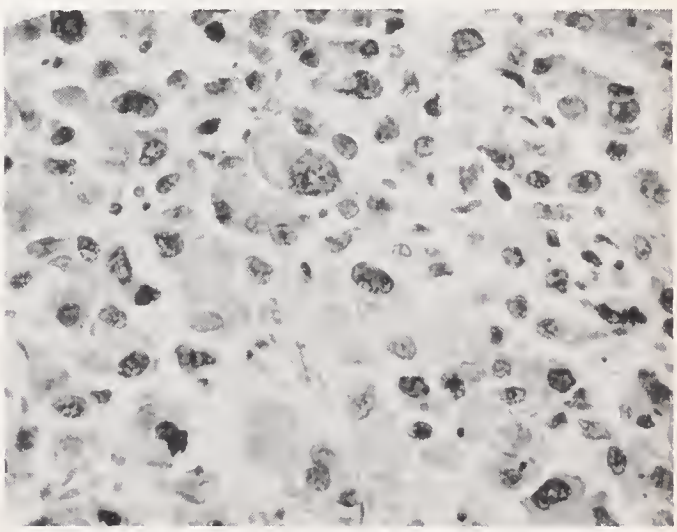

Fig. 5. Higher pawer view of the hepatic neoplasm. The tumar cells are similar ta hepatic cells except they are larger and their nuclei are pleomorphic and hypertraphied and hyperchramatic. Sinusoidal vascular channels are present between the tumar cells. X 344.

beculae, with intervening endothelium-lined sinusoids. There was no connective tissue stroma between the tumor cells, and some of the tumor cells contained bile pigment.

Metastases from a hepatocellular carcinoma are characteristically not widespread and in some fatal cases may be absent. In about 60 percent of the cases there are extrahepatic metastases at the time of autopsy. The lungs and regional lymph nodes are the most frequent sites of metastases. Rarely metastases occur in distant locations. In this case, we found metastatic tumor in the peribiliary lymph nodes and in both lungs. A single small metastatic nodule was present in the lower lobe of each lung. The pulmonary metastases were about 1 cm. in size and were apparently too small and perhaps too low in the lung field to have been visible on the available roentgenograms.

There is a significant difference in the frequency with which carcinoma of the liver develops in the various forms of cirrhosis. While reported figures are at variance, a consensus is that 30 percent of hepatic carcinomas are superimposed on postnecrotic cirrhosis, 30 percent on portal (alcoholic type) cirrhosis, 20 percent on various other forms of cirrhosis, and 20 percent are not associated with cirrhosis of any type. Viewed in another way, at autopsy, 13 percent of postnecrotic cirrhosis have superimposed carcinoma, while cancer of the liver is present in about 3 percent of cases of portal cirrhosis and in 7 percent of cases of the pigmentary cirrhosis associated with hemochromatosis.

While the clinical findings indicating probable hepatic carcinoma in a patient with cirrhosis (abdominal pain, severe and accentuated ascites, signs of vena cava obstruction, weakness, and weight loss) are well established, in most published series of hepatocellular carcinomas the clinical diagnosis is made infrequently. In one study it was reported that the diagnosis was made in only about 18 percent of all cases coming to autopsy.

The esophagus was the seat of large varices. Elongated, distended, blue veins were visible through the esophageal mucosa, even in the collapsed state at autopsy. Eroded sites in the esophageal mucosa were present over the crests of some of the bulging varices, showing the usual process that leads to hemorrhage from esophageal varices.

The spleen was not significantly enlarged, weighing only 190 gm. However, histologic study revealed definite fibrocongestive splenomegaly.

Interestingly enough, as frequently occurs in cirrhotic patients, there were peptic ulcers in the stomach. Two small ulcers, each about 3 or 4 mm. in diameter, were located on the posterior wall in the antrum. There was slight scarring around one of these lesions, and we considered both to be subacute peptic ulcers.

In spite of the fact that this patient was overtly emaciated, there was adipose tissue invasion of the heart muscle. This finding has frequently been reported in chronic alcoholics. The reasons for this are not clear. Usually the fatty invasion is more severe in the right ventricle, but in this case the left ventricle was affected.

This patient had rheumatic fever at age 13. Gross examination of the heart at autopsy showed fine vascularization of the mitral leaflets, which is a reliable indication of previous rheumatic fever. The leaflet itself was quite pliable, however, and there was no evidence of valvular stenosis or insufficiency.

Regarding the cervical carcinoma for which this patient had been treated several years previously, no residual neoplasm was found. The cervix was atrophic and fibrotic. In the uterine fundus were two large leiomyomas, and there were numerous dense, fibrous, peritoneal adhesions around the uterine fundus. Histologically, the cervix contained partly hyalinized scar tissue characteristic of late postirradiation changes, but no residual carcinoma.

The only other finding requiring comment was a minor degree of astrocytic glial swelling in the deeper layers of the cerebral cortex. Many of the astrocytes were slightly swollen, their nuclei were vesicular, and their nuclear membranes were slightly wrinkled. This swelling of the astrocytic nuclei is a well known feature of hepatic coma and hepatic insufficiency.

The main pathologic diagnoses were:

1. Hepatocellular carcinoma with metastases to regional lymph nodes and lungs and obstruction of portal vein.
2. Portal (Laennec type) cirrhosis.
3. Esophageal varices with erosion.
4. Slight fibrocongestive splenomegaly.
5. Ascites.
6. Astrocytic nuclear swelling (hepatic encephalopathy).
7. Two subacute peptic ulcers of stomach.

8. Fibrosis of cervix uteri (no residual carcinoma).

9. Healed rheumatic mitral valvulitis without valvular deformity.

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Routine Typhoid Immunization Not Recommended

Surgeon General William H. Stewart of the Public Health Service says that routine typhoid immunization is *not* recommended in the United States. His statement was based on a report of the Public Health Service Advisory Committee on Immunization Practices, published by the Communicable Disease Center in Atlanta. The Committee, composed mostly of experts from without the Service, recommended selective immunization in the following situations: (1) Intimate exposure to a known typhoid carrier as would occur with continued household contact. (2) Community or institutional outbreaks of typhoid fever. (3) Foreign travel to areas where typhoid fever is endemic.

The Advisory Committee stated, "Although typhoid vaccine has been suggested for individuals attending summer camps or those in areas where flooding has occurred, there are no data to support the continuation of these practices."

The incidence of typhoid fever in the United States has declined steadily for many years, less than 500 cases are reported annually, and a continuing downward trend can be expected. Cases are sporadic and are primarily related to contact with carriers rather than to common source exposure.

Referring to vaccines for paratyphoid A and B, which are typhoid-like fevers, the Advisory Committee stated, "The effectiveness of paratyphoid A vaccine has never been established, and recent field trials have shown that available paratyphoid B vaccines were ineffective. In view of these data and recognizing that paratyphoid A and B antigens when combined with typhoid vaccine may increase the occurrence of vaccine reactions, use of paratyphoid A and B vaccines is *not* recommended."

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NEWS

Texas Physicians To Gather Oct. 1 For Annual Public Relations Conference

The 1966 Texas Medical Association Public Relations Conference, ordinarily held in September, is set for Oct. 1 at TMA Headquarters Building, 1801 N. Lamar, Austin. This meeting date represents a rescheduling of the Conference, originally planned for Sept. 17. Various schedule conflicts required the change to October this year.

MR. POWERS

Five main speakers and two panel discussions will highlight the 1966 Public Relations Conference. Speakers will include Robert B. Wolcott, Jr., president of the Public Relations Society of America (PRSA); John J. Powers, Jr., president and chief executive officer of Chas. Pfizer & Co.; John J. Henry, editor of *Babson's Washington Forecast Letter;* Dr. J. A. Sabatier, Jr., of Baton Rouge, La., member of the American Medical Association Committee on Quackery; and Roy Pfautch, president of Civic Service, Inc., and member of the American Medical Political Action Committee.

Panel discussions will include "New Federal and State Medical Programs and the Physician," Saturday morning, and "Reaching the Public Through Positive Public Relations," Saturday afternoon. The former will contain discussions of Medicare; Office of Economic Opportunity programs; Heart Disease, Cancer, and Stroke in Texas; and Kerr-Mills in Texas. The latter panel will cover discussions on developing news media relations, organizing an effective speakers bureau, and planning a community relations program of physician participation.

An Orientation Program for new and transfer members will be held at the same time as the Conference, also at the Headquarters Building. Both the Conference and the Orientation Program will run the full day on Saturday.

Members of the Woman's Auxiliary will hold a board meeting on Thursday, Sept. 29, and a school of instruction on Friday, Sept. 30, at the Headquarters Building. Forty TMA committees, councils, and boards will meet at various times and places throughout the Conference weekend. The Executive Board will meet at 9:30 a.m. Sunday, Oct. 2, to consider and act on reports from these meetings.

TMA will furnish bus transportation on Saturday morning to the Conference from the downtown hotels. Return transportation will be provided at the conclusion of the conference that evening.

On Saturday morning, physicians are invited to a TEXPAC breakfast meeting at 7:15 a.m. in the Crystal Ballroom of the Driskill Hotel. Dr. Milford O. Rouse of Dallas, AMA President-elect, will speak.

Physicians, Auxiliary members, and guests will be served a luncheon in the basement of the Headquarters Building at the conclusion of the morning session on Saturday.

During the afternoon session, a special presentation will honor Texas doctors who have served in civilian capacities in Vietnam.

Activities on Saturday evening will include a 5:30 hospitality hour, sponsored by Charles O. Finley & Company at the Commodore Perry Hotel, and the University of Texas-Indiana University football game at Memorial Stadium. Transportation to the stadium from the Commodore Perry Hotel will be furnished by Charles O. Finley & Company.

MR. WOLCOTT

Austin hotels have reserved rooms for members of TMA who will attend the Conference. Those needing hotel accommodations may write directly to the facility of their choice to make arrangements.

Football game tickets are $5 each and may be ordered from TMA. The game will be at 7:30 p.m.

Speaking before the Conference on "Public Relations Is Human Relations," will be Robert B. Wolcott, Jr. He is president of Robert B. Wolcott Associates, a national public relations counseling firm. He established this public relations

business in 1950. Before that time he had held various executive positions with Time, Inc. He has been active in PRSA for 15 years. Before becoming president he had served as vice-president, as a member of the Board of Directors, and as a delegate to the National Assembly. He has been twice honored by the Society with its Presidential Citation for his leadership in founding the PRSA Counselors Section.

John J. Powers, Jr., will speak to the Conference on the subject "Breakthroughs in the Life Sciences." Mr. Powers has been associated with Pfizer & Co. for 25 years. He has served the firm as vice-chairman and as president. He also served as president and chairman of the company's foreign activities organized under the name of Pfizer International. He has worked closely with the medicinal products research of both the parent company and its international subsidiary. Active in affairs outside his company, Mr. Powers is a member of the board of the Pharmaceutical Manufacturers Association and of the board of the association's foundation. In addition, he is a member of the board of trustees of the US Council of the International Chamber of Commerce.

Auxiliary To Hold School, Board Meeting, Sept. 29-30

The Woman's Auxiliary to the Texas Medical Association will hold an Executive Board meeting at TMA Headquarters Building on Thursday, Sept. 29. A school of instruction, "The School of the Three E's (Excellence, Enthusiasm, Efficiency)," will be held on Friday, Sept. 30, also at the Headquarters Building.

The school of instruction will emphasize the formula for auxiliary success in 1966-67: $E = E \times E^2$. (Excellence equals enthusiasm times efficiency squared.)

Two main speakers will highlight the board meeting and school of instruction programs: Col. Vincent I. Hack of Brooke Army Medical Center in San Antonio on "The Psychology of Color" and Paul Boesch of Houston, author and professional wrestler, on "The Womanly Art of Self-Defense." Dr. Robert Moreton of Houston, chairman of the TMA advisory committee to the Woman's Auxiliary, will speak to the Executive Board members. Dr. James D. Murphy of Fort Worth, President of TMA, and C. Lincoln Williston, executive secretary of TMA, will also address the auxiliary members.

Other features of the two events will include program ideas, new films, committee workshops, and group discussions.

TWU To Offer Workshops In Rehabilitation Nursing

An opportunity that is attractive to those physicians responsible for nursing services in institutions caring for the chronically ill and disabled is being announced by Texas Woman's University. It is an opportunity equally attractive to the professional nurse who is responsibile for training and service in this field.

"All who have studied the needs of nursing agree that not only are more registered nurses needed, but for those now in the profession, more opportunities are needed for continued education," says Dr. W. D. Seybold of Houston, chairman of the TMA Committee on Nursing.

Dr. Seybold continued, "A workshop conducted by one of our outstanding state universities and sponsored by the US Public Health Service is a new step toward supplying this need. It should be welcomed by all Texas physicians and nurses."

The Houston Clinical Center of the Texas Woman's University College of Nursing will hold a series of rehabilitation nursing workshops for registered nurses beginning in September.

The four-semester-hour credit course condensed to a four-week workshop period will be offered six times between September, 1966, and August, 1967. Workshop dates are Sept. 12-Oct. 7, 1966; Oct. 24-Nov. 18, 1966; Jan. 9-Feb. 3, 1967; March 6-31, 1967; May 1-26, 1967; and July 10-Aug. 4, 1967.

The purpose of the course is to help registered nurses to expand their knowledge and to increase their skills in rehabilitation nursing techniques and to plan, implement and evaluate the concept of total rehabilitation in actual nursing care and in retraining the disabled person. There will be opportunity to give individualized and personalized nursing care to a selected patient.

Topics to be undertaken include philosophy of rehabilitation, nurse-patient relationships, activities of daily living, problems of inactivity, orthotic devices, ambulation, body mechanics, discharge and follow-up care, team care of the rehabilitation patient, and limited rehabilitation goals due to severe physiological deconditioning.

The program of instruction will be arranged to provide lectures, demonstrations, discussions, laboratory practice, clinical practice, and conferences in the basic elements of preventive and restorative nursing measures applicable to the nursing care of the patient with chronic illness or disability.

Clinical resources will include the Texas Institute for Rehabilitation and Research, and the Houston Veterans Administration Hospital. The workshop program is sponsored by the United States Public Health Service which also provides stipends of $12 per day or a total of $312 for each participant who must change residence in order to attend.

Priority registration will be given to applications from teachers in basic programs in nursing, from nurses in in-service education in hospitals and health agencies; and from head nurses and supervisors in hospitals and public health agencies.

For further information and for registration write to Miss Lucie C. M. Schultz, Program Director, Rehabilitation Nursing Workshop, Texas Woman's University College of Nursing, 1130 M. D. Anderson Blvd., Houston 77025.

New Society Formed

Investigative and clinical applications of low-temperature techniques to the eye will be promoted by the newly-formed Society for Cryo-ophthalmology.

The society hopes to hold scientific meetings immediately prior to the annual sessions of the American Academy of Ophthalmology and Otolaryngology meeting this year Oct. 16-21 in Chicago. It invites physicians interested in the investigative aspects of cryo-ophthalmology, the preservation of ocular tissue, therapeutic applications of cryogenics to various ocular diseases, and cryosurgical techniques.

Inquiries and applications should be addressed to Dr. John G. Bellows, 30 N. Michigan Blvd., Chicago 60602.

Ambulance attendants aided by police officers prepare to place wounded patient in waiting ambulance, following shooting incident from the University of Texas Tower on Aug. 1. Most victims were hit in the first 20 minutes of firing.

Efficient Care of Sniper Victims Shows Worth of Disaster Plans

Shortly after Charles Joseph Whitman began his devastating barrage of gunfire from the Tower of the University of Texas on Aug. 1, Austin medical personnel had some special problems to face.

Of the 49 persons wounded or killed by the sharpshooter, 39 were taken to the emergency facilities of Brackenridge Hospital, the city-owned hospital, in an hour and a half time period. The afternoon and evening were a nightmare undreamed of that morning. But when the worst was over, hospital and other medical officials expressed satisfaction with the way the tragedy had been handled from the medical standpoint.

A Lesson for Medical Personnel

"Have a disaster plan and rehearse it," was the advice Ben Tobias, administrator of Brackenridge, had for hospitals and medical personnel. "Having a plan and rehearsing it helped us in being able to carry through with the job we had to do," he said. The Brackenridge plan had been rehearsed on paper on March 9 of this year and in actual practice with 36 nurses labeled as casualties on March 16.

"The disaster plan was carried out well. We were fortunate to have had our two rehearsals," Mr. Tobias said.

Brackenridge has a disaster plan which calls for each department to perform a certain function. The department then is responsible for assigning and instructing personnel as to their respective duties.

How the Plan Worked

The Brackenridge disaster plan was put into effect about 12:20 p.m. on Monday, Aug. 1. A control center was set up in the emergency area to be staffed with doctors and nurses throughout the necessary time period. The switchboard operators notified all departments that the disaster plan was in effect. Lines were cleared to the emergency room.

Personnel from the admitting office came to help at the information desk. Doctors on the medical staff both within and without the hospital were contacted and asked to come to the emergency room.

The hematology laboratory began matching blood. During the first hour, blood for 40 patients was matched. Seventy units were used. Six persons were working in the hematology lab whereas normally two workers are sufficient, and eight persons served as runners and messengers. (Normally none are needed.)

Nurses returned from lunches and off-duty and came from other hospitals. Student nurses also came.

The outpatient department was cleared and made ready to help receive emergency room overflow. Two emergency cases besides the wounded from the University area were brought in during the disaster period. Other patients in the emergency area volunteered to leave in order to make room for the incoming patients.

Because of the large influx of patients, it was necessary to open a 21-bed wing of the hospital which had been closed earlier in the year because of a nurse shortage. Ten of these beds were used. Because of the larger number of nurses who

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Photographs were made by Texas Student Publications photographers Frank Armstrong and Richard Kidd.

came on duty Aug. 1, this wing was adequately staffed and accommodated patients whose doctors approved their being moved from the main building. A large number of patients were either transferred or discharged.

The need for linens required the laundry to be opened, and it remained open until 9 p.m.

The hospital purchasing department saw that supplies were brought whenever and wherever they were needed. There was no shortage of supplies at any time.

The dietary department prepared snacks for the personnel and families, members of the press, radio, and television. In addition the cafeteria was kept open around the clock two days for the convenience of families, personnel, and friends. Local vendors furnished needed supplies immediately.

Dr. Bob Pape, director of medical education, served as a resource for questions from the press from all over the country. Medical records personnel also assisted in handling the press. The social service department opened an office and speeded the flow of information on victim identification to news media.

Two justices of the peace were brought in to take care of the persons who were dead on arrival at the hospital.

Mr. Tobias said that it would be hard to estimate the number of personnel involved before some time had elapsed, but said several hundred participated. "Every patient had a doctor and nurses most of the time. Our whole surgical facilities were in operation all afternoon. Twenty-one people out of twenty-seven went to surgery or the cast room."

Problems Encountered

"The main problem was getting correct identifications of the patients. There was no identification at all available in some cases. This was due to the rapid removal of persons from one place to another."

Mr. Tobias said that in some cases obtaining correct spelling of names and correct addresses were difficult. Members of the press helped in confirming names.

Another problem was the flooding of the switchboard with calls from local, national, and foreign sources. The switchboard operators, (twice as many on duty as normally) carefully put through pertinent calls and rerouted others. Policemen with walkie-talkies were in the emergency area. These men kept hospital personnel informed of patient-bearing ambulances heading for the hospital.

A third problem involved crowds of people who clustered around the emergency area of the hospital. The housekeeping and maintenance departments helped control the flow of crowds in order to facilitate unhindered transportation of patients into the hospital.

Favorable Factors

But all occurrences were not hindrances to successful hospital operation.

"From our standpoint," said Mr. Tobias, "if the tragedy had to happen, it was lucky that it happened at a time when more personnel were immediately available. The time was convenient from a staffing standpoint."

Mr. Tobias speculated that at another time of day the sniper might not have wounded so many people; but, on the other hand, the hospital would not have had the staff to take care of the rapid influx of patients.

Also, Mr. Tobias found cooperation to be at its best. "We couldn't have asked for better cooperation all around; everything was handled wonderfully."

Surgeons Efficient

"All surgeons handled themselves very well, and all emergency room personnel did a very good job. We were pushed but working efficiently."

Dr. F. Murphy Nelson, former chief of surgery at Austin's Brackenridge Hospital, had nothing but praise to offer concerning the performance of the hospital's staff during the hectic hours during and following the Aug. 1 tragic shooting incident at the University of Texas.

More than 50 doctors reported to the hospital immediately upon hearing the news of the emergency on the radio. Some of those in the University area had some difficulty getting to the hospital because of traffic congestion. Dr. Nelson was not at the hospital when the first

Police carry deceased woman, victim of sniper, to ambulance. Presence of an ever-growing crowd plus absence of a stretcher made the job difficult.

few cases arrived. He himself had no trouble in getting there quickly, he reported. All the doctors who reported to Brackenridge, it turned out, were not needed.

Doctors were in surgery from 2 p.m. until 10 p.m. Aug. 1; however, most of the critical cases were cared for by 6 p.m.

Dr. Nelson said that the regular disaster plan worked very well. He emphasized that the triage method of selecting patients had an especially effective part in facilitating necessary patient care.

Blood Donors Numerous

Viewing the Aug. 1 tragedy from the standpoint of the Travis County Medical Society Blood Bank, John Kemp, executive director of the Travis County Medical Society, was "extremely happy with the way things worked out."

"We found out how vital the full support of the press is," Mr. Kemp said. "It is very important to maintain good relations with the news media. The operators at the switchboards of the local television and radio stations knew who I was, and my calls got put through ahead of long distance calls. Our requests for blood got on the air immediately."

Mr. Kemp had five fundamental suggestions to offer blood banks facing a similar disaster situation:

1. Be known by and have good relations with local representatives of news media.

2. Show appreciation to those who have helped immediately, or as soon as possible, after the disaster is all over.

3. Maintain constant contact with the hospitals involved and with radio and television stations.

4. Advise key personnel that in case of a disaster they should report to the blood bank immediately.

5. Insure that extra supplies are always on hand. "At 11:55 we were unaware that we were already faced with a disaster," Mr. Kemp said.

Mr. Kemp said he became aware of what was happening about 12:15 on Monday, Aug. 1. He called Dr. Douglas Terry, medical director of the Travis County Medical Society Blood Bank; and they estimated the blood they would need, considering what was on hand.

Mr. Kemp immediately called the local television stations and asked that people who knew their blood type and were in the northeast part of Austin and could get to the blood bank without risking danger in the university area to come to the blood bank to give blood. The hospital departments were busy and could not yet make an accurate estimate, so at this time the amount and types of blood needed were not actually known. Also, knowing what blood would be needed was impossible because the sniper was still at work.

Public response was heartwarming. "If Austin is any indication, we don't have to worry about people supplying needed blood in time of disaster," Mr. Kemp said. "In five minutes we had a traffic jam at the blood bank. All policemen were involved in some way with the incidents at the University: there was no one to direct traffic. Two university students got out in the street and temporarily directed traffic until police officers arrived. We had lines of people all around the building."

Mr. Kemp estimated that more than 500 persons came to give blood. The actual number of pints drawn was 120. When the need seemed to be supplied, lists were made of willing donors who could be called to return if their blood was needed. All six drawing tables at the blood bank were in use from 12:25 until 8 p.m. (On a normal day 15 to 30 pints are drawn.) Calls came from San Antonio and Dallas blood banks offering to send any amount of blood that was needed.

The blood bank's own staff members who were not on duty came to work as soon as the news of the shooting reached them. Several doctors reported to the Blood Bank immediately, and one or two remained at the bank all afternoon. There was no panic or confusion. Everyone just worked hard to get the job done.

People from all walks of life responded — students, city council members, young, old, wealthy, poor. News media and police cooperated fully. "Everybody pitched in to help," Mr. Kemp said.

Future disasters will be handled with even greater efficiency, he predicted, because the blood bank will have emergency radio system facilities in addition to the present direct telephone lines to the hospitals.

Crowds of students watch as ambulance attendants and policemen wheel wounded patient near the Tower on a stretcher. This young man was one of the less seriously injured persons wounded by Charles J. Whitman in the hour-and-a-half tragedy.

AHA To Convene in Chicago For 68th Annual Meeting

"Medicare—60 Days Later" will be the predominant theme of the 68th Annual Meeting of the American Hospital Association (AHA) scheduled in Chicago Aug. 29-Sept. 1. Twenty thousand hospital, medical, and health care personnel will attend.

The program features prominent officials in the federal government, as well as leaders in the hospital and medical fields. The latest in hospital equipment will be displayed in the McCormick Place exhibition hall.

Robert M. Ball, Commissioner of Social Security, will deliver a Medicare progress report to the August 31 General Assembly session at McCormick Place.

A two-part Medicare Clinic is scheduled for the morning and afternoon General Assembly sessions on September 1. Participants will include Social Security Commissioner Ball; Fred H. Steininger, director, Bureau of Family Services, US Welfare Administration; Walter J. McNerney, president, Blue Cross Association; Clarence E. Wonnacott, past president of AHA; Dr. Leo J. Gehrig, deputy Surgeon General, US Public Health Service; Dr. Madison B. Brown and Kenneth Williamson, both associate directors of AHA.

Dr. Charles L. Hudson, President of the American Medical Association, will address the AHA General Assembly session on August 30. His subject will be "Health Care in 1966."

Seventy instructional sessions also are scheduled during the four-day convention. Subjects to be covered include "The Manpower Crisis in Health Services"; "How To Reach Recruits in the Health Field"; "The Heart Disease, Cancer and Stroke Program"; and "Hospitals and Home Care."

Texas General Practitioners To Hold Houston Assembly

The Texas Academy of General Practice will hold its annual Scientific Assembly Sept. 12-14 at the Rice Hotel in Houston. A pre-assembly seminar, acceptable for five hours credit by the American Academy of General Practice, will take place on Sunday, Sept. 11.

The seminar will be conducted in cooperation with Eli Lilly and Company. Topics to be studied include pitfalls in the practice of internal medicine, pitfalls in handling psychiatric patients, and pitfalls in surgery as seen in general practice.

The Assembly program will include a series of scientific lectures. Subjects to be covered include refractory heart failure, physical medicine for early arthritis, fungal infections, adolescent gynecology, skeletal problems in pre-adolescence, stroke rehabilitation, acute pancreatitis, febrile seizure, gynecological emergencies, and closed head injuries in children.

Registration fee, covering both the seminar and the Assembly, is $10. Registrations will be accepted at the beginning of the sessions.

Animal Care Panel To Meet

The 17th Annual Meeting of the Animal Care Panel will be held Sept. 26-30 at the Edgewater Beach Hotel, Chicago.

Those wishing further information should write to Joseph J. Garvey, Executive Secretary, 4 E. Clinton St., Joliet, Ill. 60434.

AAPS Sets March Deadline For State Contest Entries

March 15, 1967, is the deadline for submitting state entries in the 1967 Association of American Physicians and Surgeons Essay Contest for high school students. Three winners in county AAPS contests must be determined by that time.

County medical societies and auxiliaries sponsoring contests should begin committee selection and organization in July, and schools should be contacted in September.

As previously, the choice of essay topics will be "The Advantages of Private Medical Care" and "The Advantages of the Free Enterprise System." The state contest is usually sponsored by TMA and Texas members of the AAPS. The first, second, and third place winners at the state level will be entered in the national AAPS essay contest by April 1, 1967.

Dr. Herbert C. Allen, Jr., of Houston is state contest chairman for Texas.

Texas Heart Association Plans Houston Meeting

The Texas Heart Association will hold its 31st Annual Meeting and Scientific Sessions in Houston Sept. 16-18.

The meeting, which will be headquartered at the Shamrock Hilton Hotel, will feature speeches by Dr. Charles A. Berry, director of medical research and operations at the Manned Spacecraft Center in Houston; W. B. Mansfield, Supervisory Development Division chief of Texas A&M University's Engineering Extension Service; and Dr. Lewis E. January, president-elect of the American Heart Association.

Also, members will be able to view a live open-heart operation on closed-circuit television, take part in one of eight discussion groups, and visit technical and scientific exhibits. Meeting activities will take place on Saturday and Sunday, Sept. 17 and 18.

There will be seven scientific sessions on Friday and Saturday, Sept. 16 and 17, as well as three panel discussions. Featured scientific speakers are Dr. Harriet P. Dustan, Cleveland, Ohio; Dr. Lewis E. January, Iowa City, Iowa; Dr. Thomas Killip, III, New York City; Dr. John H. Laragh, New York City; Dr. Clark Millikan, Rochester, Minn.; Dr. Philip Samet, Miami Beach, Fla.; and Dr. Jeremiah Stamler, Chicago.

Subjects to be covered in scientific sessions include renovascular hypertension, the course of mitral stenosis after closed mitral commissurotomy, comparison of coronary care unit and regular hospital care in the treatment of myocardial infarction, aldosterone and hypertension, changing concepts in the treatment of occlusive cerebrovascular disease, clinicophysiologic correlations in cardiac pacing, and the dietary management of arteriosclerosis.

Panel discussion subjects include stroke: diagnosis, management, and prevention; cardiac pacemaker therapy; and renal-adrenal interrelationships in hypertension.

Discussion groups will include consideration of relationships and responsibilities, accounting, cardiopulmonary resuscitation programs, heart career day programs, heart club programs, heart of the young executive programs, mended heart programs, and fund raising.

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State Welfare Department Assists Texans Not Covered by Medicare

ALTHOUGH IT has not been widely publicized, many Texas residents are not currently eligible for medical assistance for services listed under portions of federal Medicare legislation.

With minor exceptions, persons who were recipients of aid under the Old Age Assistance Acts, originally passed in 1936 and administered by the Texas State Department of Public Welfare, are still under the medical aid provisions of Public Law 86-778 (Kerr-Mills).

The eligibility of these individuals for complete Medicare coverage will be decided at the discretion of the Texas Legislature at the next legislative session. In the meantime, the Texas State Department of Public Welfare has made some provisions for the medical care of these persons.

Medical Care Background

Through a series of laws, the Texas State Department of Public Welfare has for many years been able to assist needy persons in Texas.

Legal provisions began in 1931 with the creation of the Child Welfare Division under the Board of Control, which extended through the Reconstruction Finance Corporation days and the Texas Relief Commission. After the enactment of the Federal Social Security Act of 1935, the 44th Texas Legislature passed House Bill 26 creating the first Old Age Assistance Commission on Feb. 14, 1936. Payments were authorized to begin July 1, 1936. The Texas State Department of Public Welfare was created by Senate Bill 36, in regular session, Jan. 10, 1939.

Since 1939 there have been many changes by the Legislature in the basic areas of aid and scope of coverage. The most notable to the physician has been the ramification of the Old Age Assistance Acts, the Aid to the Permanently and Totally Disabled, Aid to the Blind, and Aid to the Families with Dependent Children.

These are federally-subsidized state programs, created and defined by statute administered by the State Department of Public Welfare, and designed to give monthly cash assistance and services *according to need* to aged persons, to the blind, to the permanently and totally disabled, and to children deprived of parental support. Legislative enactment determines the administrative structure of the State Department of Public Welfare, the conditions under which a person may be aided, the basis on which money is drawn into the treasury through taxation, and the redistribution to eligible persons.

Following the enactment of Public Law 86-778 on Sept. 13, 1960 (Kerr-Mills), the 57th Legislature on Aug. 28, 1961, authorized medical assistance to aged recipients of assistance. The first payments were made under this law on Jan. 1, 1962.

The State of Texas, through the cooperative efforts of the State Department of Public Welfare, Group Hospital Services, Inc., the Texas Medical Association, and Texas physicians developed one of the most effective systems of medical care for the needy aged.

Title VI of Public Law 88-352, The Civil Rights Act of 1964, and Public Law 89-97 Social Security Amendments of 1965, are recent additions to the complex Legislative enactments defining operational policies of federally subsidized state assistance programs.

Current Welfare Status

The Texas State Department of Public Welfare is not operating under Title XIX. The decision for continuance of the present program or implementation of Title XIX will be at the discretion of the next session of the Legislature.

With the exception of a portion of Part A of Medicare, Hospitalization Coverage, medical assistance for Old Age recipients, is still based upon state standards of categorically eligible needy, and not the terminology of medically indigent. The Old Age Assistance recipient must be 65 years old, meet the need requirement, the requirements for citizenship and residency, and the other standards of eligibility as prescribed in the state law.

More than 900,000 individuals over 65 in Texas are covered through the Social Security Administration on Part A of Medicare for Hospitalization Benefits. The Old Age Assistance recipients within the state are no exception. The Texas Department of Public Welfare has entered into a contractual agreement with Group Hospital Services, Inc., of Dallas to pay the deductibles for Part A of Medicare for 230,000 certified Old Age recipients. More than 99 percent of the state's Old Age recipients signed up for Part B of Medicare for physicians benefits.

These certified recipients were granted money to pay the premiums through funds administered by the Department of Public Welfare for July and August, 1966. Due to federal restrictive policies, the Board and the Commissioner of Public Welfare would not recommend Texas "buy in" for its Old Age recipient group. These policies would have placed the Texas Legislature in a position of using "open end" appropriations for Title XIX implementation or lose federal subsidy of existing programs. The federal restrictions have been removed, and the Old Age recipients will have their premiums on supplementary medical insurance paid directly to the

insurance intermediary starting Sept. 1, 1966. Deductibles for Part B, physician services, are to be paid by Group Hospital Services, Inc., on a contractual basis just as is Part A.

The Vendor Medical Payment for Old Age recipients in nursing homes has not been changed.

Four hundred and twenty-two recipients of Aid to the Blind will be covered under the same contractual arrangement, because they are over age 65, have met the other requirements of need, statutory blindness, and other eligibility requirements. These Old Age recipients and Aid to the Blind recipients will have two sets of identification cards: one set from the Social Security Administration, and one from Group Hospital Services, Inc., of Dallas. No further documentation or checking for the amount of deductibles should be necessary for processing.

Other Welfare Aid

For cases involving dependent children, the State Department of Public Welfare has purchased individual insurance policies for certified Aid to Families with Dependent Children. These policies are on a straight insured basis through Group Hospital Services, Inc., of Dallas, and are effective July 1, 1966, through August 1967. These policies include the children, incapacitated second parent, or certified caretaker, or both, with whom the child lives. There are 22,109 cases involving Aid to Families with Dependent Children in Texas, representing 74,506 children. These recipients will have Blue Cross cards for identification.

Blue Cross will furnish each physician with the definition of identifiable services. Basically, this includes a $10-a-day hospital bed, board, and nursing services, with seven-day maximum coverage and allowed 90 day readmission. A $200 surgery schedule and a $3-$7 physician in-service policy with full ancillary services except blood and plasma are included. No provision has been made for obstetrical cases. In addition to the hospitalization policy, provisions are made for a $5 physician fee for five outpatient, home, or office visits; four visits occurring before July 1, 1967, and one visit during July and Aug. 1967. Subject to limitations defined within the policy, 50 percent of a physician's reasonable charges for x-ray examinations in the physician's office will be assumed by Group Hospital Services, Inc.

The State Department of Public Welfare has purchased individual hospitalization policies from Group Hospital Services, Inc., of Dallas for 10,425 recipients of Aid to the Permanently and Totally Disabled. These individuals have been certified for aid by meeting the definition of disability as defined by state statutes, need, and other necessary standards of eligibility.

Provisions for Disabled

These cases will have Blue Cross identification cards. This, too, is basically a $10-a-day hospital bed, board, nursing service, with ten-day maximum, and allowed readmission after 90 days. Again, also included are a $200 surgery schedule, and a $3-$7 physician in-service policy with full ancillary services except blood and plasma. Obstetrical care of any nature is excluded. These policies do not carry the outpatient, home, or office visit clauses, nor do they allow for payment on x-rays done in the physician's office. Welfare recipients may continue to have some allowance in their assistance check for limited physicians' services and medications.

The Department of Public Welfare has attempted, through Group Hospital Services, to defray some of the cost of medical care for the indigent of this state. The compensation, while not adequate, is limited by the moneys appropriated and is far more than has been possible in the past.

Physician Participation

Physician participation for Old Age Assistance recipients has been made as simple as possible. The insurance carrier and the carrier for the deductibles have suitable exchange of information to clear the claims without further verification other than the identification cards.

Billing may be done on the assignment plan or the itemized bill plan as shown on Medicare instructions. Few Old Age Assistance recipients will be able to pay cash for their services. If the assignment plan is not acceptable, physicians may consider the use of the personal note plan as described in the June 20, 1966 issue of *The AMA News*.

The Aid to the Permanently and Totally Disabled, and the Aid to Families with Dependent Children will be verified by the Public Assistance worker as a certified case. This is much the same process that has been used in the past for Old Age Assistance recipients.

There are State Public Welfare offices in most Texas counties with trained personnel to assist. A call to any of the offices would expedite the solution of problems concerning welfare recipients. Without the cooperation of the treating physician and the Texas Medical Association, past efforts for medical assistance to the needy would have failed. Without the physician's assistance, the present program will fail. Undoubtedly, the success or failure of the existing program will influence future additions and deletions of regulations.

Physician-Welfare Relations

A good working relationship between the Texas Medical Association and the Texas State Department of Public Welfare is being fortified by the appointment of a Medical Advisory Board for the Department of Public Welfare. Through joint efforts the principles for "Standards of Health Care Programs" adopted in 1965 by the House of Delegates of the American Medical Association should be carried out to the extent possible under law.

Physicians may not wish to participate under the Social Security Amendment of 1965, and this is their decision. The Texas State Department of Public Welfare hopes, however, for the best medical care possible for the certified needy of this state. If present policies interfere with or impair the free and complete exercise of the physician's judgment and skill, or make it impossible for him to give the type of medical care needed, he is asked to advise the indigent patient in advance that he does not wish to participate.

The Department of Public Welfare is fully cognizant of the extra paper work, inconveniences, and delays. We do, however, look forward to doctors' cooperation in keeping service to the needy in its proper place—at the top.

—Philip A. Gates, M.D.,
Assistant Medical Director,
Medical Services Division,
State Department of Public
Welfare

Tolazamide Proves Valuable As Oral Hypoglycemic Agent

Tolazamide [1-(hexahydro-1-azepinyl)-3-p-tolylsulfonylurea]; Tolinase, The Upjohn Co. (See Fig. 1) is a new member of the sulfonylurea class of oral hypoglycemics.

Holcomb[1] has reviewed the literature pertaining to diabetes mellitus, insulin therapy, and oral hypoglycemics; and he states that from a practical standpoint the sulfonylureas are currently the most important oral hypoglycemics. Holcomb also concludes that the sulfonylureas apparently do not directly affect the metabolism of carbohydrates; instead, they appear to act through a mechanism stimulating the cells of the pancreas to secrete insulin. The exact mechanism for this stimulation remains to be elucidated.

The medicinal chemists, Wright and Willette,[2] at the Upjohn Co. Research Laboratories, synthesized a series of N^4-arylsulfonylsemicarbazides; and a number of the compounds showed appreciable antidiabetic activity.

The Wright and Willette series included tolazamide which has been compared with other sulfonylurea hypoglycemics on the basis of animal and human evaluations. The clinical potencies of some of the more common sulfonylureas, using tolbutamide (Orinase, The Upjohn Co.), as base 1, are: carbutamide, 1 to 3 times as potent; chlorpropamide (Diabinese, Pfizer Laboratories), 4 to 7 times as potent; metahexamide, 10 times as potent; and tolazamide, 5 to 15 times as potent.[1]

The general structure-activity relationships for the sulfonylureas have been summarized by Mahler.[3] Referring to Fig. 2, the benzene ring should have at least one substituent, preferably in the para position. Methyl, amino, acetyl, methylthio, and chloro substitution has produced active compounds. When the R is an aliphatic moiety, the methyl congener is inactive. Some activity is associated with the ethyl analogue and maximum activity is exhibited by compounds wherein the R group possesses from three to six carbon atoms. With further increasing carbon content (length) activity decreases. Apparently, lipophilic/hydrophilic balance associated with this moiety is an important factor.

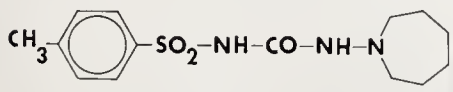

Fig. 1.

It is noteworthy that in the case of tolazamide, instead of an alkyl moiety substituted into the terminal urea-nitrogen, there is a second nitrogen atom, then the alkyl moiety. Accordingly, tolazamide is a semicarbazide derivative. Such semicarbazide derivatives generally appear to possess greater "acute potency" than related sulfonylureas.

"Acute potency" is estimated by comparing the dose of a compound needed to produce a hypoglycemic effect at four hours compared to a 1 gm. dose of tolbutamide in healthy humans.[4] For tolazamide in particular the acute potency was seven times that of tolbutamide and chlorpropamide. In addition, in normal subjects the half-life of tolazamide was 7.2 hours, as compared with 5.7 for tolbutamide and 33 for chlorpropamide. It may be noted that the p-chloro substituent confers long half-lives upon the sulfonylureas. Chlorpropamide possesses a p-chloro instead of a p-methyl substituent; and, as indicated, chlorpropamide possesses a much greater half-life.

The same pattern is revealed in the sulfonylsemicarbazides: the replacement of the p-methyl substituent of tolazamide with p-chloro yields the compound U-12, 504, which has a half-life of 16-18 hours. The lipophilicity or the metabolic stability, or both, associated with the chloro moiety may account for this difference in half-life.

Information provided by the manufacturer indicates that tolazamide appears to be metabolized to six major metabolites by man. These metabolites have been isolated from the urine of patients treated with tolazamide. Three of these six metabolites possess hypoglycemic activity. These three metabolites, which have been tested for hypoglycemic activity in rats, were found to depress blood sugar; the p-carboxyl metabolite is 0.31 times as potent as tolbutamide; the 4-carboxy-methyl metabolite possesses 1.4 times the potency of tolbutamide; and the metabolite which is hydroxylated on either the 3 or 4 position of the azepinyl cycle is 2.5 times the potency of tolbutamide. Of importance is the fact that these metabolic products of tolazamide possess hypoglycemic activity, whereas tolbutamide is metabolized to the p-carboxyl compound which is devoid of hypoglycemic activity.

In summarizing the results of clinical experience with a number of sulfonylureas, McMahon[4] states that chronic administration to more than 2,000 diabetic patients indicated tolazamide was from 5-10 times more potent than tolbutamide. There were no cases of jaundice reported; the incidence of side effects was less than 5 percent. The p-chloro analogue (U-12, 504) of tolazamide was also compared. It was of equivalent potency, and the incidence of side effects was also less than 5 percent. This p-chloro compound, however, gave rise to several cases of jaundice.

Stern[5] has proposed three requirements for a superior oral hypoglycemic agent. The drug should be of sufficiently long duration of

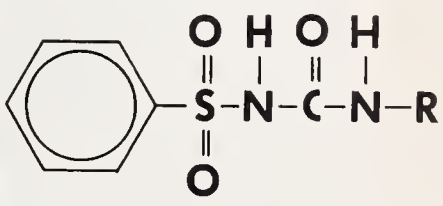

Fig. 2.

Frankly, most antihypertensives are pretty good if you give an adequate dose. I'm looking for one with a simple regimen so that mix-ups in doses and therefore the chance of side effects are minimized.

Regroton®

chlorthalidone 50 mg. reserpine 0.25 mg.

1 tablet daily brings pressure down

Advantage: Both components of Regroton are long-acting.
Average dosage: One tablet daily with breakfast.
Contraindications: History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases.
Warning: Discontinue 2 weeks before general anesthesia, 1 week before electroshock therapy, and if depression or peptic ulcer occurs. With administration of enteric-coated potassium supplements, the possibility of small bowel lesions should be kept in mind.
Precautions: Reduce dosage of concomitant antihypertensive agents by one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take particular care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use with caution in patients with ulcerative colitis, gallstones, or bronchial asthma.
Side effects: Nausea, vomiting, diarrhea, muscle cramps, headaches and dizziness. Potential side effects include angina pectoris, anxiety, depression, drowsiness, hyperglycemia, hyperuricemia, lassitude, leukopenia, nasal stuffiness, nightmare, purpura, urticaria, and weakness.
For full details, see the complete prescribing information.
Availability: Bottles of 100 and 1000 tablets.

Geigy

action to permit maintenance on a single daily dose. On the other hand, its duration of action should not be so prolonged that the effects are cumulative. Finally, the compound must be safe and relatively free from side effects.

Stern[5] clinically evaluated tolazamide in 52 diabetics who were given the drug for as long as 20 months. The half-life of the drug was found to be such that it could be given in a single daily dose. The duration was not so long as to be cumulative. No patient developed hypoglycemia. The study indicated that on a milligram basis tolazamide was highly effective in two-thirds of the diabetics evaluated, and it produced better control in 35.5 percent of cases previously treated with tolbutamide. There was no evidence of toxicity.

The Upjohn Company summarizes the therapeutic uses for tolazamide as follows: for the stable or maturity-onset type of mild or moderately severe diabetes mellitus. About one-third of diabetic patients purported to be primary or secondary failures with other sulfonylureides or with phenformin (DBI, US Vitamin & Pharmaceutical Corp.) have been reported to respond satisfactorily to tolazamide therapy. Patients unresponsive to previous oral therapy may respond to combined tolazamide and phenformin therapy. It is also noted that some patients who have developed significant side effects or intolerance to other sulfonylureides or phenformin may subsequently be successfully maintained on tolazamide.

The manufacturer also lists these contraindications: Tolinase is not indicated in the therapy for diabetic patients who have infection or severe trauma; who are undergoing surgery; who have ketosis, acidosis, or coma; or who give a history of repeated bouts of keto-acidosis or coma.

Tolazamide is not indicated in the therapy of juvenile or labile (brittle) diabetes. Tolinase has not been studied extensively in patients with concurrent liver, renal, or endocrine disease, hence its use is not recommended in these conditions and is contraindicated in patients with uremia.

More information relative to tolazamide is available through the cited references. For a concise resume on the general field of oral hypoglycemic agents and specific monographs on acetohexamide (Dymelor, Eli Lilly and Co.), chlorpropamide, tolbutamide, and phenformin the interested reader should refer to ch. 43 of *New Drugs* (1966 ed.).[6]

—Jaime N. Delgado, Ph.D., Frank P. Cosgrove, Ph.D., and Eugene I. Isaacson, Ph.D.

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2. Wright, J. B., and Willette, R. E.: Antidiabetic Agents. N^4-Arylsulfonylsemicarbazides, J. Med. Pharm. Chem. 5:815 (July 23) 1962.
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5. Stern, S. B.: Clinical Study of Tolazamide: A New Antidiabetic Sulfonylurea, J. Louisiana Med. Soc. 117:198 (June) 1965.
6. *New Drugs:* 1966 ed., American Medical Association, Chicago, 1966.

Postgraduate Courses

Psychiatry in Clinical Practice, Augusta, Ga., Aug. 24-25.—The Medical College of Georgia, the Medical Association of Georgia, and the Georgia Academy of General Practice will present this course at the Medical College of Georgia.

The program will include several areas of clinical psychiatry which frequently confront physicians in their daily practice. The course will be acceptable for 14 accredited hours by the American Academy of General Practice.

Areas covered by the course include psychosomatic medicine, behavior problems in children, and pre- and post-hospital care of the psychotic patient.

Registration fee is $40. Complimentary enrollment is granted to interns and residents. The Department of Continuing Education is aiding registrants with hotel accommodations. For registration, reservation information, or application, write Department of Continuing Education, Medical College of Georgia, Augusta, Ga. 30902. Advance registration is required.

Texas State Department of Health Laboratory Workshops, Austin.—The Texas State Department of Health will sponsor several laboratory workshops in the fall. Workshops are designed to accommodate workers in public health, hospital, clinical, and other medical laboratories. Usually, only from 4 to 12 workers can be accommodated in each workshop.

Although there is no registration fee, admittance to all workshops is by advance registration only. Applications should be addressed to Dr. J. V. Irons, Director of Laboratories, Texas State Department of Health, 1100 W. 49th St., Austin 78756.

Topics and dates of workshops are Introductory Fluorescent Antibody, Sept. 7-9; Parasitology, Sept. 14-16; Tuberculosis Mycobacteriology, Sept. 19-21; General Mycology, Sept. 21-23; Miscellaneous Bacteriology, Oct. 3-7; and Serological Techniques in the Identification of Bacteria, Oct. 10-12.

Advances in Cutaneous Medicine, Rochester, Minn., Sept. 28-Oct. 1.—The American College of Physicians (ACP) will present through the Mayo Clinic and the Mayo Graduate School of Medicine, University of Minnesota, a four-day course designed for internists and for those interested in cutaneous medicine.

Clinical subjects covered will demonstrate the relationship between cutaneous and systemic disease and emphasize the method of utilizing the techniques of clinical and laboratory investigation for diagnosis. Each instructor will correlate the fundamental advances in pathophysiology with the clinical subject matter. Basic discussions of etiology will be related to prognosis and approach to therapy.

The course has been divided into discussions which relate the skin to the organ systems including vascular diseases of the skin, endocrine diseases involving the skin, photosensitivity diseases and lupus erythematosus, the skin and joints, the skin and the gastrointestinal tract, the skin and the hematopoietic system, the collagen diseases, melanogenesis and cutaneous hypersensitivity. Panel discussions will follow each session to permit direct questions and answers.

Tuition fee for ACP members is $60; for nonmembers, $100. Only ACP members will be accepted in courses immediately upon receipt of application. The names

of nonmembers will be placed on waiting lists in the order in which they are received. Three weeks after the release of the course outline (earlier if possible) nonmembers will be notified whether sufficient room for them remains in the course. They will be officially registered and notified accordingly at that time.

Minimum course registration will be 50; maximum registration, 140.

Government personnel wishing to attend an ACP course should be registered in the course before requesting authorization from the government agency concerned.

Registration forms and requests for information should be directed to Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine St., Philadelphia 19104.

The Care of the Critically Ill Medical Patient, Syracuse, N.Y., Oct. 3-7.—The American College of Physicians will present through the State University of New York College of Medicine at Syracuse, N.Y., a five-day course in the care of the critically ill patient.

Insofar as possible, the basic physiological defects leading to critical medical illnesses will be defined and the rational management of these defects will be discussed or demonstrated.

Dog demonstrations will be presented to illustrate more clearly certain of these defects and to explain the background for their correction. The discussions will all be relatively short so that a broad spectrum of critical illnesses may be included.

Subjects to be considered include the following critical illness areas: cardiac arrhythmia, heart failure, shock, central nervous system disorders, and blood, endocrine, metabolic, and infectious diseases. Principles of patient care in these crises will be emphasized.

Minimal course registration will equal 40 participants; maximal registration will be 80. For information concerning tuition fees and course registration, see *Advances in Cutaneous Medicine* preceding.

Further information can be obtained or application made by writing Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine St., Philadelphia 10104.

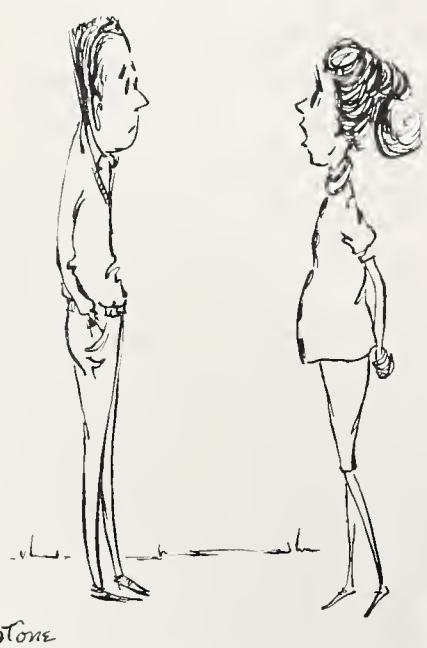
Microbiology, Atlanta, Ga.—The Laboratory Branch of the Communicable Disease Center of the US Public Health Center will present a series of laboratory refresher short courses in special subdivisions of microbiology. Various courses are being offered during an 11-month period.

Courses with registration closing dates through September are Blood Cell Morphology, Oct. 10-14 (closing date Aug. 29); Principles of Complement Fixation, Oct. 10-21 (closing date Aug. 29); Basic Laboratory Methods in Virology, Oct. 17-27 (closing date Sept. 6); Laboratory Methods in Special Medical Bacteriology, Oct. 24-28 (closing date Sept. 12); Laboratory Methods in Respirovirology, Oct. 28-Nov. 4 (closing date Sept. 19); and Laboratory Methods in Anaerobic Bacteriology, Oct. 31-Nov. 11 (closing date Sept. 19).

Information and application forms may be obtained by writing the Training Office, Laboratory Branch, Communicable Disease Center, Atlanta, Ga. 30333.

International Seminar on Medical Genetics, Birmingham, Ala., Aug. 31-Sept. 3.—The University of Alabama Medical Center will host an International Seminar on Medical Genetics Aug. 31-Sept. 3. Participants and discussants will represent the various areas of human genetics and will come from several states and foreign countries.

The program schedule will include a day of papers and discussions on each of three general topics: Aug. 31, biochemical genetics; Sept. 1, population genetics; and Sept. 2, clinical genetics. Informal discussions will take place on Sept. 3.



"Frankly, Bobby, I was expecting you to do more than send me a get well card."

The seminar is sponsored by the Medical College of Alabama's Division of Continuing Education and the Postgraduate Education Division of the University of Alabama School of Dentistry. The meeting is being supported by a Department of Health Children's Bureau grant to Drs. Sara and Wayne Finley, program directors.

Invitations to the seminar have been extended to selected non-Alabama-resident guests and to all physicians, dentists, and public health officials of the State of Alabama. Also invited to attend are representatives of various health organizations including the American Society of Human Genetics, the American Academy of Pediatrics, World Health Organization, the Kennedy Foundation, the Children's Bureau of the Department of Health, Education and Welfare, and the National Institute of Child Health and Human Development of the US Public Health Service.

The meeting, to be held in the Engineering Auditorium, will be open to all interested individuals. There is no registration fee.

Inquiries should be addressed to Cytogenetics Laboratory, Department of Pediatrics, University of Alabama Medical Center, 1919 7th Ave. S., Birmingham, Ala. 35233.

Clinical Aspects of Psychosomatic Disease, Houston, Oct. 22.—A one-day symposium will be presented by the Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston and the Frederick R. Lummis Medical Foundation.

The course, to be held in the Texas Medical Center at Houston, is intended to give practicing physicians a useful and authoritative review of current diagnostic and therapeutic approaches to common psychosomatic problems.

Further information may be obtained by writing to the Division of Continuing Education, The University of Texas Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston 55025.

Laryngology and Bronchoesophagology, Chicago, Oct. 31-Nov. 12.—The Department of Otolaryn-

gology of the Illinois Eye and Ear Infirmary and the College of Medicine of the University of Illinois at the Medical Center, Chicago, will conduct a postgraduate course in laryngology and bronchoesophagology.

The course is limited to 15 physicians and will be held largely at the new Illinois Eye and Ear Infirmary, 1855 West Taylor Street, Chicago.

The course, under the direction of Dr. Paul H. Holinger, will include visits to a number of Chicago hospitals. Instruction will be provided by means of animal demonstrations and practice in bronchoscopy and esophagoscopy at diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants should write directly to the Department of Otolaryngology, College of Medicine of the University of Illinois at the Medical Center, Post Office Box 6998, Chicago 60680.

Mycology Workshop, Oct. 25-28, San Antonio.—A cooperative program will be presented by the Texas State Department of Health Laboratory personnel along with personnel from the State Tuberculosis Hospital in San Antonio. The systemic fungi will be emphasized, and dermatophytes will be considered.

Applications for workshops at the tuberculosis hospitals may be addressed to Dr. Robert B. Skinner, Director, Division of Tuberculosis Hospitals, State Health Department, 1100 W. 49th St., Austin 78756.

Pima Indians Studied

Pima Indians of Arizona are cooperating in a health survey being conducted by the US Public Health Service to determine the prevalence of hypertension and its significance as a risk factor in heart disease. Findings will be compared with other US ethnic groups, as will results of a simultaneous study on the prevalence of trachoma and ocular tension.

Participating in the research are members of the Heart Disease Control Program in Washington, D.C., and ophthamologists of the USPHS Indian Hospital in Phoenix, in behalf of the USPHS Epidemiology Branch of the National Institute of Neurological Disease and Blindness in Bethesda, Md.

Pamphlets

"Home Care and the Incontinent Patient," a 48-page handbook, is available from Johnson & Johnson free to druggists, doctors, and institutions for distribution to consumers. These professional sources may obtain the pamphlet in quantity by writing to Johnson & Johnson, New Brunswick, N. J. 08903.

The pamphlet includes a checklist of necessities for equipping the patient's room, a discussion of the physical and emotional needs of the patient, instructions for his proper personal care, helpful hints on retraining incontinent patients, and a list of health and welfare agencies which may be of aid to the patient's family.

For doctors interested in business consultants, the Society of Professional Business Consultants has published a 1966 roster which lists individual members alphabetically and geographically, professional business consulting firms, and SPBC officers and directors. The roster is published and distributed by *Physician's Management*, *Pediatrician's Management*, *Surgeon's Management*, and *Obstetrician's Gynecologist's Management*.

Copies can be obtained by writing SPBC Executive Secretary, 866 North Wabash Avenue, Chicago, Ill. 60611.

The Health Insurance Institute has announced publication of the eighth annual edition of its booklet, "A List of Current Health Insurance Books."

Two new sections in the 48-page booklet list a variety of statistical reference sources, and background studies on Medicare. Sixteen new titles have been added to the booklet, and 17 older titles have been deleted. The new edition lists a total of 195 books, 29 periodicals, and 34 organizations dealing with the financing of health care. The booklet is divided into 12 sections. The chapter topics include general studies on health insurance, textbooks on health insurance, careers, for the consumer, family money management, background studies, and other insurance booklists.

The 1966 booklet, which lists double the number of books in the first edition (1959), has been distributed among 4,000 editors, economists, teachers, librarians, writers, insurance men, and medical, hospital and allied health care people. It serves as a reference guide on current writings on all aspects of health insurance. The institute will provide single copies of the booklet without charge. Write to Health Insurance Institute, 277 Park Ave., New York, N.Y. 10017.

Two new publications on emphysema and chronic bronchitis, "Chronic Respiratory Diseases, A Growing Menace" and "Management of Chronic Obstructive Respiratory Diseases," have just been released by the Public Health Service, U. S. Department of Health, Education, and Welfare.

"Chronic Respiratory Diseases, A Growing Menace," PHS Publication No. 1445, describes the increasing significance of these diseases and outlines the functions and activities of the new PHS control program.

"Management of Chronic Obstructive Respiratory Diseases," PHS Publication No. 1457, is a resume of the Eighth Annual Emphysema Conference held in Aspen, Colo., in June, 1965. The booklet was edited by Dr. Thomas L. Petty, of the University of Colorado Medical Center. The conference, sponsored by the Public Health Service, brought together leading authorities on emphysema to discuss up-to-date methods for treatment and care of respiratory victims. The Aspen publication contains sections on definitions, respiratory failure, right heart failure and pulmonary hypertension, antibiotics and infections, home and clinic care, pharmacologic agents, and surgery.

Single copies are available from the Public Health Service, Department of Health, Education, and Welfare, Washington, D. C., 20201. The booklets may be purchased in quantity from the Superintendent of Documents, Government Printing Office, Washington, D. C., 20402, at 30 cents a copy, or $22.50 per 100 copies.

Diagnosis:

cystitis?
pyelonephritis?
pyelitis?
urethritis?
prostatitis?

in any case,
usually gram-negative*

Therapy:

two 500 mg. Caplets® q.i.d
(initial adult dose)

Indications: Urinary tract infections caused by gram-negative and some gram-positive organisms.

Side effects: Mainly mild, transient gastrointestinal disturbances; in occasional instances, drowsiness, fatigue, pruritus, rash, urticaria, mild eosinophilia, reversible subjective visual disturbances (overbrightness of lights, change in visual color perception, difficulty in focusing, decrease in visual acuity and double vision), and reversible photosensitivity reactions. Marked overdosage, coupled with certain predisposing factors, has produced brief convulsions in a few patients.

Precautions: As with all new drugs, blood and liver function tests are advisable during prolonged treatment. **Pending further experience, like most chemotherapeutic agents, this drug should not be given in the first trimester of pregnancy. It must be used cautiously in patients with liver disease or severe impairment of kidney function.** Because photosensitivity reactions have occurred in a small number of cases, patients should be cautioned to avoid unnecessary exposure to direct sunlight while receiving NegGram, and if a reaction occurs, therapy should be discontinued. The dosage recommended for adults and children should not arbitrarily be doubled unless under the careful supervision of a physician. Bacterial resistance may develop.

When testing the urine for glucose in patients receiving NegGram, Clinistix® Reagent Strips or Tes-Tape® should be used since other reagents give a false-positive reaction.

Dosage: Adults: Four Gm. daily by mouth (2 Caplets® of 500 mg. four times daily) for one to two weeks. Thereafter, if prolonged treatment is indicated, the dosage may be reduced to two Gm. daily. Children may be given approximately *25 mg. per pound* of body weight per day, administered in divided doses. The dosage recommended above for adults and children should not arbitrarily be doubled unless under the careful supervision of a physician. Until further experience is gained, infants under 1 month should not be treated with the drug.

How supplied: Buff-colored, scored Caplets® of **500 mg. for adults,** conveniently available in bottles of 56 (sufficient for one full week of therapy) and in bottles of 1000. **250 mg. for children,** available in bottles of 56 and 1000.

References: (1) Based on 23 clinical papers, 1512 cases. Bibliography on request. (2) Bush, I. M., Orkin, L. A., and Winter, J. W., in Sylvester, J. C.: Antimicrobial Agents and Chemotherapy—1964, Ann Arbor, American Society for Microbiology, 1965, p. 722.

Winthrop

Winthrop Laboratories, New York, N. Y. 10016

NegGram

Brand of

nalidixic acid

a specific anti-gram-negative

eradicates most urinary tract infections...

- Low incidence of untoward effects; no fungal overgrowth, crystalluria, ototoxic or nephrotoxic effects have been observed.

- "Excellent" or "good" response reported *in more than 2 out of 3* patients with either chronic or acute gram-negative infections.[1]

*As many as 9 out of 10 urinary tract infections are now caused by gram-negative organisms: E. coli, Klebsiella, Aerobacter, Proteus, Paracolon or Pseudomonas[2]... However, infections of the urethra and prostate caused by non-gonococcal gram-negative organisms are believed to be less prevalent.

Florida Court Decision Could Mean Fresh Challenges for Blood Banks

ONCE MORE a court, in determining the conflicting rights of two parties, has set out reasoning which may have broad and far-reaching implications in the medicolegal field.

The Florida Second District Court of Appeals held on April 22 that the transfer of whole blood from a nonprofit community blood bank to a hospital constitutes the "sale" of a commodity. Recognizing that previous court decisions were unanimous in holding that the administration of blood to a patient in a hospital is a "service" and not a "sale," the Florida court in *Mae Russell vs. Community Blood Bank, Inc., a corporation*, 185 So. 2d 749, found a distinction between the hospital-patient relationship and the blood bank-hospital relationship insofar as the transfer of blood was concerned. At the same time, the courts even seem to cast some doubt upon the long-accepted decision that the transfusion of blood into a patient at a hospital is a service and not a sale.

In the Russell case the plaintiff was infected with serum hepatitis following a blood transfusion in a hospital. She brought suit against the Community Blood Bank, Inc., claiming that she was a "buyer" of blood and that the "sale" to her was a transaction apart from the services rendered to her by the hospital in transfusing the blood in question.

In the past, courts in all jurisdictions where the question has arisen have consistently held that the transfer of blood, whether from blood banks to hospital or hospital to patient, constitutes the rendering of a service since a "sale" would by implication warrant that the blood be pure when, in fact, harmful virus *cannot* be detected in blood. Thus, it has been held to be against public policy to hold a hospital or a blood bank strictly liable when they are supplying an item essential to health, such as whole blood. Previous court decisions have also noted that blood banks are not profit-making institutions advertising their wares for sale to the general public and that the patient receiving blood relies on the skill, judgment, and training of the physician rather than on the reputation of the blood supplier.

While the Florida court recognizes that the traditional reasoning applied in such cases has some validity, the court nevertheless states: "We are drawn to the conclusion that the law of implied warranties applies to the transaction before us."

This radical departure from past decisions is based in part on the California decision in *Gottbanker vs. Cutter Laboratories*, involving contaminated or impure Salk polio vaccine. The court held that, "The vaccine is intended for human consumption quite as much as is food. We see no reason to differentiate the policy considerations requiring pure and wholesome food from those of pure and wholesome vaccine."

The court, of course, recognizes that blood banks must be protected in order that they may continue to render the invaluable service which they do. The Florida decision provides that protection through the doctrine of "unavoidably unsafe products." The court in the Russell case quotes with approval the following language from the Restatement of Torts:

> There are some products which, in the present state of human knowledge, are quite incapable of being made safe for their intended and ordinary use. These are especially true in the field of drugs. Such a product, properly prepared and accompanied by proper directions and warning, is not defective, nor is it unreasonably dangerous.

This decision would require the court to look to the particular situation under consideration, evaluate the past experience with the product, and weigh its health-giving benefits. If the product's past experience justifies its marketing and use, notwithstanding a medically recognizable risk, then the court will not apply strict liability for unfortunate consequences which may occur when the product is used by the public cognizant of the known but not unreasonable risks.

What does this rejection of one legal doctrine and the application of another rather obscure doctrine mean? To the lawyer, it means that the defendant in such a case must now assume the burden of affirmatively proving that the product in question (a drug, whole blood, or the like) is one which falls within the purview of the "unavoidably unsafe products" doctrine. Specifically, the defendant must show that the present state of medical knowledge is inadequate to detect or remove the virus or impurity which has caused the plaintiff's injury or illness. He also bears the burden of proving that the risk of using the product in question is not unreasonable and that the public recognizes that those risks exist. Upon this proof, the court will determine that no strict liability is applied to the product.

But of far greater importance and implication than this narrow legal result of the Florida decision is the finding by a court of record that the transmission of blood is a "sale" and that blood itself is a "commodity." Thus, the transmission of whole blood, once a personal service and immune from regulation and control by those agencies authorized to regulate commerce is now found by a court to be a commodity—a commodity with a difference, or course, but still a commodity.

A practical example of how this ruling by a court may affect the medical profession's control over blood banking is readily seen by reviewing the "Kansas City case," where an action was brought by the Federal Trade Commission (FTC) against the Community Blood Bank of the Kansas City Area, Inc., the Kansas City Area Hospital Association, and 14 hospital pathologists.

Two commercial blood banks com-

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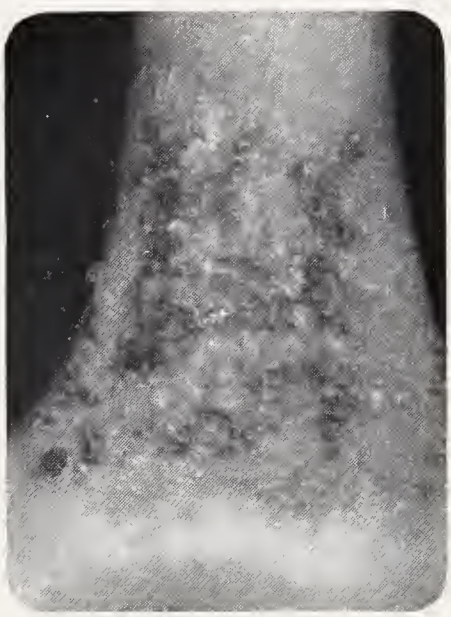
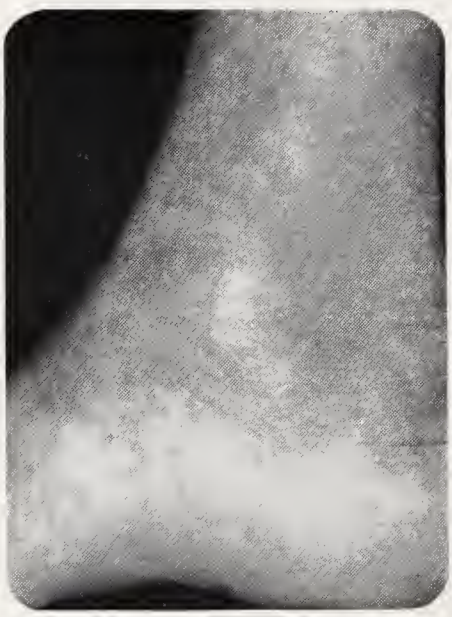


Before treatment



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plained that the Community Blood Bank, area hospitals, and pathologists were all involved in a conspiracy to restrain trade whereby the two complaining commercial blood banks were unable to sell blood. Evidence was taken for a 15-week period during the summer of 1963 by the FTC, and more than 100 witnesses were heard. In an initial decision the FTC hearing examiner ruled that a conspiracy did exist between the various defendants and that they were hampering and restraining trade. In so determining, the FTC examiner found that human blood was a commodity which was under the jurisdiction of the Federal Trade Commission.

The hearing examiner's decision was appealed to the full Federal Trade Commission and argued in December, 1964. As of this date, there has been no decision by the full Federal Trade Commission. But, while the FTC is still in the process of determining whether or not the transmission of blood is a sale or a service, and whether or not it has any jurisdiction in regulating this transmission, a court for the first time has agreed with the decision made by the FTC hearing examiner in Kansas City. A governmental agency now has a court decision which authorizes the invoking of its antitrust regulatory powers in the field of blood banking.

Certainly the decision in the Russell case and the FTC action against the Kansas City Blood Bank serve to perhaps radically change the entire legal aspects of blood banking. It is to be hoped that such rulings will give great impetus to the passage of proposed legislation such as that of Sen. Edward V. Long of Missouri, who has authored a bill which would remove nonprofit blood banks from the jurisdiction of the Federal Trade Comission and state administrative bodies of the same nature. Hopefully, such legislation will be passed.

Nevertheless, such decisions as those discussed cannot but help cause great harm to the present system of not-for-profit blood banks, often organized, supported, and sustained by local medical societies.

—Philip R. Overton, LL.B.,
Sam V. Stone, Jr., LL.B.,
Austin, Legal Counsel,
Texas Medical Association.

Texas Governor Proclaims State Nursing Home Week

Governor John Connally has designated Sept. 11-17 as Nursing Home Week in Texas.

In conjunction with this event Texas Nursing Home Association (TNHA) members will hold open house activities and the association will hold four Medicare seminars, announced John M. Crawford, TNHA executive director.

The Medicare seminars for nursing home personnel were originally scheduled for the week of Aug. 9-12. The revised seminar schedule is (1) Dallas, Adolphus Hotel, Sept. 13; Lubbock, Holiday Inn Parkway, Sept. 14; Houston, Sheraton-Lincoln Hotel, Sept. 15; and San Antonio, El Tropicano Motor Hotel, Sept. 16.

Governor Connally said the 875 nursing and custodial homes in the state "have greatly enhanced the quality of care available to the aged." He also lauded TNHA for its "substantial contribution to higher nursing home standards and to resulting improved patient care." Four hundred nursing homes are members of the association.

Personals

Dean A. J. **Gill** of the University of Texas Southwestern Medical School in Dallas has requested that he be relieved of administrative duties at the convenience of the Board of Regents.

He will return to full-time professorship in pathology.

University Chancellor Harry Ransom has appointed a faculty committee to advise the administration on nominations.

The committee will be headed by **Dr. G. Thomas Shires**, chairman, Department of Surgery. Members are **Dr. Heinz F. Eichenwald**, chairman, Department of Pediatrics; **Dr. Samuel M. McCann**, chairman, Department of Physiology; **Dr. Donald W. Seldin**, chairman, Department of Internal Medicine; **Dr. Parkhurst Shore**, professor of pharmacology; **Dr. Harry M. Spence**, chairman, Urology Division; **Dr. Vernie A. Stembridge**, professor of pathology, and **Dr. Robert Stubblefield**, chairman, Department of Psychiatry.

Dr. Truman Blocker, executive director of the University Medical Branch in Galveston, and **Dean F. C. Pannill, Jr.**, of the University's South Texas Medical School in San Antonio will serve as consultants to the committee, without vote.

Dean Gill will continue to head the administration of Southwestern Medical School until his successor is elected.

The following Texas physicians have been elected to serve on the Advisory Board of the Texas Medical Assistants Association (TMAA) for 1966-67: **Dr. Wallace H. Bassett** of Dallas, Dr. C. **Denton Kerr** of Houston, **Dr. John Lingenfelder** of Corpus Christi, **Dr. Oscar L. Morphis** of Fort Worth, Dr. **William H. Nash** of San Antonio, and **Dr. Travis Smith** of Abilene. One physician, **Dr. Howard O. Smith** of Marlin, will serve as the liaison officer between TMA and TMAA.

Eight Texas doctors were admitted as fellows of the American College of Chest Physicians in June. They are **Dr. William A. Cook** of Dallas; **Dr. Martin L. Dalton, Jr.**, of Lubbock; **Dr. John P. Darby, Jr.**, of Big Spring; **Dr. John R. Derrick** of Galveston; **Dr. Hussam A. Fadhli** of Port Arthur; **Dr. H. Edward Garrett** of Houston; **Dr. Jimmy F. Howell** of Houston; and **Dr. William Cone Johnson** of Temple.

Dr. James D. Murphy of Fort Worth, President of Texas Medical Association, has been named by Texas House Speaker Ben Barnes to the advisory committee on the Speaker's Symposium on Language Disabilities. The symposium will take place on Sept. 16 in Austin Municipal Auditorium. Dr. Murphy will attend a planning session in Austin before that date.

Dr. Charles A. Berry, chief of Center Medical Programs at the NASA Manned Spacecraft Center, Houston, was awarded the American College of Chest Physicians' Medal for outstanding contribution in diseases of the chest. The presentation was made at the organization's annual president's banquet June 26. On June 25 Dr. Berry participated in a panel discussion on "Cardiopulmonary Aspects of Space Travel" during one of the scientific sessions of the organization's 32nd Annual Meeting.

Dr. B. E. Pickett of Carrizo Springs was honored by the Dimmit County Chamber of Commerce on July 14 with a "Dr. Pickett Day" celebration. Dr. Pickett, who is still practicing medicine at the

age of 90, was president of Texas Medical Association in 1947-48.

Dr. Pickett's activities include membership in the American Medical Association and the American Academy of General Practice. He was a charter member of the American Geriatric Society and was a member and chairman of the Texas delegation to the American Medical Association for several years. He served for many years as chairman of the AMA Council on Constitution and By-Laws. He is a past president of the Texas Public Health Association.

Three Texas physicians are currently serving as lieutenant governors of the Texas-Oklahoma district of Kiwanis International. They are **Dr. Henry Forcher** of Edinburg, **Dr. William M. Center** of San Antonio, and **Dr. N. M. Atkins** of Waco.

Dr. Ronald D. Linhardt of Galveston was awarded a residency training grant by the Southern Medical Association on June 10. He is serving his residency in obstetrics and gynecology at the University of Texas Medical Branch in Galveston.

Dr. William D. Nicholson of Lake Jackson has been named "Doctor of the Year" in Brazoria County by the Brazoria County Medical Society Woman's Auxiliary.

The Auxiliary annually recognizes an outstanding doctor in the county who has contributed significantly to the medical profession and his community.

Dr. Nicholson maintains his medical offices in Freeport, where he has practiced for 20 years. He is a past president of the Brazoria County Medical Society, and for the past eight years, he has been the County Society's delegate to the Texas Medical Association.

New Books In TMA Library

Bellows, J. G.: *Cryotherapy of Ocular Diseases,* Philadelphia, J. B. Lippincott Co., 1966.

Buerki, R. A., ed.: Proceedings of the Conference *Pharmaceutical Services for Nursing Homes,* Columbus, Ohio, Ohio State University Press, 1966.

Masters, W. H., and Johnson, V. E.: *Human Sexual Response,* Boston, Little, Brown and Co., 1966.

Moss, William T.: *Therapeutic Radiology: Rationale, Technique, Results,* ed. 2, St. Louis, C. V. Mosby Co., 1965.

Siim, J. C., ed.: *Human Toxoplasmosis: Clinical Aspects and Diagnostic Problems of Toxoplasmosis in Pacdiatrics,* The Eighth International Congress of Paediatrics, Baltimore, The Williams and Wilkins Co., 1960.

Winsor, T., and Hyman, C.: *A Primer of Peripheral Vascular Diseases,* Philadelphia, Lea & Febiger, 1965.

Wood, J. E.: *The Veins, Normal and Abnormal Function,* Boston, Little, Brown and Co., 1965.

Work, H. H., and Call, J. D.: *A Guide To Preventive Child Psychiatry; The Art of Parenthood:* New York, Blakiston Division, McGraw-Hill Book Co., 1965.

NEW JOURNALS

Revista De La Asociacion Medica Mexicana—Norteamericana, Tomo I, no. 1 (Part one: Spanish Translation) Ano I (Enero y Febrero de 1966) Mexico City, Mexico, La Fundacion Minnie L. Anthony-Anderson, 1966.

International Journal of Cancer, vol. 1, no. 1, Manksgaard Publishers, Copenhagen S. (Denmark), January, 1966.

Book Notes

Surgery in America: From the Colonial Era to the Twentieth Century, Selected Writings, A. Scott Earle, (ed.) 280 pp. illus., Philadelphia, W. B. Saunders Co., 1965. $8.50.

DR. EARLE has done an excellent job in his selection of these 24 articles that represent milestones of medical progress not only in the United States but in the entire world. It should be remembered that these selections only bring us up to the 20th century. There have been so many important discoveries in the last 66 years that it will take some time to put these latter ones in proper perspective.

It was disappointing to find that Crawford Long was not mentioned in the preface to the articles on the discovery of ether anesthesia. This is understandable, however, if it is recognized that the editor was working in Sun Valley, Idaho. In this beautiful country he probably did not feel the Southern cause too close to his heart.

All of the regulars are well represented: Ephraim McDowell, Philip Syng Physick, Nathan Smith, William Beaumont, Valentine Mott, James Marion Sims, Rudolph Matas, and William Stewart Halsted—among the best.

Halsted's article on "The Training of the Surgeon" is a must for all young physicians and is worth the price of the book.

The scholarly handling of the book's material by the editor makes this reviewer feel that the upcoming *History of Surgery in America* by Dr. Earle will be excellent. This book is recommended for all medical men interested in the history of their profession.

—WILLIAM M. CRAWFORD, M.D., Fort Worth

Burns: A Symposium. Leon Goldman and Richard E. Gardner. 191 pp., Springfield, Ill., Charles C Thomas, Publisher, 1965. $7.75.

THE AUTHORS present an easily read and understood symposium on the treatment of burns in discussion form. There is nothing dogmatic in the presentation. The authors are even at times in disagreement, as noted in the first chapter on "Burn Shock."

This book covers all aspects of burns from the clinician's point of view: what to see and expect in all clinical phases from initial onset through reconstructive procedures. This book belongs in the library of all physicians who are interested in the treatment of burns.

—C. WADE GREATHOUSE, JR., M.D., Corpus Christi

The Management of Fractures and Soft Tissue Injuries. American College of Surgeons, ed. 2, 365 pp., illus., Philadelphia, W. B. Saunders Co., 1965. $7.50.

THIS SECOND EDITION to the combined *Outline of the Treatment of Fractures and the Early Care of Acute Soft Tissue Injuries* should be an addition to the library of physicians and students. It will act as a cohesive account of these two principal areas of the modern treatment of trauma.

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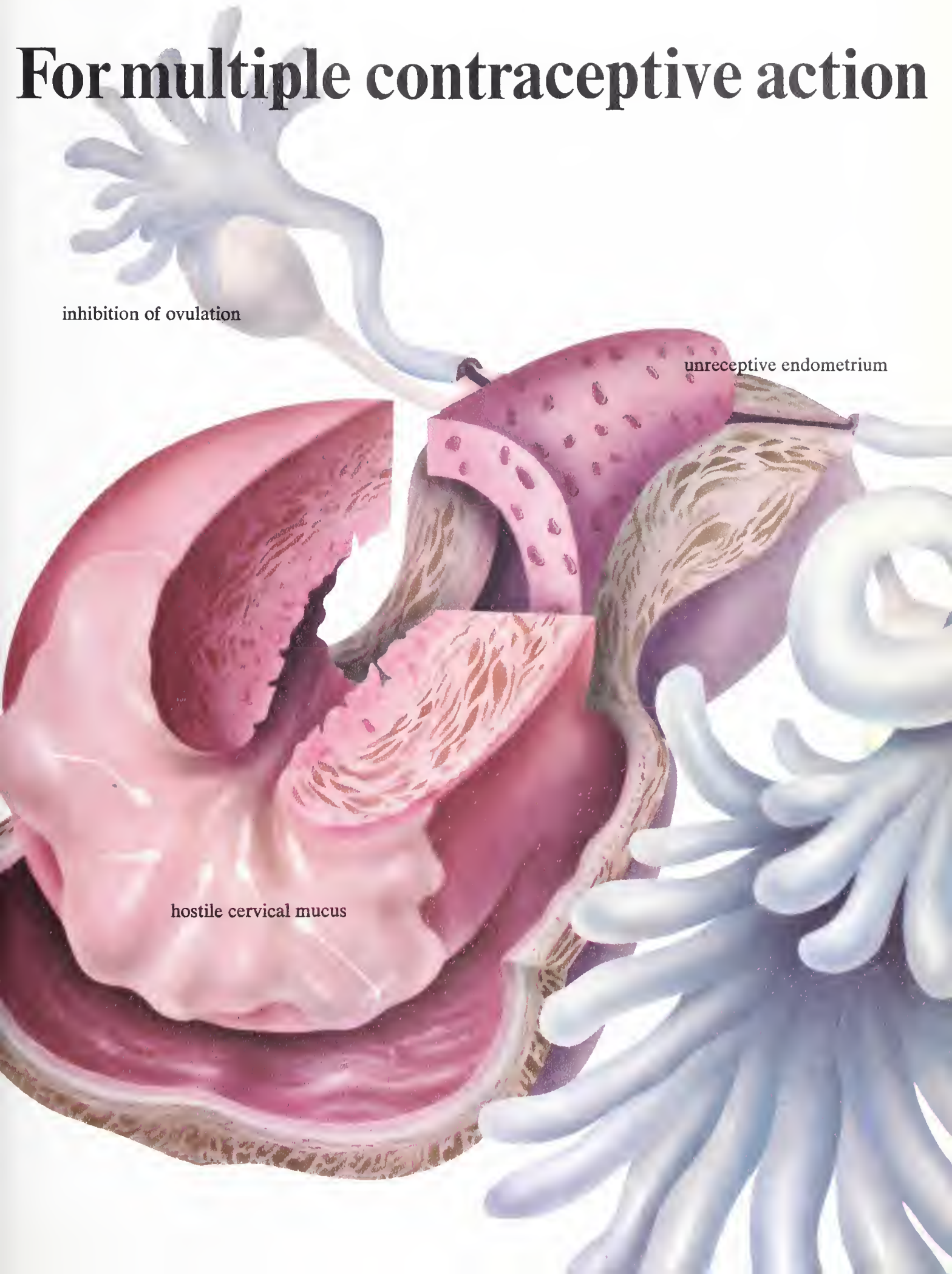

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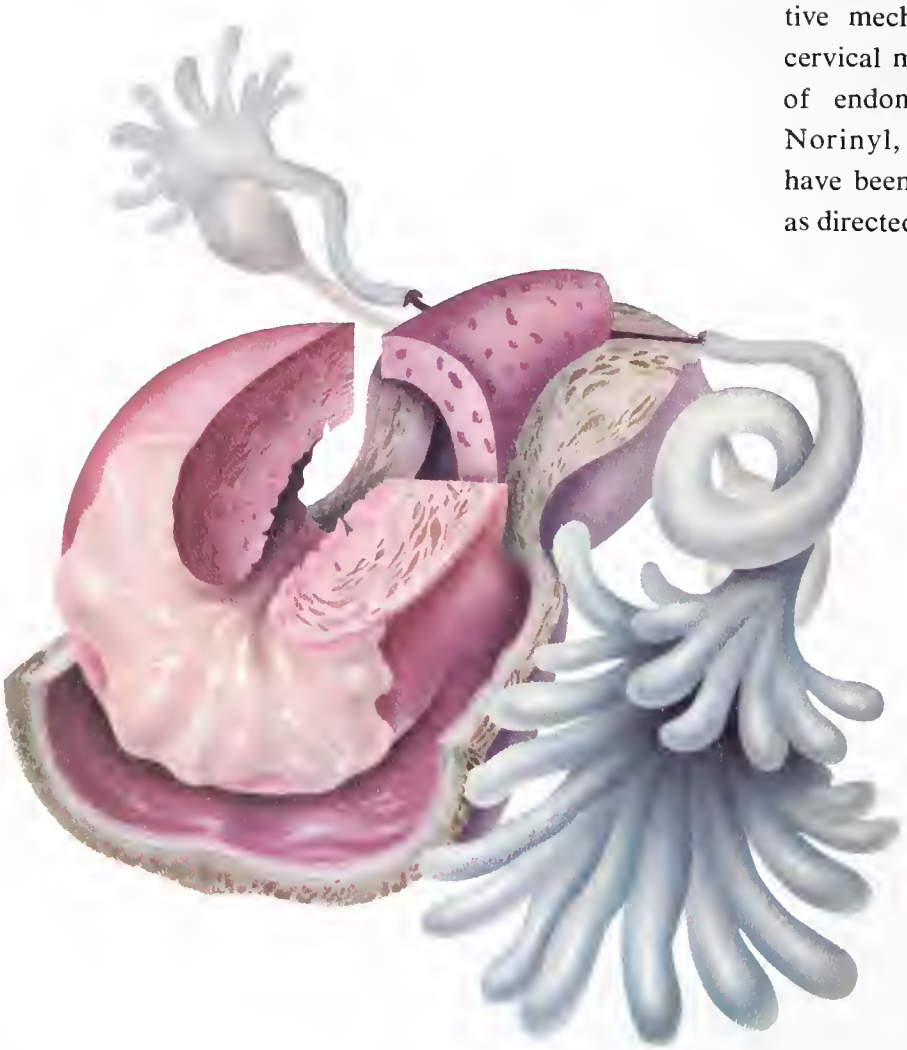

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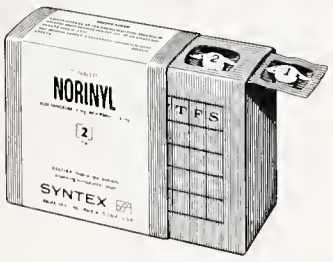

Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb. 29) 1964. 2. Bryans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

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The Heart: Its Function in Health and Disease. Arthur Selzer, M.D. 301 pages, illus., Los Angeles, University of California Press, 1966. $5.95.

THE FIRST of a series of "Perspectives in Medicine," written by Dr. Selzer, clinical professor of Medicine at Stanford University, this book is addressed to the modern reader who is not trained in medicine but who is interested in learning more about the functioning of the human body and factors in everyday living that affect people's health.

This publication serves primarily as a source of information for the patient with heart disease and for his family in implementing the physician's recommendations and aiding in doctor-patient relationships.

The History of Surgical Anesthesia. Thomas E. Keys. 193 pp., illus., New York, Dover Publications, Inc., 1963. $2.

THE MEDICAL PROFESSION owes a debt to Dover Publications for reprinting in paperback so many medical classics that have been previously unobtainable. *The History of Surgical Anesthesia* is another such book. This book was originally published by Schuman for the centennial of the discovery of ether in 1946.

The History of Surgical Anesthesia has only 92 pages devoted to the actual history. This sequence should be classed as an excellent synopsis of the subject. Along with this are many excellent illustrations and a definitive bibliography of 52 pages, which gives references for any phase of the subject that the reader will want to pursue. There is also an excellent 16-page chronology of events in the discovery of anesthesia.

The history begins with Adam and Eve in the *Old Testament:* "and the Lord God caused a deep sleep to fall upon Adam, and he slept," and continues to the present time. There is a concluding chapter on "The Future of Anesthesia" which is to be recommended.

This reviewer having recently spent a week in Rochester, Minn., with the still effervescent Chauncey Leake and Tom Keys, is very happy with the reprint of this book. It is scholarly, definitive; and the price is in the range of the paperback binding. This book is both excellent as reading material and as a reference source.

—WILLIAM M. CRAWFORD, M.D., Fort Worth

Therapeutic Radiology: Rationale, Technique, Results. William T. Moss. ed. 2, 514 pp., illus., St. Louis, C. V. Mosby Co., 1965. $18.75.

THIS IS a practical book on radiation therapy setting forth the indications for and the limitations of conventional and supervoltage methods.

The beginning is valuable because it first discusses the effects of radiation on normal tissues before it takes up the therapy of malignancies of the various organs. The factors governing the size of daily fractionation, such as cell type, volume, beam quality, and time dose relationship are covered. Practical points are discussed as guides to dosage and proper technique for treating the malignancy. Due consideration is given to the location, blood supply, and nature of the tumor bed to be included.

One will find helpful the suggestions for avoiding complications in radiotherapy whether they be in the oral cavity, nasopharynx, breast, esophagus, kidney, cervix, bladder, or central nervous system.

The reviewer was particularly pleased to read the section on radiotherapy of selected soft tissue tumors which included keloids, Dupuytren's contracture, Peyronie's disease, hemangiomas, plantar warts, Wegener's midline granulomatosis, rhabdomyosarcoma, fibrosarcoma, synovial sarcoma, liposarcomas, and Kaposi's sarcoma. These conditions are not commonly seen, and a handy reference is useful in reassuring one that he is handling the condition in an acceptable fashion.

The many helpful thoughts in this book are the result of considerable experience. It is a recommended reference book for anyone doing radiation therapy and particularly for those who are in training. The many timely suggestions in the management of those patients who are afflicted with neoplasms will be especially helpful to the latter group.

—JOE C. RUDE, M.D., Austin

Health Service To Sponsor Study of Chemical Pesticides

Development of a national monitoring network to measure levels of chemical pesticides in the nation's general population has been undertaken by the Office of Pesticides of the Public Health Service, U.S. Department of Health, Education, and Welfare.

The Office of Pesticides, focal point within the Service for programs aimed at the identification and elimination of health hazards associated with pesticides pollution, has awarded $59,500 to the Midwest Research Institute of Kansas City, Missouri, to design and establish operating criteria for the network to monitor pesticides in humans.

A primary objective of the monitoring system will be to obtain on a continuous basis scientific data on the kinds and amounts of pesticides retained in the tissues and organs of people following dietary and atmospheric exposures.

Medical research involving small groups of people has shown that certain pesticides widely used in the U.S. are retained in the human body. One of the purposes of the monitoring program is to determine if such accumulations over a long period of time might be harmful to health.

Because of the large and in-

"Scissors?"

creasing use of pesticides, and because of the possibility that body concentrations might under certain conditions be detrimental, health authorities believe it essential that broad surveillance of pesticides in people be maintained.

Development and operation of the surveillance network has been assigned to the Office of Pesticides' Intelligence System, a specialized medical information center on pesticides and other toxic contaminants.

Plans are to establish within the current year a nucleus of ten monitoring stations, chiefly in large urban centers, to collect human tissues and body fluid samples for pesticide analyses. Eventually the network is to be expanded to 135 stations to form a nationwide monitoring system.

Samples are to be collected in cooperation with physicians, hospitals, and local medical examiners. They will include human fat or adipose material, liver, kidney and brain tissues, and blood and urine. Histories of pesticide exposures also will be obtained from individuals sampled.

These data will then be continuously correlated with toxicological research findings to identify any existing or potential dangers to human health which may be associated with present or future use and exposure to pesticides.

UT Professor Reappointed To Health Consultant Post

Dr. John Autian of The University of Texas, a 1966 Annual Session speaker, has been reappointed as a consultant to the Clinical Center of the National Institutes of Health on matters dealing with drugs and plastics.

A professor of pharmacy, Dr. Autian is director of the UT College of Pharmacy's Drug-Plastic Research and Toxicology Laboratories.

Dr. Autian was honored recently on the basis of his research by being elected a member of the editorial board of the new *Journal of Biomaterials Research.*

New Fellowship Announced

A fellowship is available from Texas Tuberculosis Association to third year residents in the thoracic service (respiratory diseases). For additional information and application forms write Texas Tuberculosis Association, P. O. Box 6158, Austin, Texas 78702. Applications must be received by Oct. 1, 1966.

Texas 65 Plan Pays Claims Of More Than $10.1 Million

The Texas 65 Health Insurance Association paid claims in excess of $10.1 million during the 31 months it was in operation before being replaced by Medicare on July 1, according to H. Lewis Rietz, president of the association.

Since Texas 65 began operations on Nov. 1, 1963, following authorization by the Texas Legislature, it has paid claims to 55,879 senior citizens of the state, Mr. Rietz stated.

"This assistance, which was a public service contribution to Texas by the insurance industry, undoubtedly helped many families avoid financial disaster due to accident or illness," Mr. Rietz said.

"Though Texas 65 ceased its health insurance coverages with the coming of Medicare, we will continue in business until all claims incurred prior to July 1 have been settled, as provided in the Texas 65 policy," he continued.

Mr. Rietz said Texas 65 received an average of 81 new claims per day, of which the association had no prior notice, during the first two weeks of July.

"This is about the same average of new claims per day we were receiving prior to July 1. We expect we will continue to receive new claims for several months to come," he said.

Mr. Rietz said Texas 65 paid over $217,800 during the first two weeks of July for claims incurred before the program was terminated on the first of the month.

The 64 member companies of the Texas 65 Health Insurance Association waived the June, or final month's premium, giving the enrollees of the program one month of health insurance coverage without charge.

About 1,000 of those enrolled in Texas 65 were under-age-65 husbands and wives of enrollees, ranging in age from 45 to 65. This group is not now eligible for Medicare, therefore Texas 65 made arrangements with one of its member companies, the Southland Life Insurance Company, of Dallas, to offer them identical coverage at the same price they were paying for Texas 65.

Do I note open contempt for medical science?

Hospital Expense Increases To Record High Per Patient

Hospital expense per patient day in the nation's short-term hospitals increased by $3.90, or 9 percent, during the past year to a record $44.48, reported Dr. Edwin L. Crosby, director of the American Hospital Association.

For the 5,736 nonfederal short-term general hospitals (usually referred to as community hospitals) the AHA statistical survey shows a $798 million increase in total expenses in the last year for a new high of $9.15 billion. Of this total, $5.64 billion was paid out in wages and salaries, an increase of $493 million over the previous reporting year. The nation's community hospitals reported an increase of 53,500 fulltime personnel in 1965 which pushed total personnel employed in these hospitals to a new peak of 1,386,215. On the national average this adds up to 246 full-time personnel per 100 patients per day.

The AHA survey lists a total of 7,123 hospitals (of all types), with 1,703,522 beds, in the 1965 report. This overall total includes federal hospitals and the long-term nonfederal institutions many of which are operated by state and local governments.

In the short-term community hospitals category 5,736 institutions have a total of 741,292 beds which accommodate 91.8 percent of all hospital admissions. The average length of stay in the community hospitals in 1965 was 7.8 days and the average occupancy was 76 percent.

Recent Deaths

DR. L. G. DAVIS

Dr. Leo Goudchaux Davis, 60, Dallas ophthalmologist since 1935, died July 8, 1966, in Dallas.

Dr. Davis was born March 28, 1906, in Bunkie, Louisiana, the son of Dr. David B. and Adele Goudchaux Davis.

Having received high school education from Forest Avenue High School in Dallas Dr. Davis entered Southern Methodist University in 1924. After spending one year there, he transferred to the University of Texas to finish his premedical schooling. In 1927 he entered Baylor University College of Medicine in Dallas. He received his M.D. degree at Baylor in 1931.

Dr. Davis served his internship and his residency at Touro Infirmary in New Orleans. He did postgraduate study at Washington University School of Medicine, St. Louis, serving as a resident in ophthalmology at Barnes Hospital there.

DR. DAVIS

Coming to Dallas in 1935, Dr. Davis served on the staffs of St. Paul's and Mary Shiels hospitals. He had practiced in Dallas ever since 1935, with the exception of service as a Captain in the U.S. Army Air Force from 1942 to 1945 in San Antonio.

Dr. Davis was a member of the Dallas County Medical Society, Texas Medical Association, the American Medical Association, and the Dallas Academy of Ophthalmology and Otolaryngology.

Active in church and community affairs, Dr. Davis was a member of Temple Emanu-El, Variety Club, Columbian Club, and Cipango Club. He was also a member of Phi Delta Epsilon medical fraternity and Phi Sigma Delta social fraternity.

In 1935 Dr. Davis married Gertrude Mathis of St. Louis. Mrs. Davis; a son, Stephen L. Davis of Dallas; a daughter, Mrs. Paul L. Salzberger of Dallas; a brother, Dr. Bert E. Davis of Denton; and a sister, Mrs. Roseland Feldman of Nashville, Tenn., survive.

DR. W. C. WEDEMEYER

Dr. William Charles Wedemeyer, 76, retired Walburg physician, died May 1, 1966, in Temple of acute myeloblastic leukemia.

Since his retirement from active practice, Dr. Wedemeyer had lived on his ranch west of Georgetown.

The son of W. H. and Janie Klanke Wedemeyer, he was born Jan. 20, 1890, in Burton, Tex. He attended schools in Brenham and did undergraduate work at the University of Texas in Austin. He received his M.D. degree from the University of Texas Medical Branch in Galveston in 1917 and held an internship at John Sealy Hospital in Galveston, 1917-1918. He then served overseas with the United States Army Medical Corps in France and Germany.

Dr. Wedemeyer had a general practice in Walburg from 1920 until 1955. He served with the Veterans Administration Hospital in Marlin until his retirement in 1959.

DR. WEDEMEYER

He was a member of the Williamson County Medical Society, Texas Medical Association, and American Medical Association. He belonged to the Methodist Church and was president of his county society in 1946. He had also been president of the Walburg State Bank.

Survivors include his wife, the former Nora Braun; two sons, Carroll C. Wedemeyer and Dr. Horace Howard Wedemeyer, both of Georgetown; a sister, Mrs. D. W. Drawe of Mercedes; and one grandson, Howard Barkley Wedemeyer of Houston.

DR. L. C. SERAFINO

A long time resident and practicing physician in Beaumont, Dr. Louis Charles Serafino, died of heart failure at his home on June 22, 1966.

Dr. Serafino, 73, had practiced medicine in Beaumont since 1919. He was born there on Dec. 10, 1892. His parents were Charles and Olivia Barberia Serafino.

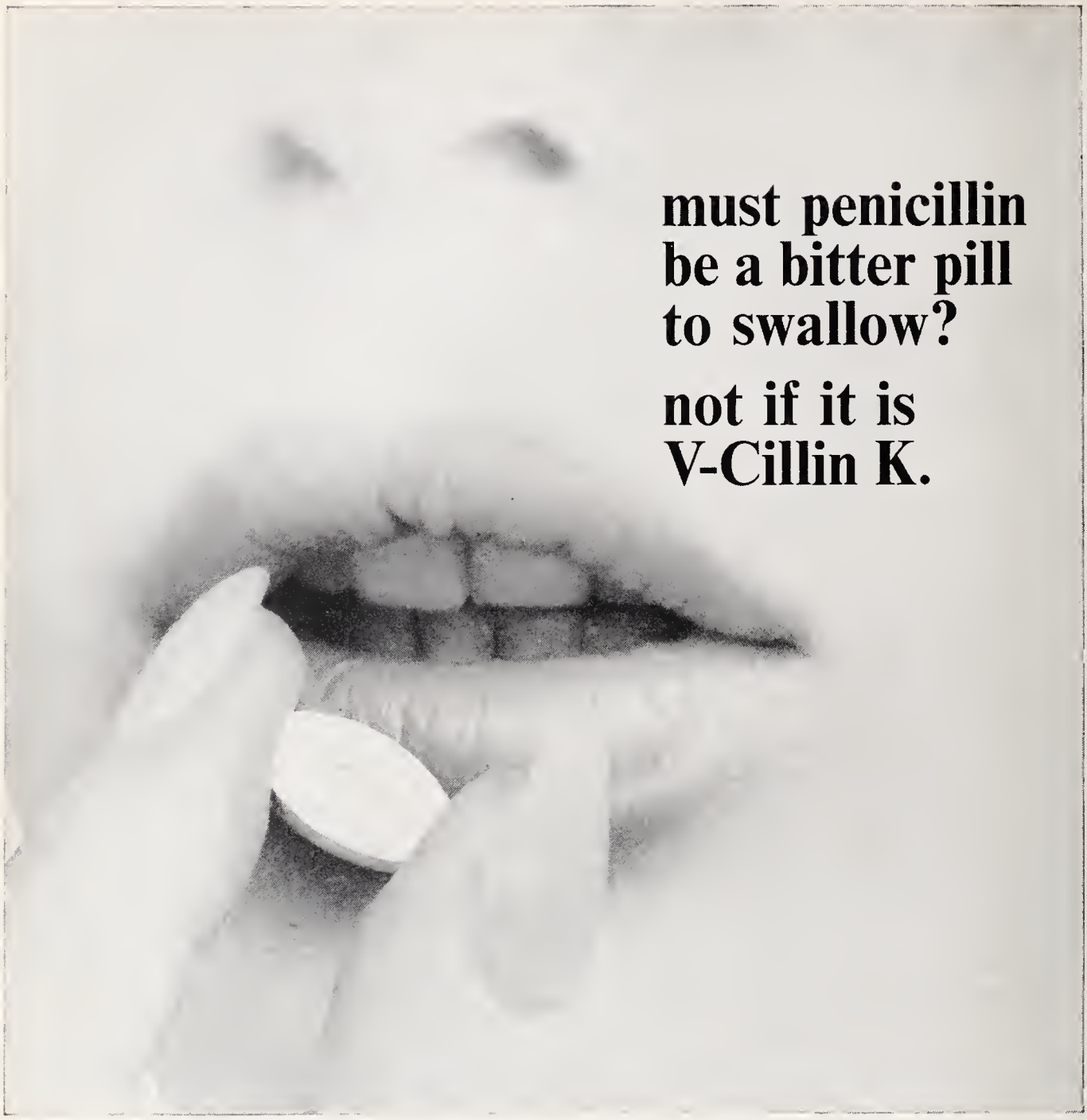
Having graduated from Beaumont High School, Dr. Serafino entered the University of Texas for premedical schooling in 1911. In 1913 he began work at the University of Texas Medical Branch in Galveston. He received his M.D. degree in 1918.

Dr. Serafino interned at John Sealy Hospital in Galveston in 1918-19. In 1919 he returned to Beaumont to practice general medicine with special interest in urology.

A member of the Jefferson County Medical Society, Dr. Serafino also held memberships in Texas Medical Association and the American Medical Association. He was also a member of Theta Kappa Psi medical honorary fraternity, the Benevolent and Protective Order of Elks, and Knights of Pythias. He served as an examiner for the local draft board for 20 years. He was a Catholic.

Dr. Serafino enjoyed the hobbies of hunting and fishing.

In 1919 in Sour Lake, Tex., Dr. Serafino married Miss Cora Ellen Lloyd. His wife survives, as do two sons, Milton L. Serafino of Palestine and L. C. Serafino, Jr., of San Jose, Calif.; and a brother, Victor Serafino of Beaumont.



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Indications: V-Cillin K is an antibiotic useful in the treatment of infections caused by streptococci, pneumococci, and sensitive strains of staphylococci.

Contraindications and Precautions: Although sensitivity reactions are much less common after oral than after parenteral administration, V-Cillin K should not be administered to patients with a history of allergy to penicillin. As with any antibiotic, observation for overgrowth of nonsusceptible organisms during treatment is important.

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COMING MEETINGS

American Medical Association, Clinical Meeting, Las Vegas, Nov. 27-Dec. 1, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Austin, Public Relations Conference, Oct. 1, 1966; Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

AUGUST

American Hospital Association, Chicago, Aug. 29-Sept. 1, 1966. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Congress of Physical Medicine and Rehabilitation, San Francisco, Aug. 28-Sept. 2, 1966. Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

SEPTEMBER

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 8-10, 1966. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Society of Clinical Pathologists, Washington, D.C., Sept. 16-24, 1966. Dr. Lester H. Hoyt, Methodist Hospital, Indianapolis, Ind. 46207, Sec.

Texas Academy of General Practice, Houston, Sept. 12-14, 1966. Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Heart Association, Houston, Sept. 16-18, 1966. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Pediatric Society, Fort Worth, Sept. 29-Oct. 1, 1966. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Blackford Memorial Lectures, Denison, Sept., 1966. Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966, Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill., Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. G. Whittlesey, 219 E. 42nd St., New York, Sec.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Gastroenterology, Philadelphia, Oct. 23-26, 1966. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, Dr. Robert A. Peterman, 3 Craig Court, Totowa Borough, N. J., Sec.

American College of Obstetricians and Gynecologists, Houston, Oct. 24-26, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, February, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American College of Surgeons, Annual meeting, San Francisco, Oct. 10-14, 1966. Dr. Samuel P. Harbison, 230 Lothrop St., Pittsburgh, Sec.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., Sec.

American Gynecological Society. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, New York City, Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Medical Women's Association, Washington, D.C., Nov. 2-5, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Philadelphia, Oct. 1-5, 1966. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Surgical Association. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis, Ind., Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Chicago, Nov. 4, 1966. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104 Sec.

Gastrointestinal Research Group, Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Private Clinics and Hospitals, Austin, December, 1966, A. A. Hubbard, Alvarado Community Hospital, Alvarado, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La., Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec., 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore, Md.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston, Pres.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Division, American Cancer Society, Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Surgical Society, Houston, Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Spring, 1966. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

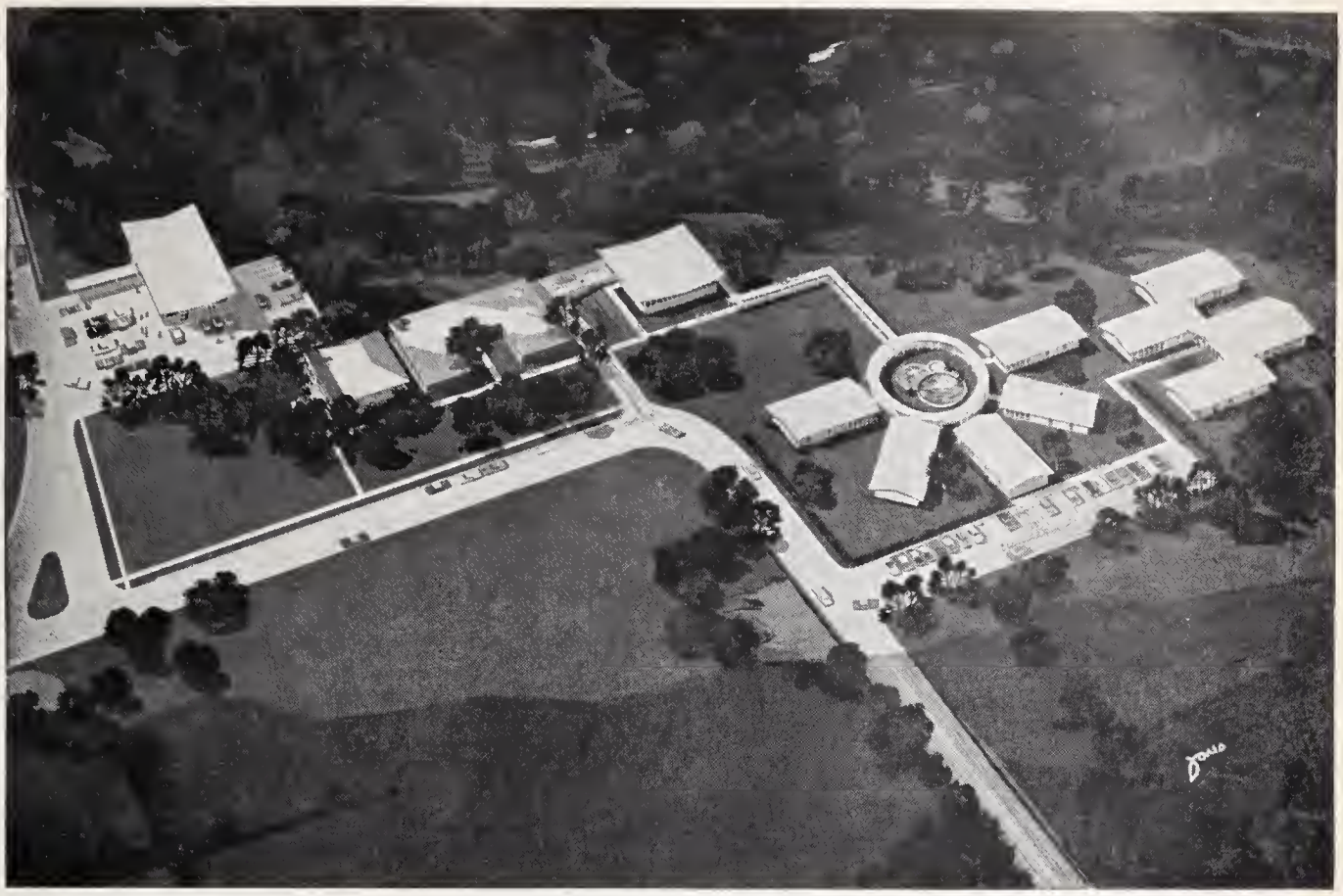
Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Southwest Regional Cancer Conference, Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.



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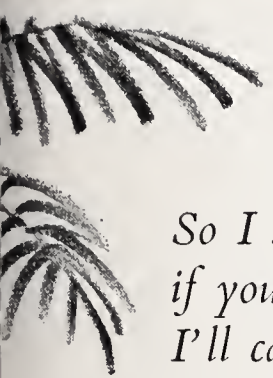


So I said, "All right, Raymond,
if you don't take your cough medicine this minute,
I'll call Doctor Peabody."

Controlling the child is sometimes as big a problem as controlling his cough. But with most children and with most coughs, the job is usually much easier with one of these effective Novahistine formulas.

If it's the useless, exhausting type of cough that often accompanies respiratory infection or allergy, you can provide prompt relief with Novahistine DH. Its decongestant-antitussive action controls frequency and intensity of cough spasms without abolishing cough reflex. And the fresh grape flavor of Novahistine DH appeals to children and adults alike.

When your diagnosis is bronchitis, complicated by thick tenacious exudates, Novahistine Expectorant is particularly useful. It not only provides decongestive action and controls the cough, but also encourages expectoration, thus easing bronchial obstruction.

Use with caution in patients with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Ambulatory patients should be advised that drowsiness may result. Continuous dosage over an extended period is contraindicated since codeine phosphate may cause addiction.

Each 5 ml. teaspoonful of Novahistine DH contains codeine phosphate, 10 mg. (Warning: may be habit forming); phenylephrine hydrochloride, 10 mg.; chlorpheniramine maleate, 2 mg.; chloroform (approx.), 13.5 mg.; l-menthol, 1 mg. (Alcohol 5%). Each 5 ml. of Novahistine Expectorant contains the above ingredients and, in addition, glyceryl guaiacolate, 100 mg.

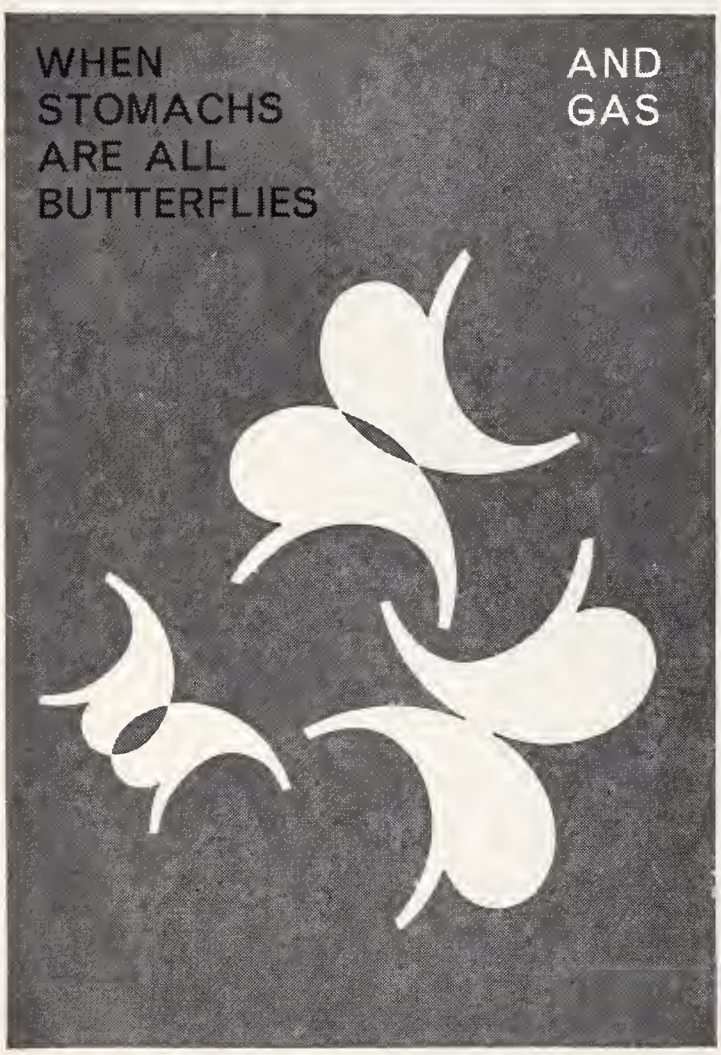
NOVAHISTINE® DH
NOVAHISTINE® EXPECTORANT

DOW **PITMAN-MOORE** Division of The Dow Chemical Company, Indianapolis

DACTILASE®

Each tablet contains:
Dactil® (piperidolate hydrochloride), 50 mg.;
Standardized cellulolytic* enzyme, 2 mg.;
Standardized amylolytic enzyme, 15 mg.;
Standardized proteolytic enzyme, 10 mg.;
Pancreatin 3X** (source of lipolytic activity), 100 mg.; Taurocholic acid, 15 mg.

*Need in human nutrition not established.
**As acid resistant granules equivalent in activity to 300 mg. Pancreatin N.F.



In chronic or acute indigestion, fluttery, gassy stomachs obtain prompt, gratifying relief through the antispasmodic, surface anesthetic and enzymatic activity of Dactilase. Dactilase decreases hypermotility and pain and reduces the production of gas. Dactilase does not induce stasis, but helps restore normal tone. It has little or no effect on enzyme secretions, but *adds* enzymes, thus contributing to the digestive efficiency of the patient.

Side Effects and Contraindications: Dactilase is almost entirely free of side effects. However, it should be withheld in glaucoma and in jaundice due to complete biliary obstruction.

Administration and Dosage: One tablet with, or immediately following, each meal. Tablets should be swallowed whole.

Supplied: Bottles of 60 and 250.

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For cold hands and feet, nothing beats hot stoves—but they *are* awkward to carry around. Now Gerilid, in good-tasting take-along chewable tablets can provide rapid vasodilation of peripheral circulation, bringing real warmth to the extremities and decreasing sensitivity to sudden temperature change. Patients *like* Gerilid and *know* they are getting relief.

WARMTH FOR COLD HANDS AND FEET

GERILID™

Each chewable tablet contains: nicotinic acid (niacin) 75 mg. and aminoacetic acid (glycine) 750 mg.

Administration and Dosage: One or two chewable tablets 3 times a day before meals. If flushing is objectionable, dosage may be lowered. However, tolerance to flushing usually develops without loss of efficacy in regard to vasodilation. The recommended dosage should not be exceeded.

Side effects: Occasional lightheadedness or transient itching which may disappear with continued use. There are no known contraindications; however, caution is advised when there is a concomitant administration of a coronary vasodilator.

Supplied: Packages of 50 chewable tablets.

Also available in liquid form as Geriliquid®, in bottles of 8 and 16 ounces.

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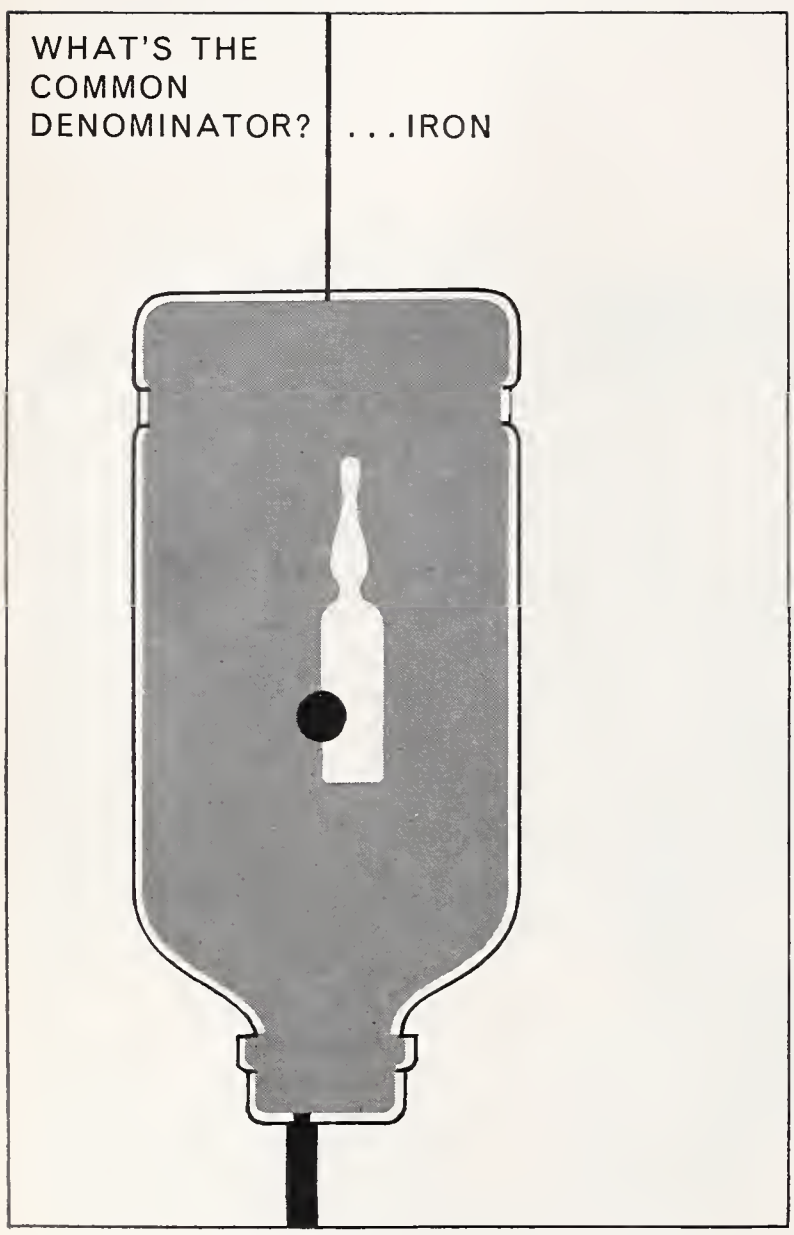
In fact, there's as much iron...250 mg. ...in a 5 cc. ampul of Imferon (iron dextran injection) as in a pint of whole blood. When iron deficient patients are intolerant of oral iron...or orally administered iron proves ineffective or impractical...or if the patient cannot be relied upon to take oral iron as prescribed, Imferon (iron dextran injection) dependably increases hemoglobin and rapidly replenishes iron reserves.



IMFERON®
(iron dextran injection)

IN BRIEF: ACTION AND USES: A single dose of Imferon (iron dextran injection) will measurably begin to raise hemoglobin and a complete course of therapy will effectively rebuild iron reserves. The drug is indicated only for specifically-diagnosed cases of iron deficiency anemia and then only when oral administration of iron is ineffective or impractical. Such iron deficiency may include: patients in the last trimester of pregnancy; patients with gastrointestinal disease or those recovering from gastrointestinal surgery; patients with chronic bleeding with continual and extensive iron losses not rapidly replenishable with oral iron; patients intolerant of blood transfusion as a source of iron; infants with hypochromic anemia; patients who cannot be relied upon to take oral iron.

COMPOSITION: Imferon (iron dextran injection) is a well-tolerated solution of iron dextran complex providing an equivalent of 50 mg. in each cc. The solution contains 0.9% sodium chloride and has a pH of 5.2-6.0. The 10 cc. vial contains 0.5% phenol as a preservative.

ADMINISTRATION AND DOSAGE: Dosage, based upon body weight and Gm. Hb/100 cc. of blood, ranges from 0.5 cc. in infants to 5.0 cc. in adults, daily, every other day, or weekly. Initial test doses are advisable. The total iron requirement for the individual patient is readily obtainable from the dosage chart in the package insert. Deep intramuscular injection in the upper outer quadrant of the buttock, using a Z-track technique, (with displacement of the skin laterally prior to injection), insures absorption and will help avoid staining of the skin. A 2-inch needle is recommended for the adult of average size.

SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylatoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

CONTRAINDICATIONS: Imferon (iron dextran injection) is contraindicated in patients sensitive to iron dextran complex. Since its use is intended for the treatment of iron deficiency anemia only it is contraindicated in other anemias.

CARCINOGENICITY POTENTIAL: Using relatively massive doses, Imferon (iron dextran injection) has been shown to produce sarcoma in rats, mice and rabbits and possibly in hamsters, but not in guinea pigs. The risk of carcinogenesis, if any in man, following recommended therapy with Imferon (iron dextran injection) appears to be extremely small.

SUPPLIED: 2 cc. ampuls, boxes of 10; 5 cc. ampuls, boxes of 4; 10 cc. multiple dose vials.

LAKESIDE LABORATORIES, INC., Milwaukee, Wisconsin 53201

LAKESIDE | PRODUCTS FOR PATIENTS YOU SEE EVERY DAY

BRING IT DOWN
AND
KEEP IT DOWN

Metatensin lowers blood pressure and keeps it low—effectively and economically. It combines reserpine with trichlormethiazide which affords more potent saluresis with less loss of potassium than from earlier thiazides. Reserpine contributes antihypertensive effect by relieving anxiety and tension. Metatensin is well-tolerated over long periods; with its effectiveness and economy it assures antihypertensive therapy you and your patients can stay with.

METATENSIN®

Each scored tablet contains:
METAHYDRIN® (trichlormethiazide)
2 mg. or 4 mg. and
Reserpine 0.1 mg.

Usual adult dose: One tablet twice daily. **Precautions and side effects:** Patients with hepatic cirrhosis or diarrheal syndromes, or under therapy with digitalis, ACTH, or potassium-losing steroids, should be observed for signs of hypokalemia. With thiazides, electrolyte depletion, diabetes, gout, granulopenia, nausea, pancreatitis, cholestatic jaundice, flushing, mild muscle cramps, constipation, photosensitivity, acute myopia, perimacular edema, paresthesias, neonatal bone marrow depression in infants of mothers who received thiazides during pregnancy, skin rash or purpura with or without thrombocytopenia, may occur. With reserpine, untoward effects may include depression, peptic ulcer and bronchial asthma. Withdraw medication at least 7 days prior to electroshock therapy, 2 weeks prior to elective surgery.

Contraindications: Complete renal shutdown, rising azotemia or development of hyperkalemia or acidosis in severe renal disease.

Supplied: Metatensin tablets, 2 mg., 4 mg.—bottles of 100 and 1000.

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New drugs take exams, too.

Today, virtually every medical school in the United States cooperates with pharmaceutical manufacturers in the clinical evaluation of new and promising drugs. Just as you might find it significantly more difficult to practice medicine without the useful new compounds made available through original pharmaceutical research in the past twenty years—prescription-drug manufacturers would find it equally difficult to obtain extensive, long-term, dependable evaluations of new therapeutic agents without the close cooperation of medical staffs and clinical facilities of medical schools and teaching hospitals. Such cooperation leads toward more effective care of more patients—the common goal of medical and pharmaceutical research—toward reduction in the cost of disease, toward increase in useful longevity.

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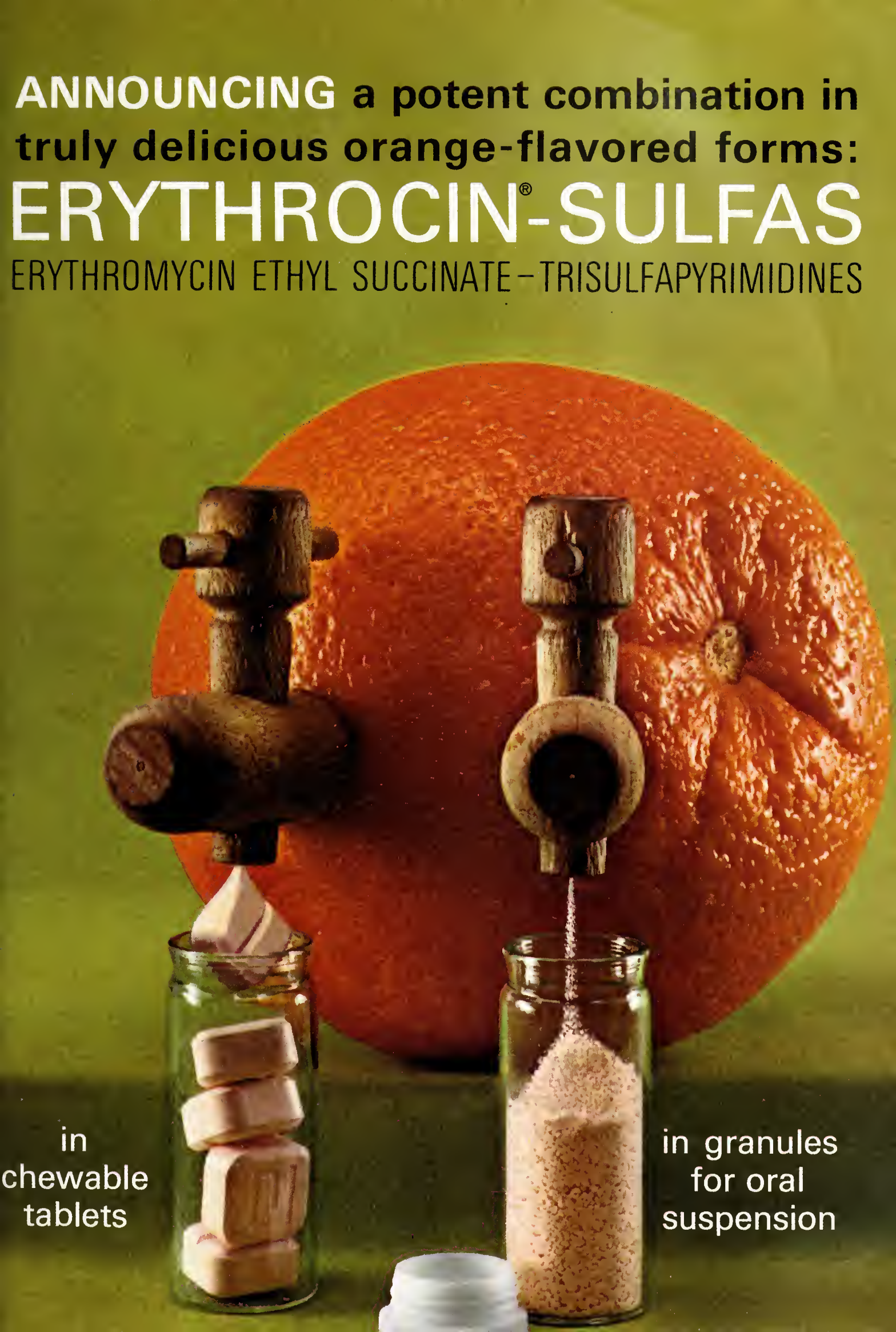

When combination antibiotic therapy is indicated...

CONSIDER: an exceptionally high cure rate in susceptible infections

The rationale: When combined, Erythrocin and the trisulfapyrimidines (triple sulfas) are indicated in infections that are more susceptible to the combination than to either agent alone. Such conditions are usually found in urinary, lower respiratory tract and chronic ear conditions.

The results: Clinical studies involving 142 young patients showed *an overall cure rate of 96.5%*. Side effects were experienced by only four of the patients.

The acceptance: The majority of the 142 patients studied expressed a definite liking for the products. *There were only two refusals.* An independent taste-test with 50 healthy children further substantiated the excellent acceptability of the orange-flavored forms.

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ERYTHROCIN®-SULFAS

ERYTHROMYCIN ETHYL SUCCINATE–TRISULFAPYRIMIDINES

In Chewable Tablets
In Granules for Oral Suspension

ERYTHROCIN®-SULFAS
Brief Summary

Indications: Use Erythrocin-Sulfas in infections more susceptible to the combination than to either agent alone. These are usually found in urinary, lower respiratory tract, and chronic ear infections.

Contraindications: Known sensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, premature or new born infants.

Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

If a reaction or overgrowth of nonsusceptible organisms occurs, withdraw the drug.

Supplied: The Granules for Oral Suspension come in bottles of 60 ml. and 150 ml. The Chewable tablets are in bottles of 50. Each 5-ml. teaspoonful of reconstituted Granules or each Chewable tablet provides erythromycin ethyl succinate equivalent to 125 mg. of erythromycin activity and 167 mg. each of sulfadiazine, sulfamerazine and sulfamethazine. 603303

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Bamadex® Sequels®

Contraindications: In hyperexcitability and in agitated prepsychotic states. Previous allergic or idiosyncratic reactions.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised; especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of pre-existing symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dose—operation of motor vehicles, machinery or other activity requiring alertness should be avoided. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.

First aid for a button popper

Second aid for a button popper

Bamadex® Sequels®

d-amphetamine sulfate (15 mg.) and meprobamate (300 mg.)
Sustained Release Capsules

By providing combined anorexigenic-tranquilizing action, BAMADEX SEQUELS Capsules help your nonshrinking patients to establish new patterns of eating less. The amphetamine component suppresses the appetite, while the meprobamate helps allay nervousness and tension. And for most patients, the *sustained* release of the active ingredients makes possible convenient one-capsule-a-day dosage.

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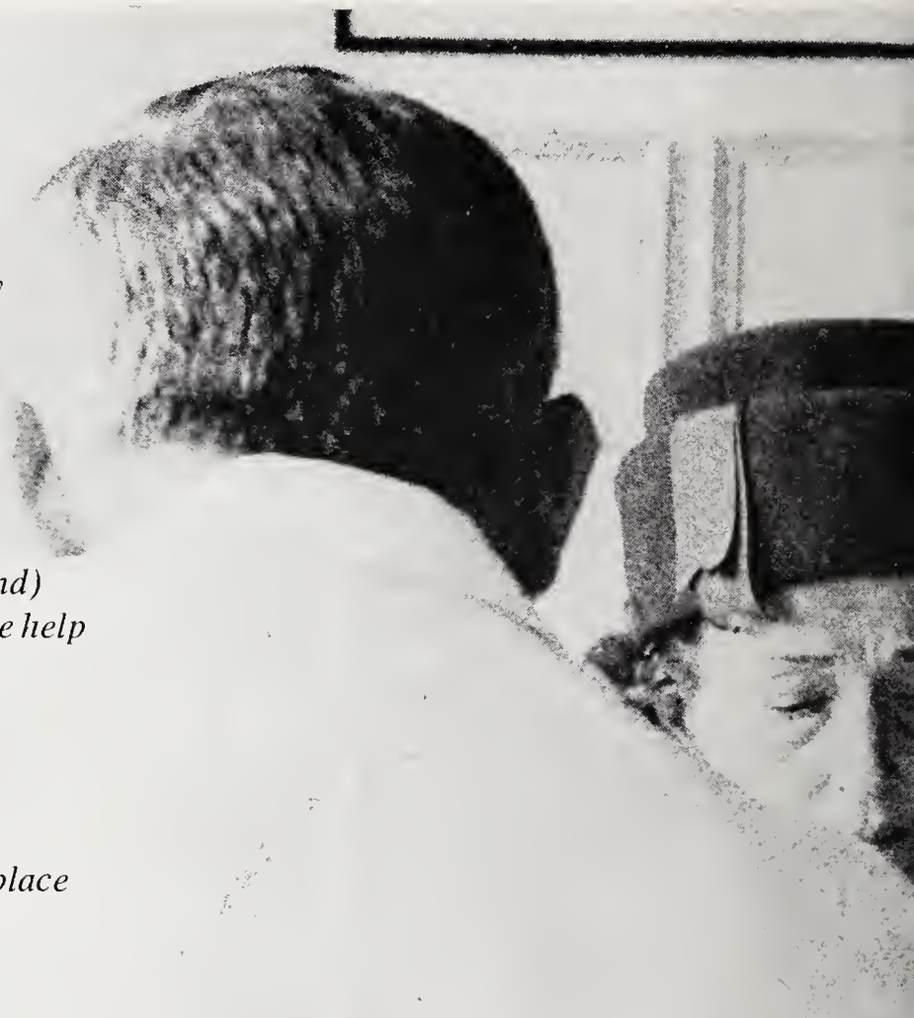
The Mediatric Age:

Unfortunately, there is no cure. But there are, largely through your own interest and direction, ways to help them back to a more active and useful life. There are medicines, too, designed to help. One such has proved useful in clinical practice:



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McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

"Mediatric (steroid-nutritional compound) capsules, one a day, seem to give definite help to debilitated patients."

Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

"Nutritional and hormone bolstering of function in the aged may have a useful place in geriatrics."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

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Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"Intensive nutritional therapy is necessary, especially in elderly people, to correct dietary deficiencies created by large losses of protein, vitamins and other nutrients."

Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.

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Designed for the "metabolically spent"

Nutritional reinforcement for those who can't – or won't – eat properly...balanced amounts of estrogen and androgen to counteract declining gonadal hormone secretion and its sequelae of premature degenerative changes...mild antidepressant for a gentle "mood" uplift...

The estrogen component in MEDIATRIC is PREMARIN® (conjugated estrogens—equine), the natural estrogen most widely prescribed for its superior physiologic and metabolic benefits.

MEDIATRIC also provides *nutritional reinforcement—blood-building factors and vitamin supplementation.* It contributes a *gentle "mood" uplift* through methamphetamine HCl.

Three different dosage forms—Liquid, Tablets, and Capsules—offer convenience and variety.

MEDIATRIC Liquid

Each 15 cc. (3 teaspoonfuls) contains:

*Conjugated estrogens—equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Thiamine HCl	5.0 mg.
Cyanocobalamin	1.5 mcg.
Methamphetamine HCl	1.0 mg.

Contains 15% alcohol

MEDIATRIC Tablets and Capsules

Each MEDIATRIC Tablet or Capsule contains:

*Conjugated estrogens—equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Ascorbic acid	100.0 mg.
Cyanocobalamin	2.5 mcg.
Intrinsic factor concentrate	8.0 mg.
Thiamine mononitrate	10.0 mg.
Riboflavin	5.0 mg.
Niacinamide	50.0 mg.
Pyridoxine HCl	3.0 mg.
Calc. pantothenate	20.0 mg.
Ferrous sulfate exsic.	30.0 mg.
Methamphetamine HCl	1.0 mg.

*Orally active, water-soluble conjugated estrogens derived from pregnant mares' urine and standardized in terms of the weight of active, water-soluble estrogen content.

MEDIATRIC helps keep the older patient alert and active; helps relieve general malaise, easy fatigability, vague pains in the bones and joints, loss of appetite, and lack of interest usually associated with declining gonadal hormone secretion.

CONTRAINDICATION: Carcinoma of the prostate, due to methyltestosterone component.

WARNING: Some patients with pernicious anemia may not respond to treatment with the Tablets or Capsules, nor is cessation of response predictable. Periodic examinations and laboratory studies of pernicious anemia patients are essential and recommended.

SIDE EFFECTS: In addition to withdrawal bleeding, breast tenderness or hirsutism may occur.

SUGGESTED DOSAGES: *Male and female:* 3 teaspoonfuls of Liquid, 1 Tablet, or 1 Capsule, daily or as required.

In the female: To avoid continuous stimulation of breast and uterus, cyclic therapy is recommended (3 week regimen with 1 week rest period—Withdrawal bleeding may occur during this 1 week rest period).

In the male: A careful check should be made on the status of the prostate gland when therapy is given for protracted intervals.

SUPPLIED: No. 910 – MEDIATRIC Liquid, in bottles of 16 fluidounces and 1 gallon. No. 752 – MEDIATRIC Tablets, in bottles of 100 and 1,000. No. 252 – MEDIATRIC Capsules, in bottles of 30, 100, and 1,000.

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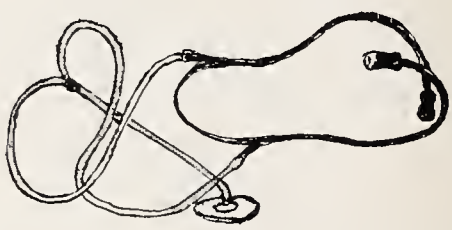

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GENERAL PRACTITIONER NEEDED: The city of Giddings, located on U. S. Highway 290, fifty miles east of Austin, and one hundred twenty miles west of Houston, is in critical need of obtaining the services of another general practitioner. Giddings is a community of 3,000 people, with 8,000 people in the county. Giddings has at present two practicing physicians, who are aware of this advertisement. Assistance will be extended by the Giddings Chamber of Commerce to any doctor who is interested. Please address all replies to Allan I. Schneider, Giddings Chamber of Commerce, Box 87, Giddings, Texas.

PEDIATRICIAN, EENT, ORTHOPEADIST, AND GENERAL PRACTITIONERS urgently needed for 10-doctor clinic, adjacent to fully accredited general hospital. Located in one of the fastest growing clean, industrialized areas in the United States; 15 minutes from metropolitan Dallas and Fort Worth. Pleasant climate year around, abundant water recreational and cultural activities available. Average age of 29 in city of 60,000. Free office rent in clinic. For more information, contact Gary W. Watts, Director of Public Relations, Great Southwest General Hospital, 444 Duncan Perry Road, Grand Prairie, Texas 75050.

PHYSICIANS (GENERAL), with Texas license or eligible for reciprocity, for 2,700 bed modern State Hospital. Near medical center (Dallas). Training affiliation with Medical School. Salary from $13,500 to $16,000. Available after September 1. Apply: Hugh N. Brown, M.D., Terrell State Hospital, Terrell, Texas.

WANTED: GENERAL PRACTITIONER OR INTERNIST to associate with established G.P. Exceptional location. Entirely new offices with complete laboratory and x-ray facilities; excellent hospital facilities within 15 minutes. Will consider guarantee, salary, percentage or renting and sharing offices and equipment to the right individual. Most desirable location in North Dallas. Contact: R. Courtney Suttle, M.D., 6211 West Northwest Highway, Preston Tower, Suite 234, Dallas, Texas.

HELP WANTED: Physicians needed for 900 bed State Mental Hospital with varied and progressive programs, good working conditions. Accredited I.C.A.H. Psychiatrist $19,000, Physicians general $16,000, ECFMG $9,600. Liberal vacation, holiday and sick leave; retirement and hospitalization insurance available. Write: Superintendent, Big Spring State Hospital, Box 231, Big Spring, Texas.

RADIOLOGIST ASSOCIATE WANTED, Delta area of Rio Grande Valley. Combined hospital-office practice. Population 55,000 with trade area of 150,000. Present examinations total 10,000 annually. Board qualification or equal in diagnosis and therapy required. No investment. Part-time or full-time, 35%-45% of net at start. Write to Lee Works, M.D., 105 West Elizabeth, Brownsville, Texas 78520.

WANTED: GENERAL PRACTITIONER AND PEDIATRICIAN to become associated with well established general practitioner in a town of 10,000 with trade territory of 45,000 on Texas gulf coast. There is no pediatrician in this trade territory. Please reply to Box 990, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED by well established Dallas internist: Board certified associate for the practice of cardiology and internal medicine. Opportunities for teaching and research as well as private practice. Salary $20,000 first year. Partnership second year. New office building adjacent to major hospital available February 1, 1966. Please reply to Leonard J. Flohr, M.D., 611 Doctors Building, 3707 Gaston Avenue, Dallas, Texas 75246.

IMMEDIATE OPENINGS in student health, male or female. Salary $14,000 per annum. 74-bed modern hospital and clinic on campus of North Texas State University, Denton, Texas. Prefer experienced general practitioners. Texas license required. Housing available. Usual school holidays, teacher retirement and other fringe benefits. President, J. C. Matthews, North Texas State University, Denton, Texas.

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INTERNAL MEDICINE: (g) Assn 14 internists; must have sub-spec; exc med schl connectns; sal, prtnrshp; univ city. (h) Jn well-qualif internist; reg emphasis, clin invest or endo; top sal; med cntr. (i) 7-mon grp; dipl in dept; modern cl bldg; early prtnr nettg area $35,000; coll twn.

OALR: (j) Oto; assn w/12 dipls; 1 oto & 1 oph; area $24,000 1st then prtnrshp; lovely twn. (k) Ophth; 13-mon grp; own cl bldg; profit basis, prtnr; resort area.

ORTHOPEDICS: (l) Jn 38-mon grp, 20 Cert; sol open, prtnr 3rd yr; med cntr. (m) Qual to hd dept; 9 spec estab'd '46; own hsp; oppor $30,000; univ cntr. (n) Assn 14 spec; dipl in dept; sal open; med schl city.

PSYCHIATRY: Elig for med dir of well-est, compreh rehob cntr; complete depts; 55-bd JCAH; sal open; farm area.

PSYCHIATRY: Jn staff, 2,376-bd long term, mental hsp; oppor od-vance to Clin Dir; area $19,000; county seat.

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PHYSICIAN NEEDED IN WEST TEXAS TOWN. General practitioner needed in Bovina, Texas. Financial assistance available to assist in building or to build a clinic. A town of 2,000 population, only 12 miles from a small hospital, 20 miles from a one hundred-bed hospital. If interested, contact Mr. Bob Estes, First State Bank, Bovina, Texas.

GENERAL SURGEON AND GENERAL PRACTITIONER to work with two general practitioners in 42 bed hospital-clinic. Present surgeon is retiring in 1967 and will leave well established practice. No investment required. Write or call M. R. Snodgrass, M.D., Box 248, Crosbytown, Texas. Phone 3761.

STUDENT HEALTH SERVICE PHYSICIAN, Texas license, to join staff of three full-time and five part-time physicians. Limited call duty and ample vacation time. Contact K. L. Nelson, M.D., Director of Student Health Services, Texas A&M University, College Station, Texas.

PSYCHIATRISTS, with Texas license or eligible for reciprocity, for 2,700 bed modern State Hospital. Near medical center (Dallas). Training affiliation with Medical School. Salary from $18,000 to $19,000. Available after September 1. Apply: Hugh N. Brown, M.D., Terrell State Hospital, Terrell, Texas.

NEED URGENTLY: This clinic and hospital can offer excellent opportunities for an internist, a general practitioner and an OB-GYN specialist in group practice with excellent remuneration and no professional overhead. Clinic located in rapidly growing West Texas city of approximately 40,000 people. Apply to Charles A. Weeg, Administrator, Hall-Bennett Memorial Hospital, Big Spring, Texas.

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GENERAL PRACTITIONER NEEDED. Population 1500, modern 50-bed hospital within eight blocks of present office. Office air-conditioned. One other doctor in town. Financial backing. 20,000 trade area. Contact Lester Drug Company, Lewisville, Arkansas.

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HOUSE PHYSICIAN: House physician to cover the hospital charity services and hospital emergency room. Each physician will be responsible for covering the emergency room one day a week. While on night emergency room call he will be responsible for care of in-patients. There will be two days off per week. Emergency room service requires remaining at the hospital. Texas license is required. Salary: $16,000 per annum, plus additional if specialty. Contact Mr. E. F. Glaze, Administrator, Memorial Medical Center, P. O. Box 5280, Corpus Christi, Texas, 78405. Telephone: Area Code 512—TU 4-4511.

GENERAL PRACTITIONER WANTED for private clinic and hospital in Gilmer, county seat of Upshur County in East Texas. Closed staff, accredited hospital, approved for Medicare. Good guaranteed income for the right man. For more information, contact M. S. Ragland, M.D., or J. R. Rickels, Ragland-Fenlaw Clinic, P. O. Box 310, Gilmer, Texas.

TWO INTERNISTS, board certified or eligible, and one pulmonary disease physician, board eligibility desirable, needed by 422 bed GM&S Hospital. Salary based on qualifications; citizenship required. Non-discrimination in employment. Apply: Director, VA Hospital, Kerrville, Texas 78028.

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GENERAL PRACTITIONER with 20 years ethical, successful practice in Michigan desires industrial, insurance, institutional or other position with regular hours in Texas. Medical education kept current. Member AAGP. Examiner for several life insurance companies. License by reciprocity. Would welcome interview and conditional contract. Contact Ira Wiggins, M.D., 205 East St., Jonesville, Michigan.

WANT TO RELOCATE in or near a city, preferably one having a medical center. My practice is restricted to general medicine (no OB and no surgery). Group practice preferred. Please reply to Box 999, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

ADMINISTRATOR, available for private hospital in large Texas town or city. Would consider unit planned or under construction. Can offer outstanding background for lucrative position. Write Administrator, Box 997, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

SURGICAL PRECEPTORSHIP desired. Will complete three year program in surgical residency, December 31, 1966. North or northwest Texas areas preferred. Military obligation completed. Please reply to Box 994, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

PART TIME OR LOCUM TENENS. Physician, Texas license, 42 years, experienced, in good health desires to work part-time (evenings) as assistant, associate, or locum tenens, for salary in general practice, in Houston, preferably neighborhood medical center. Please reply to Box 995, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

HOSPITAL OR GROUP DESIRED: General practitioner, USA born and trained. Rotating internship 15 months. Have 20 years of successful practice in Texas. Member of Harris County Medical Society. Diplomate of the National Board of Medical Examiners and member of American Academy of General Practice. Desires work with hospital or group. Would welcome interview. Bilingual. Please reply to Box 993, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED—LOCUM TENENS, Board certified internist desires locum tenens in internal medicine or medically oriented general practice. One to three weeks. Texas license. Please send complete details, regarding location, type practice, dates desired, and financial arrangements. Reply to Box 998, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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IMMEDIATELY AVAILABLE: Fully-equipped clinic of recently deceased physician and surgeon. Patients records and some personnel available. Doctor's widow will introduce. Reply to Lowry Clinic, P. O. Box 2005, Laredo, Texas.

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FOR LEASE: A partially equipped upstairs clinic in midst of down-town Conroe with offices for three doctors. Good opportunity for a pediatrician or a dermatologist. If interested contact Mrs. W. M. Holland, 1303 N. Thompson, Conroe, Texas. Phone PL 6-2662.

NORTH EAST DALLAS. New Medical Building just completed, Ferndale and Northwest Hwy. For M.D.'s and D.S.'s only. 12,000 feet of space. Will decorate to suit. In area of 45,000 people with few physicians close. Leasing now. Call DI 8-2320 or RI 2-8265.

FOR LEASE IN HOUSTON. 784 to 1500 square feet of office space in active medical and dental building in rapidly growing Southwest (Westbury) area. Ample parking. Pharmacy and laboratory in building. Write or call Barry Bowser, M.D., P. O. Box 35262, Houston, Texas. PA 3-9511.

FOR SALE: Medical office building nearly new, located across street from Kleberg County Hospital. Large, paved parking area. Ample rooms, well equipped for two doctors. The town is delightful, and the people warm and friendly. The trade area is large and opportunities for success unlimited. Building, lots, office equipment and furnishings very reasonably priced. Your interest will be appreciated. Please write Mrs. Walter E. Fosberg, 222 Otis, Kingsville, Texas, or call LY 2-5668 or LY 2-2606.

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FOR SALE: Fully equipped general practice in the Central Texas area. In 1965 grossed $40,000. Lease on building with or without equipment. Also home available. Write Box 981, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

MEDICAL OFFICE FOR LEASE: Bellaire Professional Center, 30c per square foot, 5200 block of Cedar, Bellaire, Texas. Three treatment rooms, suite, carpeted private office, hi-fi speaker, front and rear private entrances, adequate parking. Near Bellaire General and Southwest Memorial Hospitals. Attractive lease arrangements. Call MO 7-1111, or write 5200 Cedar, Bellaire, Texas.

FOR SALE, MEDICAL-DENTAL CLINIC. Modern brick approximately 2,000 square feet. Central Texas, population area approximately 150,000. Air conditioned, partitioned, ample parking. Will finance, reasonably priced. Now active. Write Box 985, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

FOR SALE—29 bed hospital in small West Texas town. Well equipped operating, delivery and emergency rooms, laboratory and x-ray. Clinic with seven treatment rooms, office and waiting room in same building. Large parking lot. Write Box 982, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

HOUSTON: Texas Medical Center. Office suite for lease. Thirty three cents square foot. Two thousand square feet or less. Free parking at door. For floor plan and picture of building, write John Hannah, P. O. Box 20032, Houston, Texas 77025 or call MO 8-0471.

FOR SALE—OFFICE EQUIPMENT, examining tables, cabinets, etc. All equipment one year old. Excellent opportunity for someone entering practice. Please reply to Kenneth R. Bennett, M.D., 2516 North Street, Nacogdoches, Texas 75961.

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FOR SALE: Your greatest opportunity. Two story clinic, brick building, completely equipped and furnished. X-ray, Ekg, etc. Year round heating and air condition. Put your jacket on and see 30 to 60 patients daily. Collections are good. $250,000 value for $75,000. 12 lower rooms, plus 3 bath rooms and 6 upper rooms plus 2 bath rooms. Same location 35 years. Desire immediate reply. R. J. Van Sickle, M.D., 100 East Methvin Street, Longview, Texas, PLaza 8-4416 and PLaza 3-4177.

IMMEDIATE DALLAS LEASE. Fully equipped clinic of recently deceased urologist and surgeon with or without furniture and equipment. Choice Dallas Oak Lawn location convenient to town, transportation and hospitals. Access to established practice and patients records. Apply to Box 100, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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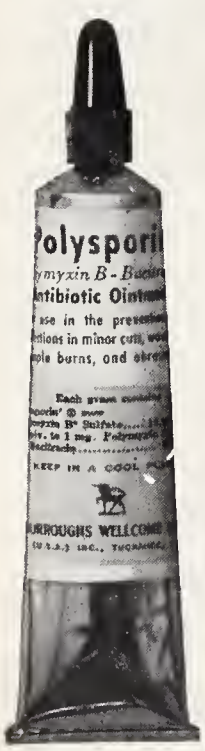

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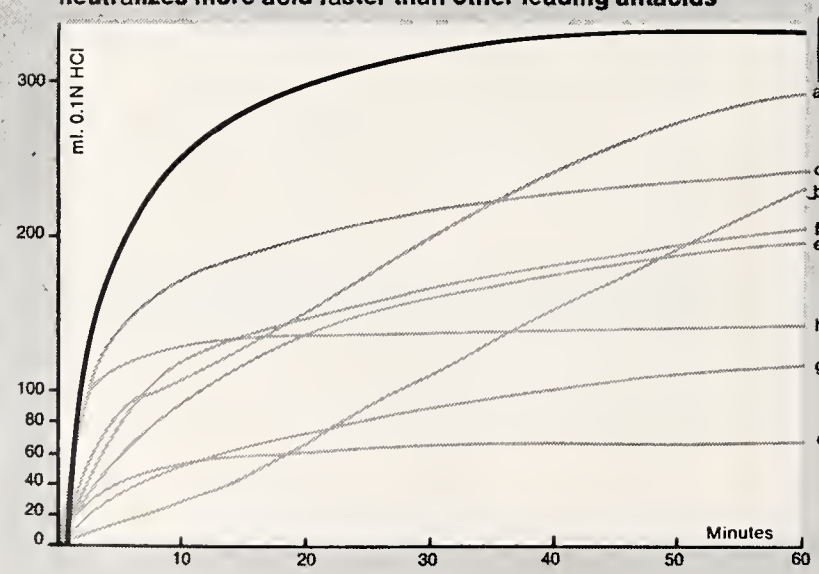

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Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

Indications: DIUTENSEN-R may be employed in all grades of essential hypertension. **Dosages:** Usual dose is 1 tablet twice daily, at morning and evening meals. However, adjustment of dosage to suit individual circumstances may be required. Please refer to package insert for full particulars. **Side effects and precautions:** The side effects observed with patients on DIUTENSEN-R have been of a mild and nonlimiting nature. These include occasional urinary frequency, nocturia, nasal congestion, muscle cramps, skin rash, joint pains due to gout symptoms and nausea and dizziness which have been reported for the individual components. Most of these symptoms disappear while the drug is continued at the same or lower dosage level. The concomitant use of digitalis and DIUTENSEN-R may increase the possibility of digitalis-like intoxication. If there is evidence of myocardial irritability (extrasystoles, bigeminy or AV block), dosage of DIUTENSEN-R should be reduced or discontinued. Nocturia in patients with marginal cardiac status and salt and fluid retention can be effectively controlled by limiting the time of administration to early afternoon. DIUTENSEN-R should not be used in patients with a known intolerance to reserpine. Package inserts furnish a complete summary of recommended cautions related to each of the ingredients of DIUTENSEN-R.

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The "Socio-geographic" mystery

Why is one man's gastric ulcer another man's duodenal?

Geographic variation in the *incidence* of peptic ulcer is a familiar fact. But the proclivity of certain *kinds* of ulcer for certain geographic areas is a recently recognized phenomenon.[1,2]

For example, in one particular Norwegian fishing village there is a tendency for patients to develop a gastric ulcer; anywhere else in Norway, ulcers are usually duodenal. Peruvians high in the Andes have more gastric ulcers than their compatriots in the lowlands. Why? Nobody knows.

Social variations, too. Even in the same geographic areas there are interesting variations. An Englishman's ulcer depends on his social standing—professional men suffer with duodenal ulcers, while workingmen have more of the gastric variety. In southern India the pattern is reversed. Here, duodenal ulcers are common among laborers and agricultural workers and rare among the upper classes.

Investigators are exploring every possible theoretical avenue in their search for the cause of peptic ulcer. Of all the factors implicated in ulcerogenesis, the one that is generally acknowledged to be of primary importance is hypersecretion of gastric acid.[3-8] Or, as one author states it: "The medical management of peptic ulcer pharmacologically is, in the final analysis, concerned largely with the effective inhibition of peptic activity."[3]

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References: **1.** Jones, F. A., and Gummer, J. W. P.: Clinical gastroenterology, Springfield, Ill., Charles C Thomas, 1960, pp. 322-3. **2.** Bockus, H. L.: Gastroenterology, 2nd ed., vol. I, Philadelphia, Saunders, 1963, p. 468. **3.** Sun, D. C. H.: Ann NY Acad Sci 99:153 (Feb. 28) 1962. **4.** Moore, V. A.: Postgrad Med 38:216 (Sept.) 1965. **5.** Dragstedt, L. R., Woodward, E. R., Storer, E. H., Oberhelman, H. A., Jr., and Smith, C. A.: Ann Surg 132:626 (Oct.) 1950. **6.** Posey, E. L., Jr., Smith, P., Turner, C., and Aldridge, J.: Amer J Dig Dis 10:399 (May) 1965. **7.** Lamphier, T. A., Siegel, L., and Goldberg, R. I.: Amer J Gastroent 37:551 (May) 1962. **8.** Kasich, A. M., and Fein, H. D.: Ibid 39:61 (Jan.) 1963. **9.** Epstein, J. H.: Ibid 37:295 (Mar.) 1962. **10.** Moeller, H. C.: Ann NY Acad Sci 99:158 (Feb. 28) 1962. **11.** Slanger, A.: J New Drugs 2:215 (Jul.-Aug.) 1962. **12.** Barman, M. L., and Larson, R. K.: Amer J Med Sci 246:325 (Sept.) 1963. **13.** Shutkin, M. W.: Amer J Gastroent 38:682 (Dec.) 1962. **14.** Fleshler, B.: J New Drugs 2:211 (Jul.-Aug.) 1962.

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Precautions: In elderly and debilitated and in children over five, limit dosage to smallest effective amount, increasing gradually as needed and tolerated. In general, concomitant use with other psychotropics is not recommended. Paradoxical reactions have been reported in psychiatric patients and hyperactive aggressive children. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Observe usual precautions in presence of impaired renal or hepatic function, impending depression and suicidal tendencies.

Adverse reactions: Drowsiness, ataxia and confusion may occur, especially in elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. Syncope occurs rarely. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis, jaundice and hepatic dysfunction) may develop occasionally, making periodic blood counts and liver-function tests advisable during protracted therapy. Individual maintenance dosages should be determined.

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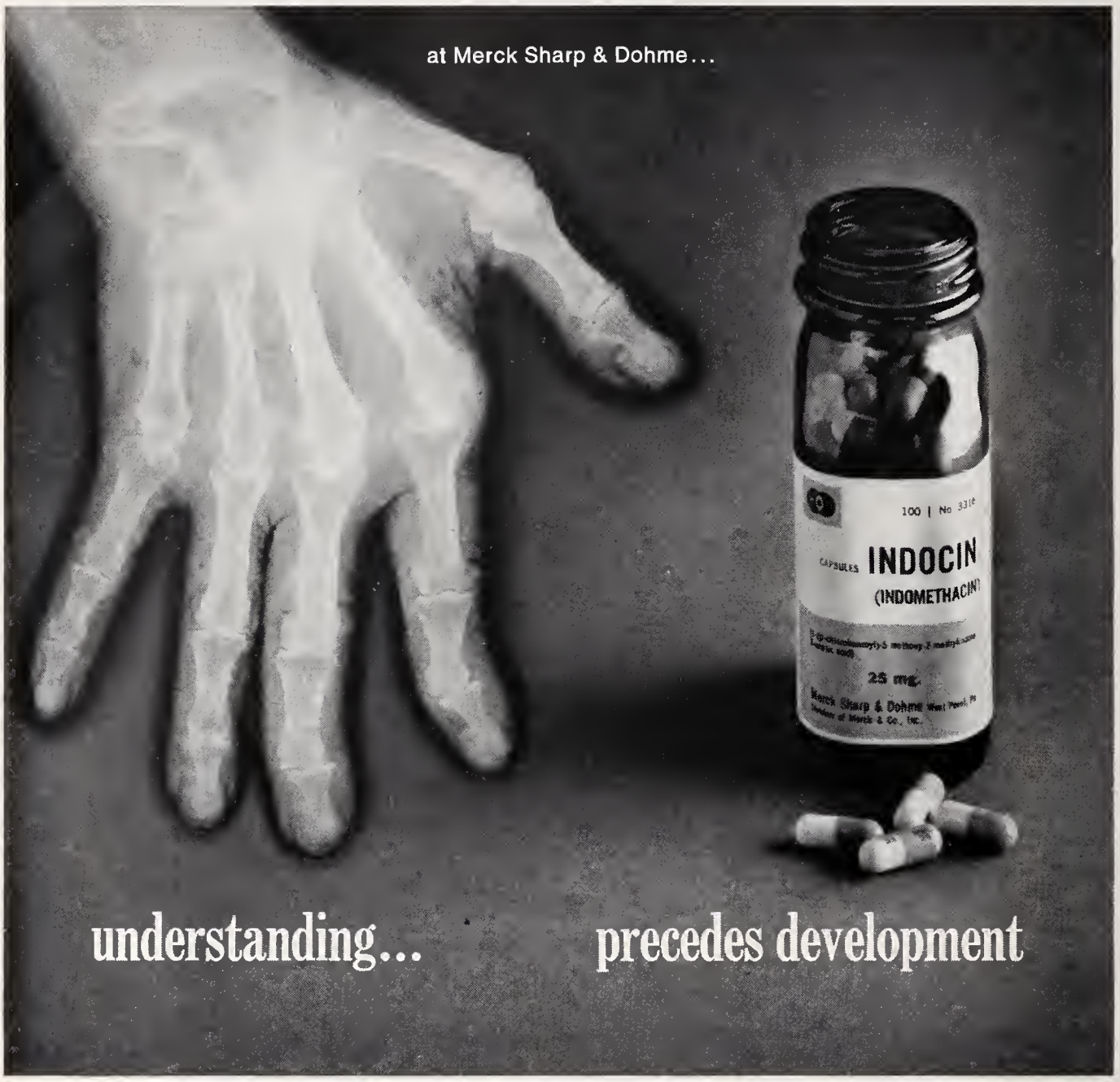

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DIVERTICULITIS
SPASTIC URETERITIS
BLADDER SPASM

are relieved by direct musculotropic action with.....

Trocinate®

BRAND THIPHENAMIL HCl

Available in 100 milligram pink sugar-coated tablets.

The high therapeutic index permits dosage sufficient to relieve spasm promptly.

Administer 4 tablets every 4 hours until relief is constant, then adjust maintenance dosage.

Trocinate *BRAND* THIPHENAMIL HCl

Directly relaxes smooth muscle spasm
Combats hypermotility
Non-mydriatic, may be used in glaucoma

Sixteen years of clinical use, with absence of untoward effects, has established the safety and effectiveness of Trocinate.

Trocinate is metabolized in the body and completely eliminated, which is a safety factor. Dosage must be sufficient to maintain the therapeutic blood level.

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THE BROWN SCHOOLS for mentally retarded and emotionally disturbed persons from infancy to maturity.

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the sedentary life is often the seat of low back pain

The human spine is not engineered for prolonged sitting at desks, pianos, typewriters and drafting boards. The stresses set up by the heavy, forward-tilted head and trunk, balanced precariously on an insufficient base, result in strain of the dorsal musculature, particularly at the low lumbar level.

The unusual muscle-relaxant and analgesic properties of 'Soma' make it especially useful in the treatment of low back sprains and strains. 'Soma' is widely prescribed ☐ **to relieve pain** ☐ **to relax muscles** ☐ **to restore mobility.**

Indications: 'Soma' is useful for management of muscle spasm, pain, and stiffness in a variety of inflammatory, traumatic, and degenerative musculoskeletal conditions. It also may act to normalize motor activity in certain neurologic disturbances.

Contraindications: Allergic or idiosyncratic reactions to carisoprodol.

Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

One instance each of pancytopenia and leukopenia, occurring when carisoprodol was administered with other drugs, has been reported, as has an instance of fixed drug eruption with carisoprodol and subsequent cross reaction to meprobamate. Rare allergic reactions, usually mild, have included one case each of anaphylactoid reaction with mild shock and angioneurotic edema with respiratory difficulty, both reversed with appropriate therapy. In cases of allergic or hypersensitivity reactions, carisoprodol should be discontinued and appropriate therapy initiated. Suicidal attempts may produce coma and/or mild shock and respiratory depression.

Dosage: Usual adult dose is one 350 mg. tablet three times daily and at bedtime.

Supplied: Two Strengths: 350 mg. white tablets and 250 mg. orange, two-piece capsules.

Before prescribing, consult package circular.

for the relief of low back sprains and strains

SOMA®
(CARISOPRODOL)

Wallace Laboratories, Cranbury, N.J.
26S01J

Butazolidin® alka

phenylbutazone, 100 mg.
dried aluminum hydroxide gel, 100 mg.
magnesium trisilicate, 150 mg.
homatropine methylbromide, 1.25 mg.

Usually works within 3 to 4 days in osteoarthritis

The trial period need not exceed 1 week. In contrast, the recommended trial period for indomethacin is *at least 1 month.*

That's why it's logical to start therapy with Butazolidin alka—you'll know quickly whether or not it works. And usually, it will.

A large number of investigators have reported major improvement in about 75% of cases. Some patients have gone into remission. Relief of stiffness and pain may be followed quickly by improved function and resolution of other signs of inflammation. And Butazolidin alka is well tolerated, especially since it contains antacids and an antispasmodic to minimize gastric upset.

Contraindications
Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. Because of the increased possibility of toxic reactions, the drug should be used with greater care in the elderly and should not be given when the patient is senile or when other potent chemotherapeutic agents are given concurrently. Large doses of Butazolidin alka are contraindicated in patients with glaucoma.

Warning
If coumarin-type anticoagulants are given simultaneously, the physician should watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Patients receiving such concomitant therapy should be carefully observed for this effect.

Use with caution in the first trimester of pregnancy.

Precautions
Before prescribing, the physician should obtain a detailed history and perform a complete physical and laboratory examination, including a blood count. The patient should be kept under close supervision and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Regular blood counts should be made to guard against blood dyscrasias.

Adverse Reactions
The most common adverse reactions are nausea, edema and drug rash. Moderately lowered red cell count may sometimes occur due to hemodilution. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vertigo or languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia are also possible side effects.

Confusional states, hyperglycemia, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hepatitis, jaundice and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently.

Dosage
The initial daily dosage in adults is 300-600 mg. daily in divided doses. In most instances, 400 mg. daily is sufficient. When improvement occurs, dosage should be decreased to the minimum effective level: this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

For complete details, please refer to full prescribing information.
6509-V(B)

Also available: Butazolidin®, phenylbutazone Tablets of 100 mg.

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Ardsley, New York BU-3804R

Geigy

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ERYTHROCIN®-SULFAS

ERYTHROMYCIN ETHYL SUCCINATE–TRISULFAPYRIMIDINES

in chewable tablets

in granules for oral suspension

When combination antibiotic therapy is indicated...

CONSIDER: an exceptionally high cure rate in susceptible infections

The rationale: When combined, Erythrocin and the trisulfapyrimidines (triple sulfas) are indicated in infections that are more susceptible to the combination than to either agent alone. Such conditions are usually found in urinary, lower respiratory tract and chronic ear conditions.

The results: Clinical studies involving 142 young patients showed *an overall cure rate of 96.5%*. Side effects were experienced by only four of the patients.

The acceptance: The majority of the 142 patients studied expressed a definite liking for the products. *There were only two refusals.* An independent taste-test with 50 healthy children further substantiated the excellent acceptability of the orange-flavored forms.

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ERYTHROCIN®-SULFAS

ERYTHROMYCIN ETHYL SUCCINATE–TRISULFAPYRIMIDINES

In Chewable Tablets
In Granules for Oral Suspension

ERYTHROCIN®-SULFAS

Brief Summary

Indications: Use Erythrocin-Sulfas in infections more susceptible to the combination than to either agent alone. These are usually found in urinary, lower respiratory tract, and chronic ear infections.

Contraindications: Known sensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, premature or new born infants.

Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

If a reaction or overgrowth of nonsusceptible organisms occurs, withdraw the drug.

Supplied: The Granules for Oral Suspension come in bottles of 60 ml. and 150 ml. The Chewable tablets are in bottles of 50. Each 5-ml. teaspoonful of reconstituted Granules or each Chewable tablet provides erythromycin ethyl succinate equivalent to 125 mg. of erythromycin activity and 167 mg. each of sulfadiazine, sulfamerazine and sulfamethazine. 603303

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Norinyl® tablets

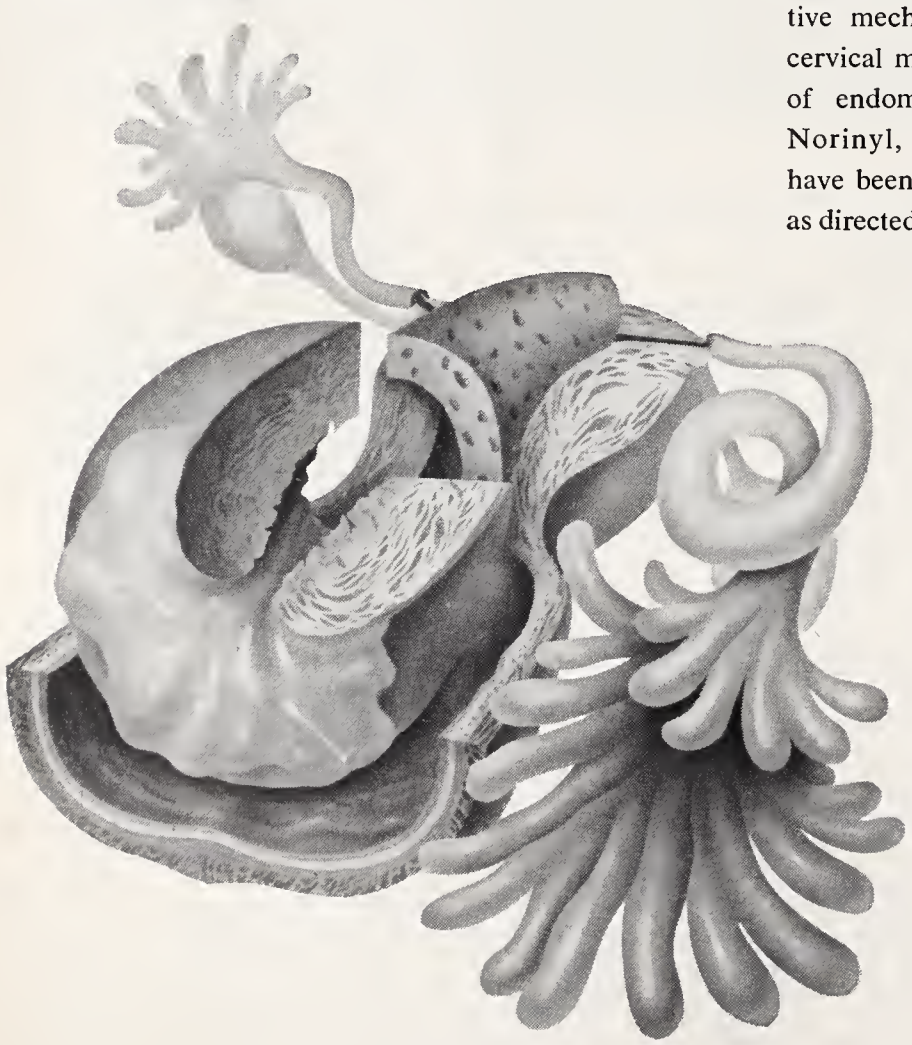
(norethindrone 2 mg. c mestranol 0.1 mg.)

for multiple contraceptive action that has produced a record of unexcelled effectiveness

inhibition of ovulation by means of 2 time-proved hormonal agents

production of a cervical mucus hostile to sperm motility and vitality

creation of an endometrium unreceptive to egg implantation



no unplanned pregnancies

Norinyl provides multiple action for maximum assurance of success. It does not depend on ovulation inhibition alone for contraceptive effectiveness. The mechanism of action of combined hormonal therapy results in ovulation inhibition reinforced by other protective mechanisms, including a hostile cervical mucus[1-13] and an acceleration of endometrial changes.[1-3,7-16] With Norinyl, no unplanned pregnancies have been reported to date when used as directed.

plus important supportive benefits that help her through those critical early months of oral contraception

low incidence of side effects

Low incidence of BTB and spotting, nausea and amenorrhea tends to minimize side effect problems and increases patient cooperation.

no confusion about dosage

An unbreakable "confusionproof" package makes it easy to adhere to prescribed dosage schedule: individually sealed tablets numbered from 1 through 20 *plus* monthly calendar record enables patient to double-check dosage intake by day and corresponding tablet number.

Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb. 29) 1964. 2. Brvans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

norethindrone—an original steroid from
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LABORATORIES INC., PALO ALTO, CALIF

Norinyl® tablets

(norethindrone 2 mg. ē mestranol 0.1 mg.)

for multiple contraceptive action

If you can hang on for a few minutes, Doctor,
I'm sure I'll start coughing again.

Some patients don't realize there's more to a cough than meets the ear.
If it's the useless, exhausting type of cough that often accompanies respiratory infection or allergy, you can provide prompt relief with Novahistine DH. Its decongestant-antitussive action controls frequency and intensity of cough spasms without abolishing cough reflex. And the fresh, grape flavor of Novahistine DH appeals to children and adults alike.
When your diagnosis is bronchitis, complicated by thick tenacious exudates, Novahistine Expectorant is particularly useful. It not only provides decongestive action and controls the cough, but also encourages expectoration, thus easing bronchial constriction and obstruction.
Use with caution in patients with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Ambulatory patients should be advised that drowsiness may result. Continuous dosage over an extended period is contraindicated since codeine phosphate may cause addiction.
Each 5 ml. teaspoonful of Novahistine DH contains codeine phosphate, 10 mg. (Warning: may be habit forming); phenylephrine hydrochloride, 10 mg.; chlorpheniramine maleate, 2 mg.; chloroform (approx.), 13.5 mg.; l-menthol, 1 mg. (Alcohol 5%). Each 5 ml. of Novahistine Expectorant contains the above ingredients and, in addition, glyceryl guaiacolate, 100 mg.

NOVAHISTINE® DH
NOVAHISTINE® EXPECTORANT

DOW **PITMAN-MOORE** Division of The Dow Chemical Company, Indianapolis

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SOLVES THE PROBLEM

for the infant who requires a milk-free diet!

- Soyalac satisfies! Baby is happy, mother is grateful, doctor is gratified.
- The nut-like taste is pleasing. Infants readily accept this hypoallergenic formula that is completely fibre-free. An exclusive process results in a consistency much like milk.
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- Excellent for regular infant feeding, too—and for growing children and adults.

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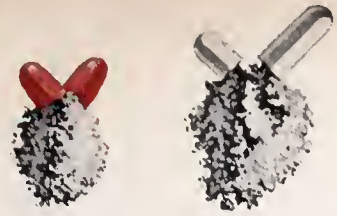

greater potency

lower mg intake per day

600 mg versus 1,000 mg

in G.U. infections

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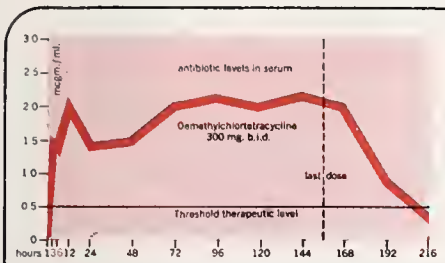
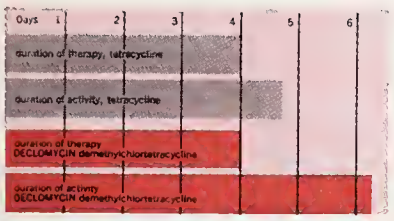

mcgm./ml.
antibiotic levels in serum
Demethylchlortetracycline 300 mg. b.i.d.
last dose
Threshold therapeutic level
hours 1 3 6 12 24 48 72 96 120 144 168 192 216

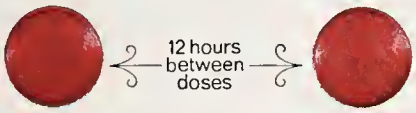
high activity

From Sweeney, W. M., Dornbush, A. C., and Hardy, S. M., Amer. J. Med. Sci. 243:296 (Mar.) 1962


Days 1 2 3 4 5 6
duration of therapy, tetracycline
duration of activity, tetracycline
duration of therapy DECLOMYCIN demethylchlortetracycline
duration of activity DECLOMYCIN demethylchlortetracycline

1-2 "extra" days' activity

after the last dose to protect against relapse



one 300 mg Tablet mid-morning

one 300 mg Tablet mid-evening

It's made for b.i.d.

DECLOMYCIN®

DEMETHYLCHLORTETRACYCLINE

Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

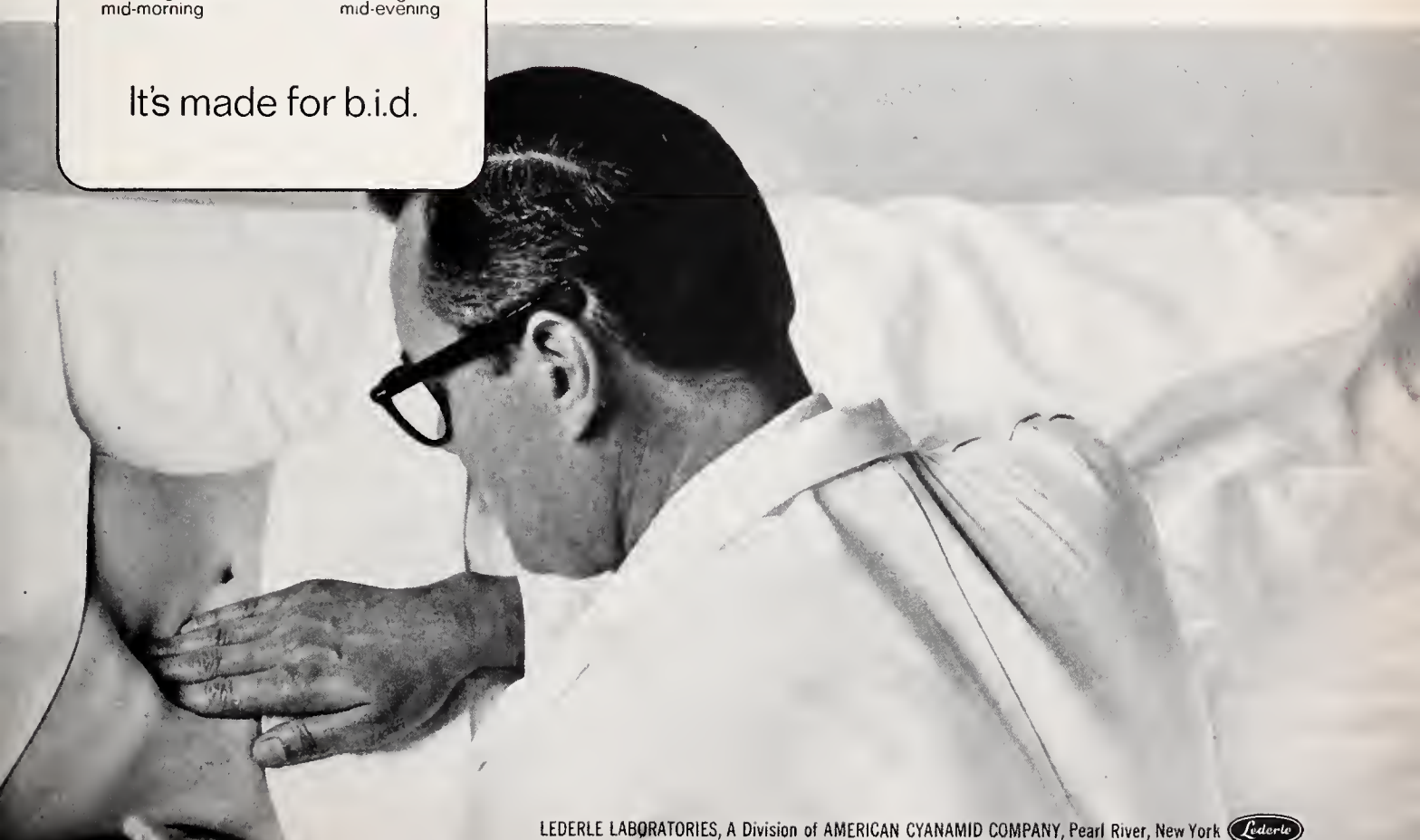
Contraindication—History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort.

Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.



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new

"Doctor, when the kids act up and nothing goes right, I get these throbbing pains in the back of my head."

a new formulation that relieves pain in tension headache and neuralgia

Dialog is a combination of 15 mg allobarbital and 300 mg acetaminophen. Allobarbital, a proven barbiturate, provides desirable sedation in patients experiencing pain and discomfort. Acetaminophen is a nonsalicylate analgesic-antipyretic, well tolerated and useful in a wide range of mildly painful and febrile conditions.

Dialog is well tolerated, even by those sensitive to aspirin. It is nonirritating to the gastrointestinal tract and has no adverse effects on the kidneys.

- Raises the pain threshold
- Suppresses the pain-producing mechanism
- Reduces emotional tension

2/3453MK—2

Dialog™

(allobarbital and acetaminophen CIBA)

Indications: For relief of pain and discomfort of simple headache; neuralgia, myalgia, and musculoskeletal pain; dysmenorrhea; bursitis; sinusitis; fibrositis. Also indicated to reduce fever and to relieve discomfort due to respiratory infections, influenza, and other febrile conditions.

Contraindication: Not recommended during pregnancy.

Caution: May be habit-forming. Do not use in patients sensitive to barbiturates or in those with moderate to severe hepatic disease.

Side Effects: Nausea, transitory dizziness, rash. Overdosage of allobarbital produces symptoms typical of acute barbiturate excess.

Dosage: *Adults:* 1 or 2 tablets every 4 hours. Not to exceed 8 tablets in 24 hours. *Children 6 to 12:* ½ to 1 tablet every 4 hours. Not to exceed 4 tablets in 24 hours.

Supplied: *Tablets* (white, scored), each containing 15 mg allobarbital and 300 mg acetaminophen; units of 3 bottles of 30.

For your convenience—prescription-size bottle of 30.

CIBA Pharmaceutical Company, Summit, N.J.

CIBA

arrest diarrhea

LOMOTIL®

Each tablet and each 5 cc. of liquid contains:

diphenoxylate hydrochloride . . . . . 2.5 mg.

(Warning: May be habit forming)

atropine sulfate . . . . . 0.025 mg.

Effectiveness: Lomotil possesses a unique degree of effectiveness in both acute and chronic diarrhea.

Convenience: Lomotil is supplied as small, easily carried, easily swallowed tablets and as a pleasant, fruit-flavored liquid.

Versatility: The therapeutic efficiency, safety and convenience of Lomotil may be used to advantage alone or as adjunctive therapy in diarrhea associated with:

- **Ulcerative colitis**
- **Acute infections**
- **Irritable bowel**
- **Regional enteritis**
- **Drug therapy**
- **Food Poisoning**
- **Functional hypermotility**
- **Malabsorption syndrome**
- **Ileostomy**
- **Gastroenteritis and colitis**

Dosage: For correct therapeutic effect—Rx correct therapeutic dosage. The recommended initial daily dosages, given in divided doses, until diarrhea is controlled, are:

Children: Age	Total Daily Lomotil Dosage	Lomotil Liquid Dosage (Each teaspoonful [4 cc.] contains 2 mg. of diphenoxylate HCl)
3-6 months	3 mg.	½ tsp. 3 times daily
6-12 months	4 mg.	½ tsp. 4 times daily
1-2 years	5 mg.	½ tsp. 5 times daily
2-5 years	6 mg.	1 tsp. 3 times daily
5-8 years	8 mg.	1 tsp. 4 times daily
8-12 years	10 mg.	1 tsp. 5 times daily

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) Based on 4 cc. per teaspoonful. Maintenance dosage may be as low as one-fourth the initial daily dose.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be kept out of reach of children since accidental overdosage may cause severe respiratory depression. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

Everyone says she's a barrel of fun

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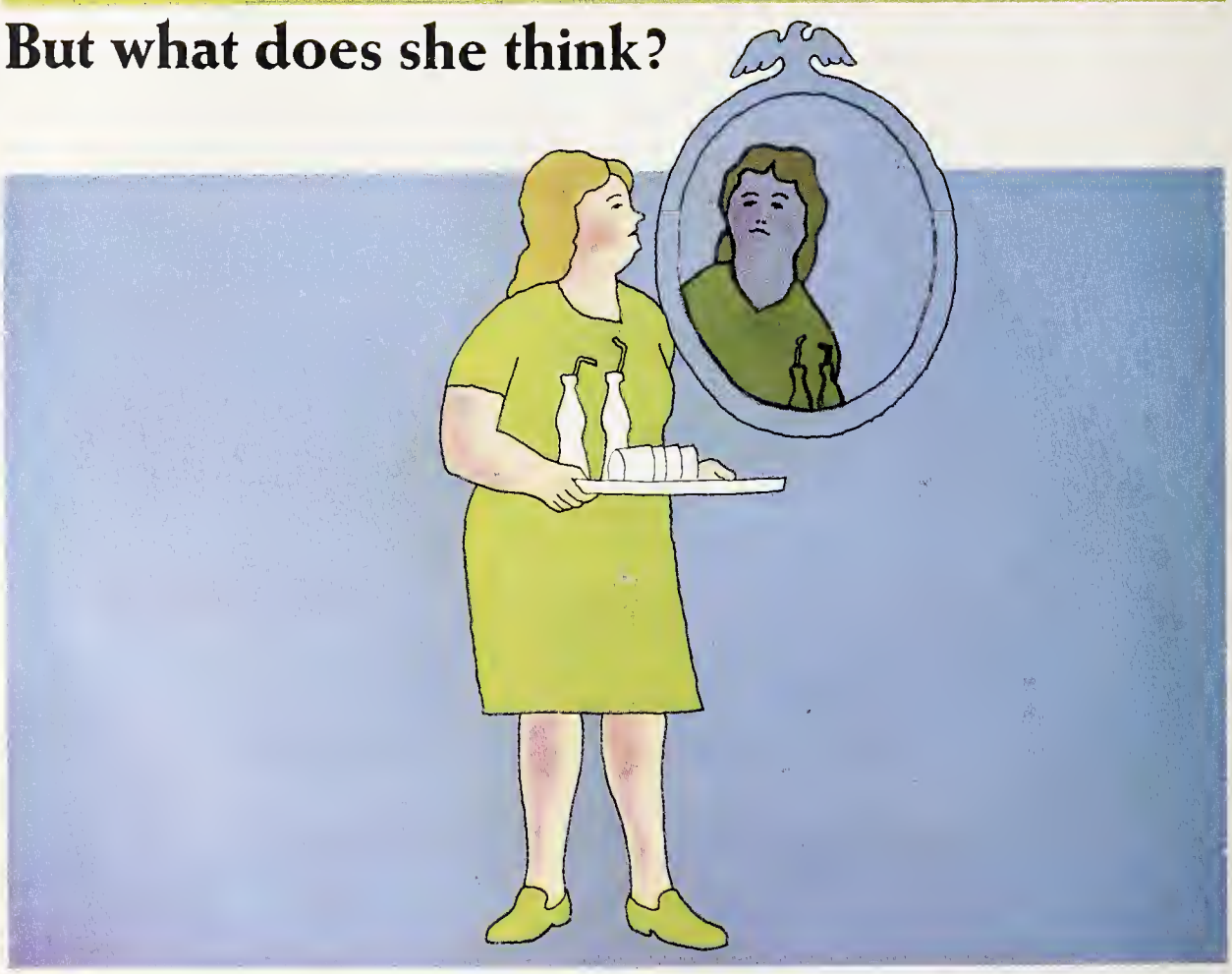

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to help establish a new dietary pattern

Contraindications: Dextro-amphetamine sulfate: in hyperexcitability ond in agitated prepsychotic states. Previous allergic or idiosyncratic reactions to meprobamate.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriote caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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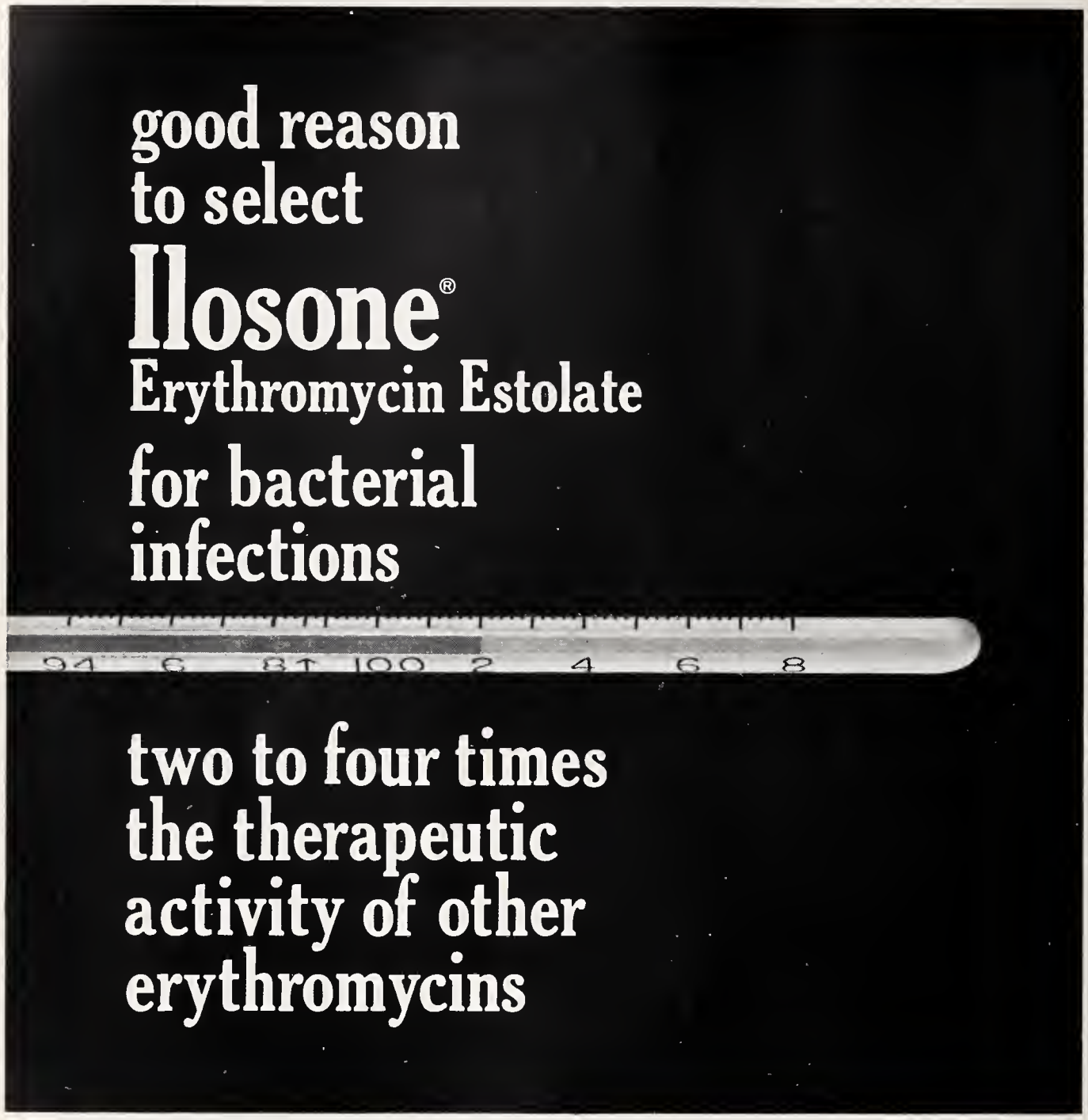

CONTRAINDICATIONS: Ilosone is contraindicated in patients with a known history of sensitivity to this drug and in those with preexisting liver disease or dysfunction.

SIDE-EFFECTS: Even though Ilosone is the most active oral form of erythromycin, the incidence of side-effects is low. Infrequent cases of drug idiosyncrasy, manifested by a form of intrahepatic cholestatic jaundice, have been reported. There have been no known fatal or definite residual effects. Gastro-intestinal disturbances not associated with hepatic effects are observed in a small proportion of patients as a result of a local stimulating action of Ilosone on the alimentary tract. Although allergic manifestations are uncommon with the use of erythromycin, there have been occasional reports of urticaria, skin eruptions, and, on rare occasions, anaphylaxis.

DOSAGE: *Children under 25 pounds*—5 mg. per pound of body weight every six hours. *Children 25 to 50 pounds*—125 mg. every six hours. *Adults and children over 50 pounds*—250 mg. every six hours. For severe infections, these dosages may be doubled.

Available in Pulvules®, suspension, drops, and chewable tablets. Ilosone Chewable tablets should be chewed or crushed and swallowed with water.

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TEXAS MEDICINE

VOLUME 62 NUMBER 9 SEPTEMBER 1966

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From Status Quo, Otolaryngology Tools Have Dramatically Changed

IN 1950 I heard Sir Duke Elder state, in London, that ophthalmology had reached its limit of progress with then available materials and knowledge. The same statement could have been made of otolaryngology as well. Since then, however, science has provided new materials, apparatus, and knowledge, making possible a number of significant advances.

Inert metal and plastic materials now are being embedded in the body every day with apparent impunity, and these materials are successfully performing physiological functions—indefinitely, we hope. Witness the use of prostheses for restoration of ossicular function in the middle ear. The Zeiss operating microscope has made this possible for middle ear surgery, as it has also made possible the removal of small eighth nerve tumors through a translabyrinthine approach. There are apparently many more of these eighth nerve tumors than we suspected. In one series of 100 postmortem temporal bone examinations, 2 percent had asymptomatic eighth nerve tumors. The latest development in the diagnosis of these tumors is the use of a myelogram x-ray, with Pantopaque used to fill the cerebellopontile angle region. The procedure reportedly leads to diagnosis in more than 90 percent of the cases.

A new implement for otologic examination is the electronystagmograph, perfected at the University of Uppsala, Sweden, by Dr. Aschan. This device makes use of the electrical stimuli of the recti muscles while the patient's eyes are closed, eliminating certain eye movements, to make a graphic record of eye movements not observable when the eyes are open. Many cases of neurological disorders, notably multiple sclerosis, are thus detected. Through the use of the electronystagmograph, it was discovered that the hangover period following a drinking bout was concurrent with nystagmus in the opposite direction of that present during the initial period of intoxica-

tion. At the time of this discovery, the only drug used successfully to suppress nystagmus during the hangover period was Phenergan.

Audiometers have provided more knowledge about the other part of the eighth nerve, the auditory portion. The pure-tone audiometer tests many hearing abilities. The patient's threshold for speech is recorded by live voice or from a phonograph recording. The Békésy audiometer produces different patterns, each significant regarding possible lesion.

Because all of this equipment is sometimes needed for a diagnosis, more and more hearing and speech centers are being developed. Since complete diagnosis is time-consuming, requires highly skilled technicians, and expensive equipment, there doubtless will be more and more group practice in this field as a consequence. With the findings of the past 15 years, audiology has developed into a paramedical science.

Another new development is silicone, which is helping significantly where stints are needed in laryngeal grafts following crushing injuries to the larynx. The injectable form of silicones gives promise of voice improvement in patients with paralyzed vocal cords.

The Hemovac has virtually eliminated the fistula complication of laryngectomy, as well as allowing for early ambulation, thus lessening other complications. Cobalt radiation has increased the percentage of cures of supraglottic malignancies and may well obviate surgery in certain cases of in situ carcinoma of the larynx.

Advances, of course, can sometimes bring problems or cause us to overlook older, valuable treatments. A recent editorial devoted much space to the therapy of acute otitis media in children. Disappointingly enough, myringotomy was not even mentioned. I cannot condone the practice of relying solely on antibiotics for a bulging middle ear. Many patients treated with antibiotics are left with some impaired function. I am aware, of course, that the pathological process in the middle ear in such instances is reversible; however, not all conditions revert to their former state. A myringotomy should be performed on every bulging and inflamed tympanic membrane. Moreover, every patient with seventh nerve paresis that does not show some improvement within two weeks should have the benefit of an examination by an otolaryngologist, and decompression should be performed in patients with so-called Bell's palsy if the patient is not improved within four weeks from the onset of paralysis. Too many cases are allowed to continue to the point that complete recovery is impossible. Within recent years, tests have been developed which permit not only location of the site of the lesion in almost every case but also make possible a determination of whether the cause is Bell's palsy or another factor.

All in all, advances offer us an exciting future. The biomedical sciences and industry in general promise faster and more astounding advances in the next 15 years than those of the last. With computers entering the medical diagnostic field, medical applications of the laser, the maser, ultrasonic waves, and countless other devices, what can't the future bring forth?

—JAMES T. ROBISON, M.D., Austin.

Sudden Deafness— A Medical Emergency

SUDDEN PROFOUND sensorineural hearing loss in one or both ears should be considered a medical emergency even though the cause is usually not known. It is seldom bilateral and the patient will tend to minimize his symptom unless it is associated with vertigo. He may complain of a "stopped-up" ear noticed upon awakening, or he may have become aware of hearing loss while answering the telephone. He usually will complain of an associated buzzing or ringing tinnitus which may have preceded the hearing loss.

Upon examination, if the ear appears normal and there is no evidence of conductive loss, the physician must resist the temptation to treat the patient for an obstructed eustachian tube, usually an erroneous diagnosis. With the aid of a 256 tuning fork and the Weber test, the practitioner can determine that the lesion is not in the middle ear if hearing is referred to the better or normal ear. He should then refer the patient to an otologist immediately, preferably making the appointment himself and requesting that the patient be seen the same day or within 24 hours. The severity of a unilateral hearing loss is often not appreciated until an audiogram is made.

A sense of urgency is stressed because of the possibility that the sudden hearing loss may be the result of occlusion (spasm, thrombosis, or embolism) of the internal auditory artery or its cochlear branch. The internal auditory artery is a relatively small vessel, branching at right angles from the basilar artery or the anterior-inferior cerebellar artery. It is an end-artery that divides into cochlear and vestibular branches and usually has no anastomosing or collateral circulation. Kimura and Perlman[1] reported that when this artery was temporarily occluded experimentally in the guinea pig, ganglion cell degeneration often occurred in less than 30 minutes. Complete obstruction of the arterial supply to the cochlea produced widespread degeneration of end organs, ganglion cells, stria vascularis, ductus cochlearis and spiral ligament.

Schuknecht and associates[2] believe that many cases of sudden deafness are the result of viral labyrinthitis. They reported their histopathologic studies of the temporal bones of four patients who had typical histories of sudden deafness and concluded that the pathologic changes were consistent with those found in known cases of viral labyrinthitis (mumps, measles, maternal rubella). The greatest changes involved the organ of Corti, always most severe at the basal end of the cochlea.

Although the cause of sudden deafness is unknown in any given case when it first occurs, several possibilities may be considered:

1. Vascular insufficiency (internal auditory artery or cochlear artery).
   a. Spasm; functional vasospastic changes (sludging) in younger individuals.
   b. Thrombosis.
   c. Embolism.
   d. Hemorrhage; unlikely that pathology would be limited to cochlea or that deafness would be the only symptom.
2. Viral labyrinthitis (mumps, clinical or subclinical, measles, or influenza virus).
3. Hydrops; of cochlear origin or as a part of Meniere's disease.
4. Retrocochlear lesions, central nervous system tumors, multiple sclerosis, syphilis, tuberculosis, trauma.

At first examination, the cause of the hearing loss cannot be determined with any degree of certainty, whether the etiologic factor is vascular spasm, thrombosis, or a viral infection such as subclinical mumps. Hence, to offer the patient every chance for recovery of his hearing, in most cases the treatment, although empirical, should be all-inclusive from the beginning.

The mainstay of treatment has been vasodilation with flushing dosage of nicotinic acid, intravenous and subcutaneous histamine, intravenous procaine, and repeated cervical sympathetic blocks. Diuretics and a low sodium diet are commonly used when hydrops is suspected.

A plea is made here for more frequent early administration of anticoagulants along with vasodilators in sudden deafness. For example, in a middle-aged or older patient, vasodilators alone should not be tried for several days or a week on the assumption that the problem was caused by vasospasm. After a week there may be no improvement or the hearing loss may have increased, and it may become apparent that this case was actually due to thrombosis of the cochlear artery. By such time the ischemia in the cochlea will have produced irreversible damage and it is too late for anticoagulants to be beneficial. It seems justifiable and prudent for the otologist to take the responsibility of initiating his own anticoagulant therapy if he is unable to obtain the consultation of an internist on the same day. One internist has assured me that there is no appreciable risk involved in administering heparin subcutaneously or intramuscularly for one or two days until the patient can be observed more closely by an internist.

Hersperger[3] outlines a regimen of heparinization as follows:

1. Draw blood sample to determine Lee-White coagulation time.
2. Give heparin, 50 mg. subcutaneously, every six hours, adjusting the dosage according to clotting times.
3. Maintain clotting times (drawn five hours after the previous injection of heparin) at two to three times the pretherapy, or control, clotting time. In older patients, clotting time is regulated at two times the control time.

Others have advocated as much as 350 mg. of heparin at once intramuscularly and then 200 mg. every 12 hours for several doses. The average dosage is 250 to 350 mg. daily for the acute clinical period.

Nicotinic acid should be given in flushing dosage, usually 100 to 150 mg. 30 minutes before meals.

If there is reason to suspect a viral etiol-

ogy, particularly in the younger persons, gamma globulin and steroids may also have a place in the treatment of this condition.

After therapy has been initiated, further audiologic tests and other diagnostic studies can be made to differentiate between cochlear and neural or retrocochlear lesions. The important point is that the case of sudden deafness may be due to occlusion of the internal auditory or cochlear artery and that appropriate treatment must be begun immediately.

—OLIVER W. SUEHS, M.D., Austin.

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2. Schuknecht, H. F., et al.: The Pathology of Sudden Deafness, Laryngoscope 72:1142 (Sept.) 1962.
3. Hersperger, W. S.: Sudden Deafness—A Medical Emergency, Bulletin The George F. Geisinger Memorial Hospital and The Foss Clinic, 12:123 (1960).

Life Insurance Medicine Requires Different Philosophy

ALMOST EVERY practicing physician at some time in his career will have occasion to deal with problems involving life insurance. Relations of medical directors with the medical public have taught us that the life insurance business is poorly understood by many practicing physicians. A discussion of this subject may provide better mutual understanding of our common problems.

Life insurance underwriting may be defined as the classification of life insurance risks into categories of individuals whose mortality ratings have been determined by actuarial experience. The following factors must be taken into consideration:

1. Age
2. Occupation
3. Finances
4. Morals
5. Habits
6. Habitat
7. Build
8. Medical history
9. Physical findings

It is immediately apparent that the medical factors constitute only a part of the basis upon which the classifications are made.

The numerical system of rating is used. Table 1 represents a group of 1,000 individuals, each of whose age was approximately 52 years. The normal or standard death rate in such a group is 10 per 1,000 per year. This standard rate is designated as 100 percent. If in a certain group with a common impairment we should expect 15 deaths per 1,000 per year, the numerical rating applied to the group would be 150 percent, or Table B. The percentage of increased mortality is then translated into dollars and added to the standard premium (Table 1). Other rates are given accordingly.

In discussing the medical phases of life underwriting, we should consider for a moment the difference in the philosophy between a clinician and a medical director. Most medical directors have had experience in clinical medicine and believe they understand its problems fairly well. The clinician is trained to deal with individuals, most of whom are ill. It is his function to combat the illness and restore the individual to a normal state of health, if possible. He is concerned chiefly with the patient's present condition and immediate future. In this work he calls upon all his resources of medical knowledge to diagnose the illness and to bring about a state of health. Most of his patients are ill; he is not too often consulted for minor subclinical or preclinical conditions which do not give rise to symptoms. The clinician is not often asked to predict individual longevity except in seriously ill individuals; and then his prognosis is usually guarded.

On the other hand, it is the function of the medical department of a life insurance company to predict longevity. While the clinician is in the business of relieving human ails, medical directors are in the business of relating dollar-value to human lives.

As may be noted from Table 1, an increase in mortality of two to three deaths per 1,000 per year cannot pass unnoticed because our mortality margins are so narrow. For example, in healthy men at age 40, four deaths per 1,000 per year are expected. The individual has a 99.6 percent chance of living to age 41. However, if he has an impairment for which six deaths per 1,000 are expected, he has a 99.4 percent chance of living to age 41. It requires a substantial extra premium to cover the two extra deaths. Most clinicians would recognize that they do not have the tools to make such sensitive predictions. We must, therefore, depend on the statistician and not the clinician for guidance.

Because of this narrow margin of safety, we are interested in many minor, preclinical,

TABLE 1.—*Numerical Rating of 1,000 52-Year-Old Individuals*

	No. of Deaths Per 1,000	% of Mortality Increase
Standard	10	100
Table A	12.5	125
Table B	15	150
Table C	17.5	175
Table D	20	200
Table E	22.5	225
Table F	25	250
Table H	30	300
Table J	35	350
Table L	40	400
Table P	50	500

impairments, such as obesity, slight albuminuria, slight elevation of blood pressure, and minor heart murmurs. These are not of particular significance to the clinician because they are asymptomatic and as a rule little can be done for them; and if something could be done, the problems are not of sufficient bother to the patient for him to make any sacrifices.

Mortality studies regarding various diseases or impairments are constantly being conducted by individual companies and by combined investigations. The results of these are published in the proceedings of the Association of Life Insurance Medical Directors of America, the Medical Section of the American Life Convention, and the Actuarial Society of America. Medical directors are guided by this information in classifying substandard risks.

With such a great difference in philosophy between clinical and life insurance medicine, it is not surprising that there should be differences of opinion regarding the insurability of some applicants. The good physician soon develops a confidence in his clinical findings and judgments. The medical departments of life insurance companies hold his clinical judgment in high regard. However, in the event he has passed a favorable opinion on the insurability of an applicant whom he has examined or upon one of his patients, even though he has found an impairment, and the policy is issued on a substandard basis, he would naturally feel indignant, embarrassed, and resentful. He may be entirely sincere and confident in his judgment and see no reason why he should not reassure his applicant and the agent, too. His first impression is that the medical departments of life insurance companies are ignorant of medicine or that the company is trying to rob the public. He does not realize that the company is using principles and experiences of which he is unaware.

Because of this difference in philosophy, it would be wise for the clinician, particularly one who is an insurance examiner, to assume the attitude of a fact-finder and leave the judging of insurability to the medical director.

—FREDERICK FINK, M.D.,
Dallas, Medical Director,
Union Bankers Insurance Company.

Photosensitivities Around the Home

ARTICLES commonly found around the house and garden can cause dermatitis due to contact with the article and activation by exposure to sunlight. This phenomenon which we call photosensitivity can be difficult to diagnose because of the complex chemical nature of many common household substances. The photosensitizing substance could be an unsuspected additive, such as coloring in candy.

About 1900, J. Prine employed eosin (bromeosin, tetrabromofluorescein) systemically in 26 epileptic patients in an effort to use bromine to control the seizure disorder. He was unaware of any photosensitizing properties of eosin yet he noted the appearance of erythema on the exposed areas of the skin of these patients. Prine considered air, instead of light, the precipitating contact substance. This is probably the first recorded instance of photosensitization.

For this discussion, we will assume that photosensitization from ingestion of drugs and systemic disorders, such as that caused by a disturbance of porphyrin metabolism, have been ruled out.

An eruption in the exposed areas (face, neck, extremities and the V of the upper chest) always makes sunlight a suspected factor. We then start an investigation regarding the many known sensitizers which may have been contacted.

In the garden, a person may contact plants such as parsley, parsnips, carrots, figs, ragweed, or celery which are diseased by "pink rot," a mold. In fact, one of the first instances of eruption due to contact with vege-

tation was noted in Europe where contact with meadow grass plus sunlight caused a skin eruption.

We must be sure that orchard sprays containing tars or phenothiazines have not been used or that contact with these substances in contaminated clothing has been eliminated.

In the kitchen, contact with the rind of the Persian lime can cause an eruption as does benzocaine used in many burn ointments.

In the bedroom where cosmetics are applied, perfume and cologne, especially cologne, cause a characteristic erythema where applied. This is called berloque dermatitis.

In the bathroom, or where the household medicines are used, we see most of our offending substances. These include toilet soaps which contain dibromsalen or a soap often used in hospitals which formerly contained tetrachlorosalicylanilide have caused sun sensitization. This substance has been discontinued, but it is occasionally possible to find an old supply.

Bithionol is a potent antiseptic, used for six years before cases of photosensitization were attributed to it. A common first-aid cream was the most frequent offender. This substance was removed from the product, but old supplies in the drug store or in the medicine cabinet are still found. Other products used as shampoos, medicated soaps, and cosmetic creams are reported to still contain the substance.

Antihistamines used locally, especially Phenergan, cause photosensitivities. Coal tar products applied as medicine or encountered accidentally, such as from asphalt, before sun exposure, are common causes of exaggerated sunburn.

Those handling chlorpromazine can develop a photosensitivity that may take as long as four years to clear, and can cause cross sensitivities to other drugs such as paraphenylenediamine, a common dye, and to Taractan, a tranquilizer.

One of the early sun sensitivities was traced to sulfanilamide taken internally. The other sulfa derivatives used externally can also cause trouble. Psoralens used locally in the treatment of vitiligo can be sensitizers, and oddly enough, sun protective creams, especially those containing para-aminobenzoic acid can do just the opposite.

It is easy to see the complex nature of the investigation necessary to find the offending substance in sunlight reactions. Fortunately, a person must develop an allergy to the substance before he reacts, thus causing the incidence of photosensitization to be relatively low. The problems of a dermatologist are always challenging, but never more so than in the diagnosis of photosensitization.

—J. Franklin Campbell, M.D.,
Fort Worth.

An Ophthalmologist's Plea To the Internist and GP

One of my saddest and too frequent daily office experiences is seeing, for the first time, an elderly patient who has unknowingly lost a major portion of the field of vision in one or both eyes from chronic glaucoma of 15 to 20 years' duration.

Unfortunately, chronic glaucoma flashes no symptomatic red light to alert patients and their doctors to the insidious loss of the off-center areas of vision that advances gradually with the victim unaware of its progress.

The sight of thousands of patients would be saved if each general practitioner or internist purchased a tonometer for his office, and a bottle of 0.5 percent Ophthaine or another suitable topical anesthetic. If he then included in his routine physical examination a tonometer measurement of the intraocular pressure (time investment, 20 seconds) of all of his patients over age 35 he would contribute a great deal toward sight conservation. This extra examination would detect thousands of patients with chronic glaucoma who are now losing their fields of vision, and allow their referral to an ophthalmologist for further evaluation and therapeutic management.

Since ophthalmologists believe that the doctor of medicine can most reliably diagnose glaucoma, and since most patients never see an ophthalmologist, but only get "glass fittings" by nonmedical practitioners, doctors of general medicine can help solve one of our most outstanding problems in sight conservation.

"Finger palpation" of the eyes is unreliable for evaluation of intraocular pressure. The proper use of a tonometer can be learned in a few minutes and will, we hope, become a part of the practitioner's complete general physical examination.

—Carroll W. Browning, M.D., Dallas.

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SEPARATION OF PHYSICIANS' FEES FROM HOSPITAL CHARGES

At a recent meeting of representatives of the Texas Hospital Association and the Texas Medical Association, separation of physicians' fees from hospital charges was discussed at length.

Texas Medical Association believes it is preferable and desirable for each physician to bill his patient directly for the services that he personally contributed to the diagnosis and treatment of the patient. The Texas Hospital Association believes that direct billing by the hospital-based specialists should be determined by the local medical staff or at the community level after adequate education of the public.

Radiologists are very anxious to have separate billing immediately, whereas many pathologists are happy with their present arrangement and wish to ease into direct billing.

There are indeed problems involved which will take time to resolve. For instance, the physician and hospital administrator must make the public aware of the reasons for the separation of physicians' fees and hospital charges. Insurance carriers' costs will increase when these charges are separated, and new insurance contracts will reflect the added costs. Until new insurance contracts are written, there will be some inequities and confusion.

Direct billing is not mandatory, and no "deadline" has been established by the TMA House of Delegates. The medical staff and the administration of the hospital must take the initiative at the local level to solve their particular problems in the best interests of the patient, the hospital, and the profession.

Sincerely,

James D. Murphy M.D.

James D. Murphy, President,
Texas Medical Association

A Key Site of Action of the Protoveratrine A in Salutensin

"The main function of the carotid sinus is regulation of the blood pressure...."[1]

The veratrum component of Salutensin acts here (and in the myocardium), initiating "...a reflex fall in blood pressure through a generalized vasodilation and fall in heart rate."[2]

SCHWENK

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BECAUSE IT ENHANCES THE BODY'S OWN MECHANISMS FOR REDUCING BLOOD PRESSURE

In mild to moderate hypertension:

Salutensin enhances the body's own mechanisms for lowering blood pressure. The veratrum component of Salutensin acts on the carotid sinus and myocardial receptors, initiating "...a reflex fall in blood pressure through a generalized vasodilation and fall in heart rate."[2] To achieve this reflex modification of hypertension, Salutensin utilizes protoveratrine A.
In addition, to facilitate and maintain blood pressure reduction, Salutensin incorporates reserpine and a highly effective thiazide.
In general, side effects have been reported infrequently but may include those listed in the therapeutic summary.

Simple dosage—low-cost therapy: Many patients on Salutensin respond to 1 tablet *b.i.d.* Long-term economy is assured, since dosage can frequently be lowered after initial control is established.

Available: Prescription-size bottles of 60 tablets.

References: 1. Editorial: JAMA *191*:592 (Feb. 15) 1965. 2. Meilman, E., in Moyer, J.H.: Hypertension, Philadelphia, W.B. Saunders Company, 1959, p. 395.

BRISTOL THERAPEUTIC SUMMARY
For complete information consult Official Package Circular.
Indications: Essential hypertension.
Warnings: Small-bowel lesions (obstruction, hemorrhage, perforation) have occurred during therapy with enteric-coated formulations containing potassium, with or without thiazides. Such potassium formulations should be used with Salutensin only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting or gastrointestinal bleeding occurs.
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meet the authors

ALVIN E. RODIN, associate professor of pathology, Medical Branch, Galveston, writes that art and science are as vital to the teaching as to the practice of medicine. **Education; The Neglected Science in Medical School, p. 50.**

H. EDWARD MADDOX, III, Houston otolaryngologist, stresses the need for early diagnosis and treatment of tumors of the eighth cranial nerve. **Acoustic Neuroma Surgery, p. 57.**

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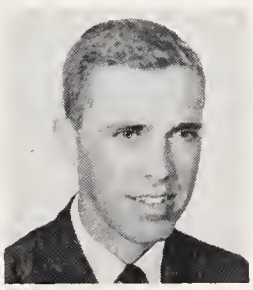

BOB R. ALFORD, assistant professor, and **F. EUGENE PRATT,** resident, are with the Department of Otolaryngology, Baylor University College of Medicine, Houston. **Intracranial Complications from Otitis Media, p. 66.**

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PETE SOTERES, second-year resident in internal medicine at Parkland Memorial Hospital, Dallas, makes the case presentation in this month's CPC. He is a graduate of the Medical College of Alabama. **Clinicopathologic Conference, p. 83.**

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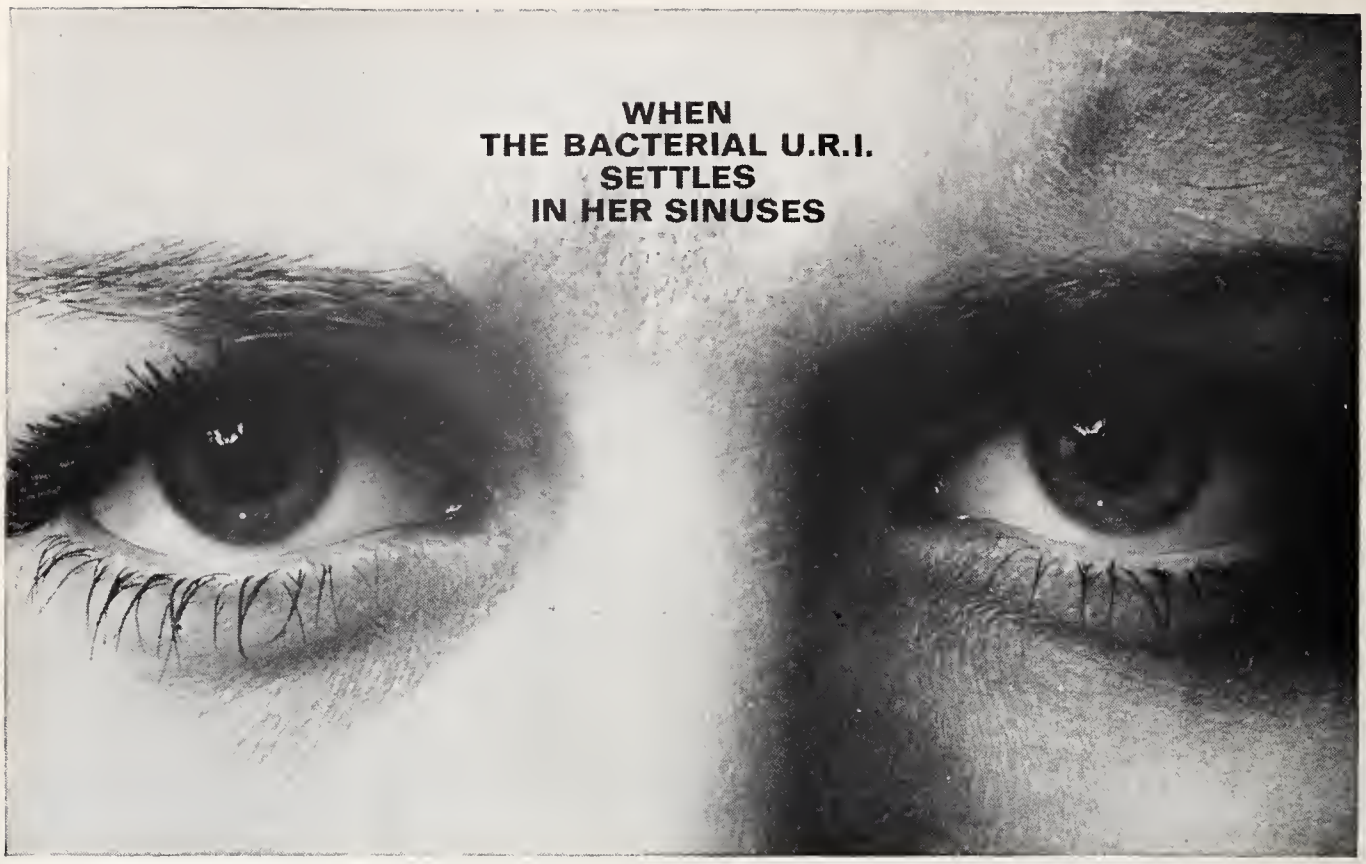

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Education

The Neglected Science in Medical School

The Art and Science Of Medicine and Education

REVIEW ARTICLES

MEDICINE HAS many traditions, one of the most persistent being that it is both an art and a science. It was Hippocrates who first established art as one of the basic tenets of medicine in his famous aphorism, "Life is short and the art long."[1] In fact, we still give it at least lip service in our own time. But is it true, or need it be true today? Does not the phenomenal growth of scientific biologic knowledge in the past century replace the necessity for art in medicine? Surely art is not needed when scientific knowledge is sufficient to understand and cure many of man's ailments.

But what is art? In short, it is the ability for aesthetic discrimination and selection. To the artist the objective is the arrangement and correlation of lines and colors in such a fashion that intrinsic beauty and meaning are achieved, where these were not apparent in the individual lines or colors themselves. To the doctor the objective is the marshaling and ordering of all significant parameters of a patient's inner and outer environment in such a fashion that the utmost in physical and mental well-being is achieved. But many of these parameters are not scientific in the sense of chemical, physiological or morphological criteria. They concern, in part, a feeling for and an understanding of the patient as an entirety, as an individual, as a reacting part of the human race. To quote Lester Evans, "Man cannot be subdivided endlessly; moreover, the most critical tissues escape the scalpel; it is the entire organism which must be studied."[2]

There are some who say that today the art of medicine is vanishing, that in our eager grasping for factual knowledge and research grants, we have lost sight of the patient. Surely, the sum of the parts does not add up to the complete individual. Something is missing—the factor of individuality as expressed not only by living and breathing, but also by feeling, thinking, and reacting. The purely scientific approach, unseasoned with the humanity which comes with the study of the patient instead of merely his disease, is cold, impersonal, and inadequate. Perhaps this is in part the explanation for the decreasing stature of the patient-doctor relationship, and the lack of public resistance to socialized medicine.

I believe that the present scheme of medical education has been and is neglecting the art of medicine. The medical student who is belabored by two years of innumerable and sometimes unrelated data, isolated from the entirety of the patient; and who is then subjected to innumerable and sometimes unrelated specialists, each with his own segmented field, cannot help but become a disease-oriented rather than a patient-oriented physician—one who does not apply the science of medicine in the context of the entire

Presented at the meeting of the TMA Committee on Liaison with Medical Schools, April 13, 1966, Austin.

Medical education is rapidly developing as an acceptable and essential specialty. Many schools have or soon will have undergone extensive review and revision of their curriculum. Any school which is not at present subjecting itself to rigorous examination of its objectives and methods is likely to be, within the next decade, a substandard and inadequate medical school.

ALVIN E. RODIN, M.D.

spectrum of the patient's existence. But if this be so, where does the fault lie? Does it lie in the educational methods themselves, or in the wrong application of these methods? Possibly it lies in both, for education, like medicine, is both an art and a science.

The science of education is based on well-established psychological principles of learning, remembering, and understanding. Some of the more meaningful principles for medical education include:[3]

1. The integration of new material with the old so that the new does not replace the old. How often and how tragic that many students, once they have completed a course and survived its examination, shut off this area of knowledge from their intellectual armamentarium. How advantageous and how meaningful it would be to intimately relate all medical sciences with each other and above all with the patient.

2. The enhancement of motivation of students so that their enthusiasm and learning ability will be increased. We have all seen some medical students become discouraged, if not emotionally disturbed, during the first or second year of basic medical science indoctrination. Some of this, at least, may be related to the fact that their humanistic reasons for entering medicine have been frustrated by the absence of patient orientation and patient contact. Some of these students, at least, could be salvaged and their motives reinforced by direct patient contact from the time their medical training begins.

3. The active participation of students in the learning process so they will know and remember through understanding rather than solely through memorization. We are all aware of students with high achievement in examinations who lack basic comprehension or even prolonged retention of the material. These regurgitative parrots have been handed knowledge on a passive platter, not of gold, but of fact-crammed lectures and condensed, outlined handouts, primers and textbooks. They have not been directed to seek out knowledge for themselves, and probably never will after that magic day when they are given their diplomas. How much better for them to learn how to learn, and how to want to learn. Surely instruction and encouragement in the use of the library would be of more lasting benefit than stuffing their minds with countless and uncorrelated facts. Most of our students are college graduates who are aware of, and capable of, self-guided learning. Recently, for example, 25 sophomores at Harvard successfully petitioned to be taken off the formal lecture curriculum and proceed on a more informal basis.[4] One quotation from Thomas C. King will suffice: "We should be teaching skills which make our continued learning possible, not merely providing our students with a snapshot of the current state of our knowledge; and we should be teaching them to apply these skills in a humane way."[5]

We must also consider the art of teaching. This consists of an ability to select, relate and synthesize facts into a meaningful and stimulating unity, much as the successful

artist does with lines and colors, and the true physician with biological, psychological, and environmental facts. Success in the art of teaching requires not only a detailed knowledge of a subject but also a broad familiarity with all related fields of endeavor, and, above all, an earnest desire to teach. Unfortunately, the selection of teachers in medical schools is based on the premise that skills in the art and science of teaching are automatically possessed by any Ph.D. or M.D.—that teaching is something that requires no special talent or training, and that perhaps it is not quite as desirable as research experience. This is not to say that there are no good teachers in medical school, but, as Gilbert Highet wrote, "Most people are clumsy at learning and teaching, not because they are stupid, but because they have not thought about it."[6]

From these considerations it is evident that both medicine and education are a combination of art and science. Of further importance is an understanding of the interrelationships between these two types of art and science. These can best be appreciated in historical context.

Historical Relationship Of Medicine and Education

The historical varieties of medical education can be delineated by considering the two extremes in methods—the preceptorship and the lecture. The preceptorship is probably the oldest method, being practiced by Hippocrates. A significant modification was the introduction of bedside teaching, first begun by Monte at Padua in the 16th century and finally popularized by Osler four centuries later. The lecture was first widely introduced into medical education in the Middle Ages, when it was used as a vehicle for the rigid propagation of the teachings of Galen. The demonstration is basically nothing more than a lecture with abundant use of audiovisual aids. The first major demonstration consisted of anatomical dissections in the Middle Ages, performed to the intonation of Galen's anatomical writings. The modern closed-circuit television is merely a grandiose, albeit excellent, method for demonstration.

During the 16th to the 19th centuries medical education evolved along two distinctly separate lines. One was the formal, highly structured university school with its heavy emphasis on the works of Hippocrates and Galen, its snobbish use of Latin, and its relative lack of practical experience. The other type of medical training was of lower professional and social stature. It consisted of a preceptor arrangement with an emphasis on practical experience, a relative lack of book-learning, and the use of the common vernacular. Although many of the products of this informal education were close to quackery, some achieved immortality by their contributions to medicine. One of the best examples is Ambroise Paré, a 16th century French surgeon who, unhampered by the often erroneous teachings of the university schools, revolutionized the treatment of wounds.

In colonial America the first form of medical education consisted of preceptorship. At the same time was a proliferation of many transient, proprietary schools, most of which offered medical diplomas after a course of several months, and some of which eliminated even this prerequisite. The first real advancement in American medical education came with the founding of the Medical College of Philadelphia by Drs. John Morgan and William Shippen, Jr., in 1765.[7] This was a major achievement because of the insistence on university affiliation, and its emphasis on research and proper educational prerequisites. However, the ignoble proprietary schools continued unabated.

By the beginning of the 20th century, American medical education in general was in a pitifully inadequate state; a few exceptions included the University of Texas Medical Branch. The situation had become so extreme that the Carnegie Foundation appointed Abraham Flexner to investigate the state of medical education and submit recommendations for improvement. His report of 1910 revolutionized medical education because of its emphasis on a more scientific approach, a strengthening of preclinical departments, and increased prominence of research.[8] The Flexner report served as the initiation of our present day curriculum with its stereotyped structure of two years of basic science consisting of a conglomeration of lectures, demonstrations and laboratories; and two years of clinical work with fewer lectures and introduction of preceptorship work on the hospital wards and in the outpatient clinics. Thus, at present, medical education is structured on the historical tradition of both the university lecture-oriented, and the preceptorship-oriented schools of the Middle

Ages. Admittedly, the content differs now, but the basic formats are quite similar.

Both the lecture and preceptorship are needed today, but are used in the present curriculum to foster certain approaches to medicine which can be detrimental to an integrated understanding of the patient and his disease. The lecture and the preceptorship are all too often the vehicle for the propagation of knowledge related to only a single specialty. This provides an artificial division of biology, which in nature is not demarcated into preclinical sciences or clinical specialties, but instead, forms one unified pattern from molecular structure to clinical signs and symptoms. Quite pertinent is a quotation from the noted educator Alfred North Whitehead: "The result of teaching small parts of a large number of subjects is the passive receptivity of disconnected ideas, not illuminated with any spark of vitality."[9] Surely, now is the time to go beyond the educational methods of the Middle Ages and teach our students in a format which is more in keeping with modern biological and educational principles.

New Trends in Curriculum Design

There have been two major trends in curriculum design within the past decade—teaching by subject committees, and the integration of premedical and medical training. Western Reserve University School of Medicine, in 1952, was the first medical school to break away from the classic pattern of segmented, independent teaching by departments.[10] Instead, instruction is centered on subject areas, with a committee of departmental representatives responsible for the organization and presentation of each. The curriculum is divided into three phases. The first phase, the study of biology basic to medicine, occupies the first year. Subjects include, in order of presentation, cell biology, tissue biology, neuromuscular systems, metabolism, cardiorespiratory systems, integrating mechanisms and reproduction. The next one and one-half years are devoted to the second phase, called principles of medicine. The various body systems are considered, with all aspects of each system presented in an integrated manner. Members from each department participate where indicated in each unit. The third phase is devoted to the care of the patient. Also part of the Western Reserve innovation has been the assignment of the students to patients or families at the beginning of their medical training, and the participation of each student in a research project.

The major advantage of this integrated curriculum is that all aspects of a biologic unit are given at the same time and correlated for maximum understanding. This is in contrast to the usual discordant traditional curriculum where different aspects of the same item or system are presented at different times, and often with little correlation. The usual objections to the integrated program are the amount of time involved in its organization, and the weakening of departmental identity. However, at Western Reserve the departments have remained as strong units, with their usual budgets, independent research, and graduate courses. The multidisciplinary teaching of medical sciences has been adopted in part by several other schools, the University of Virginia being the latest. Its basic rationale has been well-expressed in the 1965 Coggeshall report on medical education: "There is growing realization that it is unrealistic to assume that effectively articulated education, progressing in logical sequence from the rudiments to mastery, can be provided through segmented programs under different jurisdiction. A most significant implication of emerging trends is that medical education should, in the future, be planned as a continuum—a continuous process with all elements carefully integrated and under coordinated leadership."[11]

On the clinical level, integration involves what Lester Evans has termed "multi-disciplinary comprehensive patient care."[2] This entails the study and care of the patient not only in the hospital but also at home, work, and play. It also demands that medical students be taught, not only from the separate viewpoints of the internist, surgeon, social worker, and so forth, but also from the all-embracing point of view of a sick human being. To meet this objective several medical schools, including those of Cornell University, University of Colorado, Temple University, and University of North Carolina have established general, interdepartmental, comprehensive outpatient clinics for fourth-year students.[12]

The other major development in curriculum structure was pioneered particularly by Johns Hopkins University School of Medicine in 1959. After two years of college, premedical and medical courses have been combined in a five-year academic program. In

the early years liberal arts, natural sciences, and medical sciences are combined, with the granting of an A.B. degree at the end of the second year. In 1961, Northwestern and Boston Universities went even further, accepting high school graduates for a six-year medical diploma course. Johns Hopkins and Northwestern have offered these integrated courses for only a minority of their students, whereas Boston has structured its entire curriculum on this basis. An even more radical change was proposed during the Endicott House Conference on Medical Education in 1965.[13] Recommended was condensation of years of college through postgraduate training to nine years.

Advantages usually given for this system of integrated university-medical training are avoidance of unnecessary duplication, saving of one or two years, and full coordination and continuity between premedical and medical subjects. In addition, the best students can be selected for training at an earlier age. On the sociological and philosophical level, it may obviate the detrimental tendency for medical students to be completely isolated from other sciences, from the humanities, and from society. After four years of wearing blinkers, is it no wonder that the average physician is considered uncultured? To prevent this, the new Mt. Sinai School of Medicine, New York, is introducing courses in behavioral sciences, social anthropology, philosophy, and ethics during its four-year course.[14] Partial integration at Stanford has been attempted by moving the School of Medicine from San Francisco to the University at Palo Alto.

There are several indications that the changes described do not represent a few isolated, local incidents. A recent survey by the AMA Council on Medical Education has revealed that 18 schools are planning for major curriculum changes in the near future.[14] In addition, several of the newly developing schools have announced curriculum innovations. Multidisciplinary programs are being established at the developing College of Medicine, Pennsylvania State University, Hershey, and University of California San Diego School of Medicine. An integrated premedical and medical curriculum is being organized at Michigan State University College of Human Medicine in East Lansing, and at Brown University Program in Medical Science in Providence, R.I.

These considerations lead to only one conclusion—that a wave of unrest and change is taking place in American medical education, equivalent in magnitude to the period following the Flexner report. And perhaps it is none too soon. This is not to say that every medical school should immediately adopt any of the newer programs or that they are not fraught with difficulties, inconsistencies, and imperfections. Admittedly, any one type of curriculum is not the best for every school. There are many local factors, such as size of school, state or private affiliation, needs of the immediate community and so forth. But it is precisely for these reasons that each school must, at regular intervals, subject its curriculum to rigorous examination.

Methods for Curriculum Change

The guide lines for such an introspection can be formulated from the experience of several schools. The first major step is the organization of an active working committee, separate from the curriculum committee, and responsible to the dean and entire faculty. This committee is given several terms of reference, and reports its recommendations to the faculty at definite intervals. The entire procedure of review can be organized into three successive phases.

1. *Determination of Objectives.*—An outline of objectives is useless if it consists of platitudes or generalities. Instead, the objectives must be stated in terms of what the student is expected to be able to do after completion of his training. For example, stating that the objective is the production of good doctors is of no value. Instead, the objectives must consist of what specific knowledge, habits, basic skills, and ethical principles are desirable. Once these are delineated by the committee and approved by the faculty, the next phase begins with a question: Does the current curriculum fulfill the desired objectives?

2. *Evaluation of Current Curriculum.*—At this point we may well say that we have always evaluated our students and courses by frequent examinations. However, what have we been trying to determine—how much the student has succeeded in memorizing? what grade to place on his record? or whether the specific, predetermined, objectives of the curriculum have been met? For example, if ability at problem solving is a major objective,

then we must test for this parameter, rather than for straight recall of facts. On still another level, we can provide students with attitude questionnaires in order to determine whether or not they are developing attitudes consistent with the school's objectives. Still further, we can gather statistics on our graduates. For example, if the school's objective is primarily the production of general practitioners, then how many graduates go into this field? Only by such searching and constant appraisal can we be certain that our objectives are being realized. But, of course, we must first know what our objectives are.

3. *Reorganization of the Curriculum.*—If it is found that the present curriculum fulfills our objectives, fine. Leave it alone. But if not, then it must be altered. Admittedly, this requires considerable experimentation, but research in medical education is no less respectable than in the basic and clinical sciences, and certainly can be as fruitful.

The pattern of medical school introspection which has been outlined is not easy in execution. It is time consuming and requires considerable perseverance, if not diplomacy. But the rewards more than justify the few years of labor. The benefits are many. Foremost is the development of the most suitable curriculum for a particular school and its community. Others include the increased awareness and interest of the faculty in education, and the increase in morale of both the faculty and student body.

But the task is not completed on establishment of a new curriculum. Science, economics, and social needs are continually changing, and therefore objectives and curriculum of the school must be reevaluated at regular intervals. To assist in this, departments of medical education have been established in many schools including the Universities of Virginia, Illinois, Southern California, and Rochester; Western Reserve University; Albany Medical College of Union University; and the Medical College of Georgia. These departments are composed of educators, psychologists, and physicians, whose objectives include assisting the faculty as consultants in all educational matters, conducting research in medical education, and evaluating teaching programs, methods, and results. Such a department is essential in every medical school if it is to achieve its basic purpose: educating the physicians of the future.

Objections to Curriculum Change

Several objections have been repeatedly and vehemently voiced against this extensive process of evaluation and change.

1. "Our present curriculum has been producing excellent physicians for many years." But how do we know whether or not most of our graduates practice modern, scientific medicine? There have been only a few objective assessments of the adequacy of medical practice. In 1956, Osler Peterson[15] reported the first such study, which was on 88 general practitioners in North Carolina. The results are disturbing to say the least. For example, 78 percent took inadequate histories, 86 percent did not examine breasts routinely, 71 percent did not perform neurologic examinations, 66 percent never used an ophthalmoscope, 64 percent did not usually do vaginal examinations, 45 percent performed examinations with the patient dressed, and 43 percent used inadequate sterilization techniques for syringes and instruments. Treatment was no better: 85 percent used shotgun therapy for anemias, 67 percent gave antibiotics indiscriminately for upper respiratory infections, 62 percent provided inadequate perinatal care, and 46 percent made no effort to avoid drug reactions. In light of these findings, the mildest statement that can be made is that there is some room for improvement.

2. "There is no proof that any one type of curriculum produces better doctors than another type." But this statement misses the whole point. Changes are not made on the basis of vague generalities like better doctors, but to produce doctors with certain specific skills, habits and principles that have been deemed desirable by the school.

3. "Change just for the sake of change is a waste of time." Of course, the counter statement is that what is desirable is not change in itself, but an investigation into whether or not change is required.

4. "The only important factors in education are a well-motivated student and a good teacher." This statement has several implications. First, it assumes a one-to-one student-teacher ratio that is unobtainable in medical school. Second, it assumes that students are and will remain well-motivated. However, curriculum structure and teaching techniques play a major role in the continuing motivation of students and the effectiveness of their relationship to the faculty. Third, it assumes that additions to the medical faculty are

made on the basis of teaching ability, an assumption that is quite erroneous. The only wonder is that there are some good medical teachers today, when appointments are made primarily on the basis of research interest and achievement.

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Test Detects Stroke-Prone Persons

A simple 60-second screening test to detect stroke-prone persons has been demonstrated by a Veterans Administration physician. By pressing the thumb against the neck artery for about 30 seconds, a doctor may determine if there is an obstruction in the arteries which would cause the flow of blood to stop.

Dr. Stephen A. Hegadus, Director of the Surgical Research Laboratory, VA Hospital, Perry Point, Md., said a patient will lose consciousness momentarily if there is a block in the artery to which no pressure has been applied. This "carotid compression test" has been used successfully on more than 200 patients ranging from 30 to 92 years old.

If the patient shows positive results from the test, arteriography is performed to locate the obstruction.

Eleven patients who had obstructions detected by this method consented to operations in which the clot was successfully removed.

Acoustic Neuroma Surgery

H. Edward Maddox, III, M.D.

All persons with unilateral sensorineural hearing loss should have a battery of audiologic and roentgenologic studies for acoustic neuroma. Methods of diagnosis and surgical treatment are reviewed.

REVIEW ARTICLES

RECENT ADVANCES in audiometry (acoustic) and radiographic techniques have made early diagnosis of tumors of the eighth cranial nerve a practical procedure for every otolaryngologist. A person with unilateral sensorineural hearing impairment, especially if it is accompanied by some loss in discrimination of speech, should have the benefit of advanced audiologic studies. Even the patient with a unilateral tinnitus should be examined carefully to rule out this lethal tumor. A ringing in one ear may be the earliest clinical manifestation of this lesion in many patients. Slight unsteadiness also develops relatively early in patients with acoustic nerve tumors. This symptom should alert us to the possibility of the lesion. Our time-honored, and often abused, diagnosis of Meniere's disease or syndrome is no longer safe without first ruling out a tumor. A number of patients with acoustic nerve tumors have had, at onset, typical signs and symptoms of labyrinthine hydrops. Acute sudden deafness secondary to vascular occlusion or viral inflammation has been the initial symptom of tumor of the eighth cranial nerve in several cases.

Audiometric Testing

One cannot help but be impressed with the wide variety of audiometric curves seen in cases of acoustic nerve tumors. In the majority of patients the unilateral sensorineural loss is predominantly in the high frequencies (67 percent) (Fig. 1).[5] The onset of the deafness is, as a rule, slow and insidious, but in an occasional patient, sudden and severe loss of hearing may be the initial clinical symptom of the neuroma. Standard Spondee and phonetically balanced word tests routinely reveal the patient's inability to distinguish speech. These poor scores seem especially unusual when the patient has only a relatively mild pure tone loss, and should alert one to the real possibility of an acoustic nerve tumor. Speech discrimination scores below 60 percent were recorded in 78 percent of House's group of 114 patients with neuroma.[5] Although this percentage is high, it must be pointed out that 22 percent of patients with this tumor have good speech understanding. Therefore, one should not be misled by this score alone.

All patients with unilateral sensorineural hearing impairment should have a further battery of audiometric tests. The short incre-

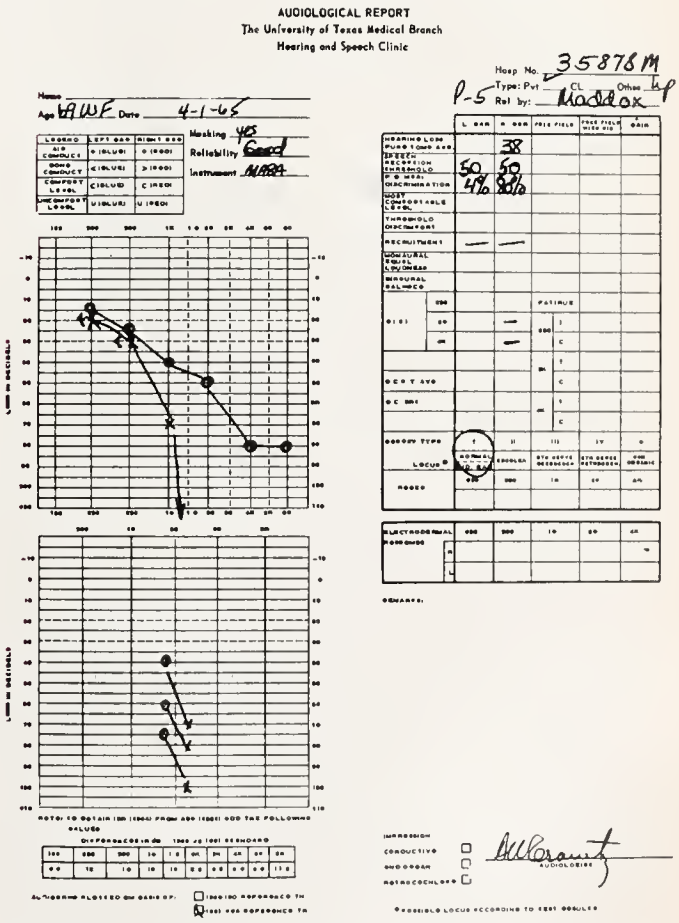

Fig. 1. Audiogram of a patient who had a tumor of the left acoustic nerve shows 50 db. sensorineural deafness in the left ear with only 4 percent speech discrimination.

ment sensitivity index (SISI), the alternate binaural loudness balance test for recruitment, and tests for tone decay or fatigue will tend to pinpoint the site of involvement as in the cochlea or more central (retrocochlear).

Also, Békésy audiometry, whenever available, should be done to rule out an eighth nerve tumor. It must be emphasized that these advanced tests are run as a battery and can only be interpreted together to lend more evidence to the case.

Vestibular Testing

Caloric testing should be done routinely. Either the conventional, or a modified, cold water test or electronystagmography will reveal significant changes in vestibular function in a high percentage of acoustic tumor patients. With either method of testing, 93 percent of individuals have measurable unilateral canal paresis. If facilities for electronystagmography are available, an added bonus is present; 20 percent of acoustic tumor patients have spontaneous nystagmus with the eyes closed. This nystagmus generally beats opposite the involved ear, and is not seen when the eyes are open.

X-Ray Diagnosis

Accurate roentgenologic diagnosis of lesions in the cerebellopontine angle can be difficult because the patient often has no detectable bone involvement. In the past, the otolaryngologist has frequently suspected a tumor because of the audiometric data only to have the possibility dismissed when plain films do not indicate one. It is becoming more evident, however, that enlargement or erosion of the internal auditory canal or meatus is not an early sign, but occurs only after a large expanding lesion has been present for many months or years. Three different roentgenographic projections are used to view the canal: (1) the Caldwell, or transorbital, shows the entire canal from an anterior-posterior aspect; (2) the Stenvers projection exhibits the meatus well looking at the canal end-on; and (3) the Chamberlain-Towne shows the canal from above. All three are essential for an accurate evaluation of this remote bony area. In the best of hands, these x-rays are 61 percent diagnostic and 26 percent suggestive.

The Myeloencephalogram

Development of the technique for posterior fossa myelography in recent years has added another important tool for the use of the otolaryngologist and neurosurgeon in the early diagnosis of acoustic nerve tumors. Posterior fossa myelography with iophendylate (Pantopaque) as the constant medium, is a safe and accurate method for the diagnosis of cerebellopontine angle lesions and can be carried out with equipment available in most departments of radiology (Figs. 2 and 3). This examination will definitely establish presence or absence of a tumor when the clinician is faced with normal plain films. Scanlan[5] reports 98 percent accuracy with his technique. He had one false positive and one false negative result in 100 patients.

Differential Diagnosis

Many different tumors may arise in the cerebellopontine angle, but the acoustic nerve tumor is by far the most common. The other two tumors most often identified in this area are primary cholesteatomas and meningiomas.

Surgical Approaches

House and his associates have listed four surgical approaches to the acoustic tumor: (1) the middle fossae approach for small tumors when some useful hearing may be present; (2) the translabyrinthine approach for tumors of moderate size in which there is

Fig. 2. Myeloencephalogram shows a moderate size tumor of the left acoustic nerve.

no brain stem involvement or elevation of protein in the cerebrospinal fluid; (3) the classical suboccipital operation; and (4) a new trans-sigmoid two-stage approach. This procedure involves a first stage translabyrinthine and suboccipital bony removal and clamping of the sigmoid sinus. A second stage is carried out five to ten days later with ligation of the sinus, opening of the dura and removal of the tumor.

Case Material

During the past year we have examined 11 patients with cerebellopontine angle tumors at the University of Texas Medical Branch. This work has been a joint effort of the Division of Otorhinolaryngology and Neurosurgery. We are deeply indebted to our neurosurgical colleagues for these opportunities and their excellent cooperation during every phase of this work. Nine of these patients had acoustic nerve tumors. All of the tumors were unilateral except one. A young girl with neurofibromatosis exhibited bilateral involvement. One patient had a primary cholesteatoma involving the petrous tip and angle, and one patient, a 43-year-old man, had a craniopharyngioma extending into this area.

Surgical Technique

The classic neurosurgical approach to acoustic tumors, with some modifications, is not a new one but offers, as a rule, excellent exposure for their complete removal. This approach was used in each of our cases. Myelograms indicated that the tumors were of a size not suitable for translabyrinthine removal.

The patient is placed in a sitting position with his head flexed as much as possible. This position obviously is a unique one for the otolaryngologist, and there are problems in using the microscope which do not occur when the patient is prone. Neurosurgeons believe the sitting position is necessary and most important to keep blood from pooling in the wound. It does offer excellent dependent drainage. The patient's legs are wrapped to prevent edema and an indwelling catheter is inserted.

After much mechanical experimentation, a satisfactory alignment of the operating microscope was obtained (Fig. 4). During our first surgical procedure, the operator stood in order to use the microscope. Subsequently, a stand was fashioned to enable the surgeon to sit on an elevated stool.

The vertical incision is made over the occipital area and extended to the neck. Cautery is used liberally. After hemostasis, the occipital bone is exposed and an opening made by a burr is enlarged with ronguer for-

Fig. 3. A large acoustic nerve tumor is seen on the myelo-encephalogram.

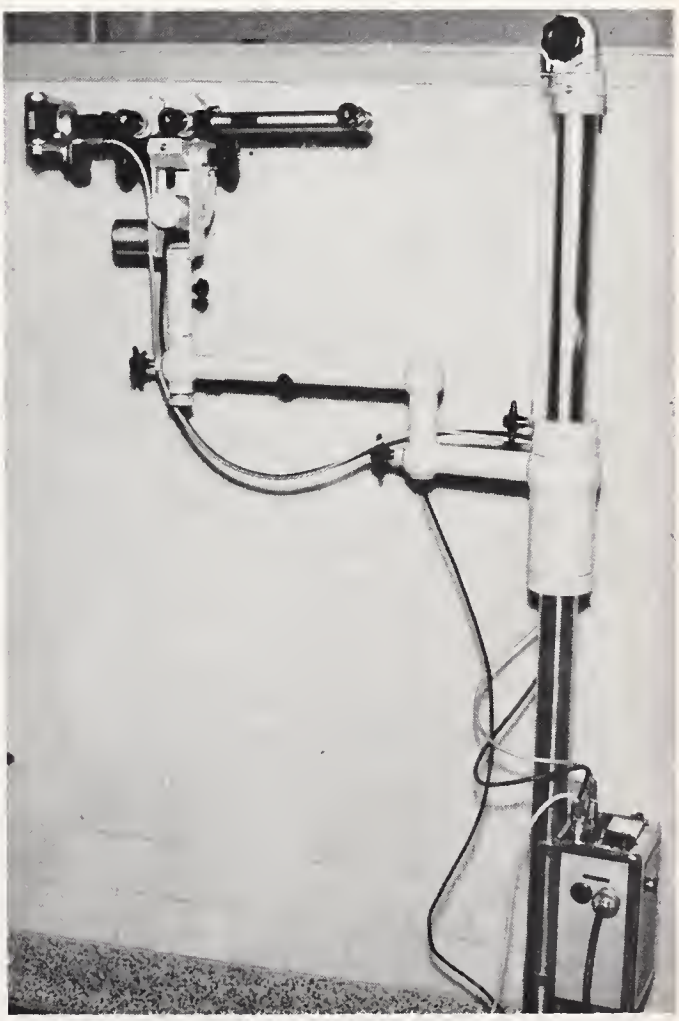

Fig. 4. Zeiss operating microscope used in suboccipital approach to acoustic tumors.

ceps. The bone removal is limited superiorly by the transverse sinus and laterally by the mastoid air cells which must be considered a potential source of contamination. The midline limits the bone removal medially. A catheter may be placed in the ventricle if increased intracranial pressure is suspected. After removing as much bone as possible, the surgeon makes a cruciate dural incision and retracts the dura from the cerebellum.

Gentle retraction, medially, of the cerebellum gives a clear view of the posterior aspect of the petrous pyramid and the internal auditory meatus. It may be necessary, however, to resect the lateral one-third of the hemisphere, using cautery and suction. A self-retaining retractor is inserted to keep the cerebellum out of the field.

During their training, all otolaryngologists study the anatomy of the posterior petrous area; however, a brief general review might be in order. The operative field is limited superiorly by the attachment of the tentorium and the superior petrosal sinus. Inferiorly, the 9th, 10th, and 11th cranial nerves are clearly visible as they exit through the jugular foramen. The tumor itself occupies the medial aspect of the field. The endolymphatic sac, located approximately halfway between the meatus and the sigmoid portion of the lateral sinus (approximately 11 mm. from the meatus) can be identified. The dura is tightly adherent to the posterior aspect of the petrous pyramid and is quite avascular.

The facial nerve, the intermediate nerve and the acoustic nerve all enter and traverse the internal auditory canal. Their entrance is usually obscured by the tumor mass. The fibers of the intermediate nerve form a small bundle in the meatus between the seventh and eighth cranial nerves. In the canal the facial nerve is situated anteriorly and superiorly while the acoustic nerve is more posterior. The nerves are surrounded by a common dural sheath or extension. Shortly after entering the meatus, the acoustic nerve divides into its several branches: the cochlear division goes anteriorly and inferiorly to an inferior relationship to the facial nerve at the fundus of the canal. The vestibular division runs posteriorly and divides into a superior vestibular nerve to the utricle, anterior and lateral canal ampullae, and an inferior vestibular nerve which receives impulses from the saccule and posterior canal ampullae. At the fundus of the internal auditory canal, a transverse bony plate, or crista, separates the facial and superior vestibular nerves from the cochlear and inferior vestibular nerves.

In an effort to determine the exact relationship of these small structures to one another and to obtain more specific measurements than are available in the literature, ten temporal bones were dissected and detailed notes made of these relationships.

The internal auditory meatus is elliptical rather than round and varies greatly in size. The average dimensions, however, are 4 by 6 mm. About 5 mm. of bone, covering the canal posteriorly, needs to be removed to expose its contents. The midpoint of the transverse crista at the fundus of the canal is 6 mm. from the posterior lip of the meatus. The vestibule lies 7 to 8 mm. from this posterior rim also. The underneath surface of the stapes footplate is clearly visible in the vestibule. The vestibular aqueduct and its endolymphatic sac lie 11 mm. from the meatus, and 4 mm. below the meatus the 9th, 10th, and 11th cranial nerves are encountered as they exit through the jugular foramen.

The acoustic tumor, unless it is minimal in size, obscures the internal auditory meatus with its mass. The tumors as a rule are rather vascular and have the pinkish-yellow appearance typical of a neurilemoma found anywhere in the body. If the tumor is gently retracted medially, the rim of the meatus is brought into view. This helps greatly to orient the operator, and its identification is essential before the drilling is started. Eighth nerve tumors arise from the sheath of the nerve, most often deep inside the internal auditory canal. As the tumor grows and expands the canal, it will eventually move through the meatus into the cerebellopontine angle. In early stages, tumors may be completely confined to the canal. This factor is important because certainly with microsurgical techniques a more complete intracranial removal of the tumor can be accomplished than with conventional procedures.

After the operator is fully oriented to the field, the dura over the posterior aspect of the petrous temporal is excised and reflected with a small duckbill elevator. Care is taken to avoid manipulating the tumor, which would cause much bothersome bleeding. A point is selected, about 2 to 3 mm. behind the posterior lip of the internal auditory meatus, to begin the drilling. We used a Wullstein handpiece and a variety of small cutting

burrs. Diamond burrs are often used after the nerves are exposed. A crescent-shaped cavity is formed with the drill and is gradually enlarged. After 4 to 5 mm. of bone has been removed, the pink discoloration of the dural extension can be seen through the bone. This is quite similar in appearance to the dura seen on the tegmen during mastoid surgery. It is important during the drilling to leave the posterior meatus lip intact. This prevents any trauma to the tumor or the structures in the meatus by the running burr. The exposure of dural sheath is gradually enlarged in all directions. It is best, although not essential, to leave a thin layer of bone over this dura until most of the bone removal has been accomplished. Superiorly, in several of our temporal bone dissections, we found large petrous-mastoid air cells which might confuse the operator. After drilling is nearly completed, the thin layer of bone covering the dura is removed with small picks and the dural extension is excised with a sickle knife. If the tumor is relatively small, the contents of the internal auditory canal are clearly seen bathed in spinal fluid. However, if the tumor is large it fills the entire canal and makes identification of the nerves impossible until some of this neoplastic mass is removed. If the preoperative roentgenograms show enlargement of the meatus or, especially, erosion of the canal, one can expect to find tumor in the canal, which presents a much more difficult surgical problem. Upon retraction of the acoustic nerve branch, the facial nerve comes into view in its anterior superior position in the canal. Smaller tumors may be seen arising directly from the eighth nerve which also confirms this nerve's identification. The cochlear and vestibular divisions of the nerve are also readily located when a small tumor is present. Next, the acoustic nerve is cut, distal to the tumor, and the tumor mass with attached segment of nerve is gently separated from its companion seventh nerve and delivered from the canal. In large tumors it is impossible to identify the eighth nerve, and the tumor mass in the canal is slowly removed using small ear cup forceps and suction until the deeply placed seventh nerve is positively located.

The main bulk of the tumor is next attacked. We prefer to decompress the tumor mass extensively by intracapsule removal of a portion of the neoplasm. The tumor capsule, if attached to the pons medially, is gently separated from this vital structure.

As far as saving the facial nerve is concerned, the removal of the deep portion of the tumor over the attachment of the nerve to the pons appears to be critical. The internal auditory canal is packed with Gelfoam after the tumor has been completely removed. Strict attention is paid to absolute hemostasis. The soft tissues are closed by the neurosurgeon in the customary manner.

Conclusions

No matter what aspect of acoustic neuroma is under discussion, one always returns to the absolute fact that early diagnosis is the single most essential objective. We have not yet been fortunate enough to find this tumor early, but several important points concerning diagnosis should be repeated. Audiometric studies are obviously the basis for screening all patients for the neoplasm. Any person with a unilateral sensorineural hearing loss, especially if accompanied by some loss in discrimination for speech, should have the benefit of further specialized hearing tests such as the SISI, tone decay, and alternate balance tests. Every otolaryngologist can perform the cold water caloric test in his office; it takes but a few minutes. Caloric abnormalities are found in 96 percent of patients with acoustic tumors. Pantopaque studies of the posterior fossae are extremely accurate in diagnosing small, early tumors. Other roentgenological findings such as widening or erosion of the canals are late signs. Traditional symptoms of trigeminal and other cranial nerve involvement indicate large tumors. High cerebrospinal fluid protein levels also occur only late in the tumor's development.

This type of surgery cannot be a one-man affair. There is a definite need for an organized team approach. We have all learned a great deal from these cases, and the knowledge provides an excellent basis for improvement of our future surgery.

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Extrusion of Transtympanic Ventilating Tubes: Preliminary Report on Permanent Myringostomy by Use of a Fixed Prosthesis

RESEARCH & INVESTIGATION

TRANSTYMPANIC ventilating tubes have been successful in the treatment of chronic tubotympanitis with effusion. They were originally inserted through a myringotomy incision to provide prolonged drainage of the middle ear[1], but it rapidly became apparent that after the fluid was initially removed, no further drainage occurred.

Ventilation of the middle ear by these tubes, with resultant relief of the negative pressures occurring in the condition, seems to prevent the reformation of the effusion no matter what the cause of the eustachian tube dysfunction.[2] Although there have been some physiologic objections raised and experimental evidence to the contrary, clinically this holds true. As long as the tubes are in place the ambient pressures are equalized and no effusion develops.

So dramatic has been the hearing improvement following the insertion of these tubes that it is doubly disappointing when they are extruded and the fluid and hearing loss recur, for no matter what their size, shape or substance, tubes that are not fixed are eventually extruded (Table 1).

Causes of Extrusion

The mechanism of extrusion is a source of conjecture. Some of the proposed causes have been:

Recurrence of Infection (Table 2).—Concurrent infection occurred 46 times during the observation period of 218 cases in this series. The infections were usually the results of a purulent rhinitis with otorrhea occurring through the lumen of the ventilating tubes. When this complication developed nearly half of the ventilating tubes were extruded prematurely, either immediately or shortly after the infection subsided. Silastic tubes seemed to withstand the results of infection best.

However, since the tubes were retained in 24 infected ears in this group, and the majority of patients had no infection, this factor cannot be considered the primary cause of tube extrusion.

Obstruction of the Lumen Causing the Tube to "Pop Out."—In this series, several tubes became plugged with dried blood, serum and debris. However, unless secondary infection developed, the tubes were not rejected for this reason.

Tissue Reaction to the Prosthetic Material.—Foreign body reaction to the prosthesis material has commonly been blamed for its rejection by the eardrum. To retard this eventual rejection, tubes with flanges were designed to provide greater resistance by their broader contact with the medial surface of the tympanic membrane. Evidence in Table 1 indicates that tissue rejection of the prosthesis is a factor, because in this series the more inert the material, the longer it remained in place.

If tissue reaction to the prosthesis were the main rejection factor, then tubes placed in the anterior part of the eardrum should be rejected from the same site. They are not. When tubes are observed at regular intervals after insertion, it is apparent that they are not rejected but follow an orderly course across the eardrum, eventually to be "pulled out" by the migratory action of the squamous epithelial elements as the tubes pass from the tympanic membrane onto the canal wall at the annulus. Grommet-type or flanged prosthetic tubes placed through the drum will invariably follow an anterior to posterior course during the process of extrusion. I have never seen a flanged tube of any *mass* move off the eardrum in an anterior or su-

Eventually, all transtympanic ventilating tubes are pulled out of the ear by the forces of epithelial migration. After study of extrusion factors in 218 cases, an attempt was made to stabilize the tube by affixing it to the malleus. In 12 such cases, results after one year are encouraging.

J. L. TURNER, M.D.

TABLE 1.—*Time Before Extrusion of Various Types of Flanged Tubes in 218 Cases.*

	Polyethylene	%	Teflon	%	Silastic (Donaldson)	%
Number of ears	40		62		116	
Time (weeks)						
Average	10.9		24		23.9	
Range	4-10		4-68		6-48	
Majority of cases	6-8	55	8-16	51	20-26	52

perior direction. Straight, lightweight tubes of polyethylene have been observed to follow such a course. These findings tend to correlate with the somewhat divergent reports of Stinson[3] and Litton[4] regarding the paths of epithelial migration over the surface of the tympanic membrane.

Epithelial Migration

The process of removal of foreign bodies, both those on the surface of the eardrum[5] and those that have penetrated through the eardrum[3], by the migratory action of squamous epithelium has been known for many years. First described by Stinson[3] and later recalled by Litton[4], this phenomenon of peripheral and lateral growth of squamous epithelial elements from the center of the eardrum has become increasingly recognized as a factor which is necessary not only for prevention of disease in the normal external meatus, but a characteristic which must be duplicated in any plastic repair of the tympanic membrane or external canal.

Stinson[3] stated that epidermis migrated in its original plane anterior to posterior, except around the short process of the malleus where it proceeded in whorls, and over the pars flaccida where it fans out radially. He gave no exact rate of migration, saying that it was not uniform but was most rapid in the posterior and inferior quadrant.

Litton[4] concluded from his experiments

TABLE 2.—*Extrusion of Tubes After Infection.*

Tube Material	Infections	Associated Extrusion Yes	No
Silastic	26	8	18
Teflon	14	8	6
Polyethylene	6	6	0
Total	46	22*	24†

*During or shortly after infection.
†Tubes remained in place for average time or longer.

that migration proceeded from the umbo in a radial manner at an average rate of .05 mm. per day. Storrs[6], in testing his total eardrum fascia graft reconstructions, used a lacquer dot placed at the estimated center of the reconstructed drum and observed that the dot moved posteriorly at an average estimated rate of .12 mm. per day. The rapid rate, more than twice that of Litton's experiments, was attributed to an increased vascularity of the new tympanic membrane.

I hoped that forces existed in the patterns of epithelial migration reported by Stinson, Litton and Storrs which could be utilized to delay the eventual extrusion of a prosthesis by balancing the current of migration against the gravitational pull of the prosthesis mass and the drag of the flanged portion of the tube against the medial surface of the eardrum. Since there was some difference of opinion as to paths and rate of migration, further investigation was begun. The lacquer dot meth-

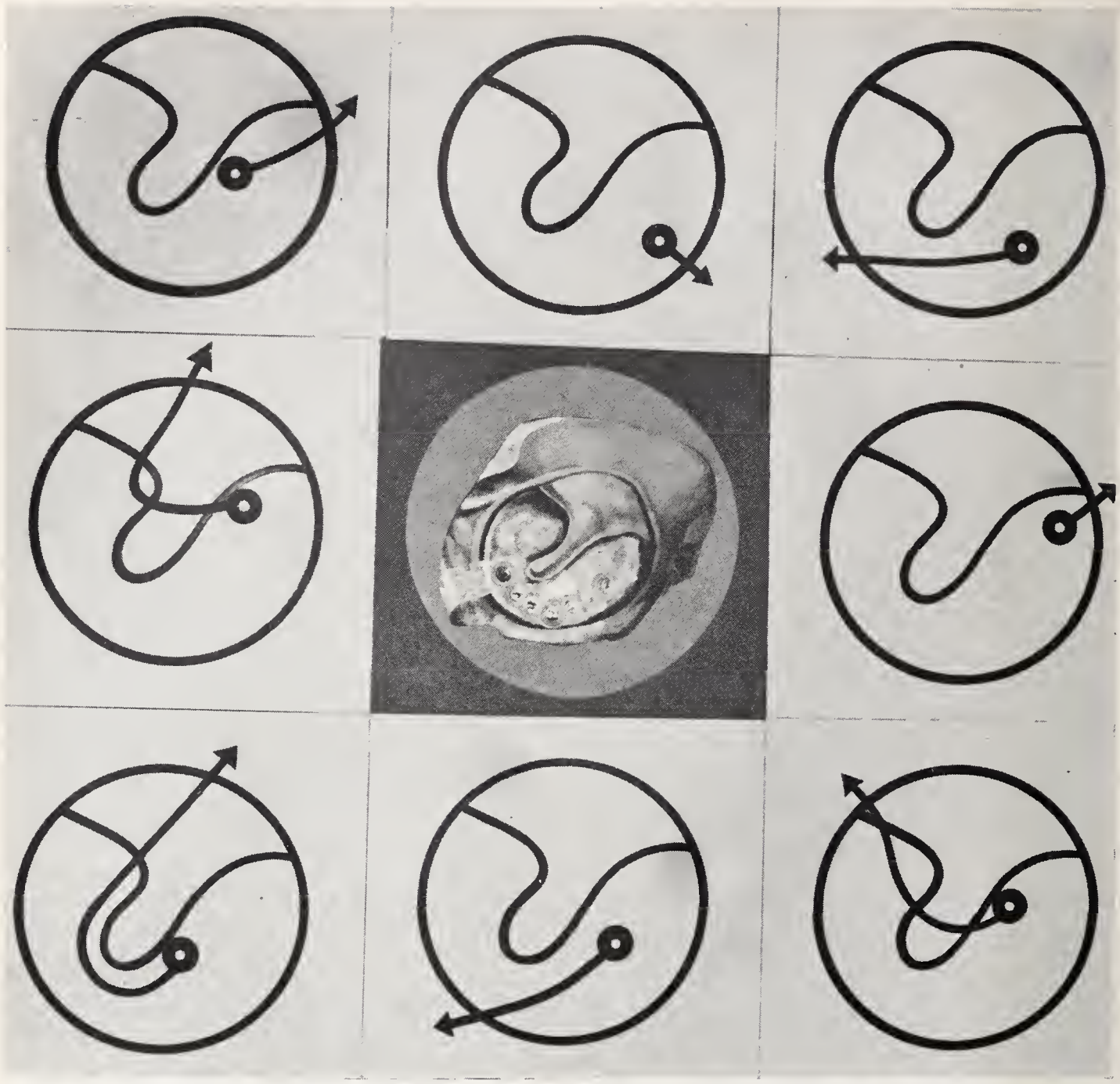

Fig. 1. Migratory patterns observed when lacquer dots were placed on various areas of the tympanic membrane surface. In the center, a multi-exposure illustrates the usual posteriorly directed pathway of extrusion of a "collar button" type prosthesis placed through the anterior-inferior segment of the eardrum.

od of Storrs was used because lacquer has greater mass than ink, and also has the ability to penetrate the deeper epithelial layers because of its acetone content. The dots were placed in the areas of predominant interest for this study, the anterior superior quadrant and the region immediately anterior to the handle of the malleus. Forty ears were so tested. The patterns of migration are shown in Fig. 1. Both the whorl patterns reported by Stinson and the centrifugal paths demonstrated by Litton were seen repeatedly. In all cases, however, there was a strong tendency toward posterior migration whenever the equator of the eardrum was approached.

Placement of Tubes

To make use of these observed migratory currents, all tubes in a second series of cases were placed in the extreme anterior superior quadrant of the eardrum *or* just adjacent to the anterior one-third of the malleus handle. Silastic tubes were used exclusively.

By comparing records of previous rates of extrusion in cases of replacement, and differences in rates of extrusion in cases of initial bilateral tube insertions, it has been shown that pre-malleus or anterior superior placement delays extrusion by an additional 6 to 12 weeks. It is thought that the anterior

superior pull of the epithelial migration currents in these areas temporarily counterbalances the gravitational pull on the mass of the prosthesis and delays its eventual entry into the predominant posterior migratory stream. A similar explanation can be made for the behavior of tubes placed immediately adjacent to the anterior one-third of the handle of the malleus. Here the whorl-like, superiorly directed pattern of migratory action causes them to abut and hang against the malleus until once again the gravitational pull puts them into the posterior stream. Repeated observations of prostheses placed in these areas confirm this hypothesis.

Permanent Myringostomy

The additional weeks of pressure equalization afforded by strategic placement of transtympanic ventilating tubes have been advantageous, but are still not enough. Following extrusion of the tubes, the recurrence rate of middle ear effusion in this series was 34 percent. In other series, it has been reported as high as 40 percent.

Experience with transtympanic ventilating tubes has shown that as long as they remain in place they are capable of correcting for eustachian tube dysfunction. For the patient whose chronic middle ear effusion resists any and all modes of treatment, a permanent substitute for the malfunctioning eustachian tube is necessary.

Since all tubes, regardless of design and placement, are eventually extruded by epithelial migratory action, some point of fixation is necessary if they are to resist this pull. An initial attempt was made to produce a permanent myringostomy by fixing a prosthesis to the handle of the malleus, since that is a logical point for such an attachment.

A Silastic tube of a modified Donaldson design was fashioned with a wire loop at its waist. The incision through the drum circumscribed the handle of the malleus. The tube was placed anterior to the malleus and the loop manipulated as far up the handle as possible. A few pieces of Gelfoam were used to cover the incision and stabilize the prosthesis during the first few days of healing. Initial results in 12 cases, observed for one year, are encouraging. As yet, there has been no significant displacement of the tubes, and these and subsequent patients show no greater tendency toward complications than do those with transtympanic ventilating tubes placed free in the tympanic membrane.

Summary

1. In 218 cases of middle ear effusion, the only treatment was the use of transtympanic ventilating tubes. The various times before extrusion of tubes of different prosthetic materials are compared.

2. The migratory action of the squamous epithelial elements of the tympanic membrane surface is proposed as the main cause of extrusion of transtympanic tubes.

3. Patterns of migration of squamous epithelium as demonstrated by observed movements of lacquer dots over the surface of the eardrum are described. Results in utilization of the forces present in this migratory action in the placement of transtympanic tubes is reported.

4. Recurrence of effusion followed extrusion of the ventilating tube in 34 percent of the cases.

5. An initial report is made of attempts to produce a permanent myringostomy by fixing a prosthetic transtympanic tube to the handle of the malleus.

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Intracranial Complications From Otitis Media

CASE REPORTS

THE INCIDENCE of intracranial complications from acute and chronic otitis media has been reduced as a result of introduction and widespread use of chemotherapeutic agents for bacterial infections. The availability of well trained otolaryngologists, improved transportation facilities, and the public's health awareness have also influenced the incidence of these complications. The serious nature of intracranial problems and their occurrence in spite of improved health measures, however, suggest that a review of the clinical features and management of each complication would be timely. The diagnostic features of various types of intracranial complications from otitis media are presented, as well as our experiences with several of these cases that have been seen in the Baylor Affiliated Hospitals Program during the past four years.

Background

Since the time of Hippocrates (460 B.C.), it has been recognized that acute infection of the middle ear is associated, at times, with intracranial problems and death. However, it was not appreciated until the 1700's (Morgagni) that the ear infection preceded the intracranial complication. Since that time, the clinical features and natural history of each type of intracranial complication have been well documented.

Until the introduction of chemotherapeutic agents for infection, the incidence of intracranial complications was reported as 3 percent of all patients with suppurative disease of the middle ear. Two-thirds of these complications occurred in patients with chronic otitis media. Although the overall incidence of otogenic intracranial complications today is reduced, Dawes[1] reports that chronic ear disease continues to be responsible for the same proportion of them.

The mortality from these problems has also been reduced. In a recent report of complicated ear disease cases at the University of Michigan, Proctor[2] observes that before antibiotics, 39 of 129 patients with intracranial complications died, but only one death occurred in 27 similar cases since the introduction of antibiotics.

Pathologic Mechanisms

Several factors influence the development of an intracranial complication from otitis media. Undoubtedly, the type and virulence of the organism, its susceptibility to available chemotherapeutic agents, the adequacy of therapy, and host resistance are important considerations. An associated chronic systemic illness (such as diabetes or leukemia) may influence the development of one of these complications.

Several pathways for the intracranial extension of infection have been described. In acute ear infections the usual mechanism of spread is by the progressive thrombophlebitis that is often associated with infection. This permits the inflammatory process to spread through intact bone. Another frequent mechanism of extension into intracranial structures is bone erosion, which exposes the intracranial structures directly to the inflammatory disease process. The bone erosion method of spread is more frequent in chronic

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Features, clinical examples, and management of intracranial complications from otitis media are presented. Since classical signs and symptoms may be masked by drugs which control infection, the clinician must be alert for more subtle indications that the pathologic process is extending toward the brain.

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ear diseases (for example, cholesteatoma); however, it also is the mechanism in acute coalescent mastoiditis. Another manner in which infection may spread is by extension along a preformed pathway such as the round window, dehiscent sutures or jugular bulb, skull fracture, or a surgically created pathway (fenestration, stapedectomy, or mastoidectomy with dura exposure).

Types of Intracranial Complications

The three major types of otogenic intracranial complications are meningitis, venous sinus thrombophlebitis, and brain abscess. Other types are extradural abscess, perisinus abscess, subdural abscess, and otitic hydrocephalus.

Meningitis and venous sinus thrombophlebitis account for the majority of all complications. In 199 cases during the years 1944-1960, reported by Dawes[1], 195 had meningitis or venous sinus thrombophlebitis or both. His figures emphasize that more than one complication may occur in the same patient. During the past four years, in the Baylor Affiliated Hospitals Program, nine patients with intracranial complications of otitic origin were selected for special study. Four of the patients had complications associated with chronic middle ear disease. In the other five patients the complications developed from acute otitis media. There were 13 complications among these patients. Table 1 summarizes the complications and deaths that occurred.

Clinical Features and Case Reports

Extradural Abscess.—In patients with otitis media, the development of an extradural abscess may go unnoticed until it has produced other complications. The presence of pulsating purulent drainage from an ear suggests this possibility. Headache and low grade fever may be other symptoms. Signs of localized meningitis may be present.

A 57-year-old woman with senile dementia developed otorrhea and later became comatose with fever, irregular respiration, positive Babinski's sign, and nuchal rigidity. Ear culture contained a coagulase-positive *Staphylococcus aureus*. Cerebrospinal fluid studies revealed 3,370 leucocytes, low sugar and high protein levels, but no microorganisms on Gram's stain smear or culture. This patient was treated with antibiotics, and a mastoid exploration was performed. At surgery an epidural abscess was found. She made a rapid recovery clinically.

This patient had localized meningitis as a result of bone erosion by infection in the tegmen area of the ear. Mastoid exploration in extradural abscess is an effective means of determining the pathological problem that exists and of eradicating it. Direct surgical treatment of this complication will lessen the likelihood that generalized meningitis or other intracranial problems will develop.

Meningitis.—This must be suspected when patients with otitis media develop headache, fever, irritability or drowsiness, vomiting, or convulsions. Clinical signs include neck stiffness or opisthotonus, positive Brudzinski's or Kernig's sign, or both, convulsions, and later paralysis and coma. As intracra-

TABLE 1.—*Intracranial Complications from Otitis Media in Nine Patients. Baylor Affiliated Hospitals, 1962-1966*

	Complications	Deaths
Meningitis	7	3
Sinus thrombophlebitis	2	
Brain abscess	1	1
Extradural abscess	2	
Otitic hydrocephalus	1	

nial pressure rises, the pulse may slow, blood pressure rise, respirations become irregular, and projectile vomiting appear. Spinal fluid studies demonstrate grossly cloudy spinal fluid with numerous polymorphonuclear leucocytes, reduced sugar level, and evidence of microorganisms on smear and culture.

The following two cases illustrate the clinical features in meningitis. In one patient, meningitis was associated with acute ear disease. In the other, a chronic ear disease had existed for years before meningitis occurred.

A 67-year-old man had onset of otitis media one week prior to admission to Methodist Hospital. He had been treated for three days with antibiotics. Two days after he discontinued therapy, he had onset of headaches, fever, neck stiffness, mental confusion, and purulent exudate from the right ear. Brudzinski's and Kernig's signs were positive. Cerebrospinal fluid contained 6,000 leucocytes and gram-positive diplococci. Spinal fluid sugar was low and protein elevated. The ear culture was *Diplococcus pneumoniae*. This patient was treated with large doses of penicillin intravenously and intramuscularly (23 million units per day). He made a slow clinical improvement; however, mental confusion persisted for several weeks. Initially, he had severe hearing impairment bilaterally but gradually regained useful hearing in the left ear (Figs. 1 and 2).

A 55-year-old man with a ten-year history of bilateral otorrhea was admitted to Jefferson Davis Hospital with mental confusion, temperature 101 F., nuchal rigidity, bilateral cholesteatoma, and hyperactive reflexes. Cerebrospinal fluid contained 6,000 leucocytes (70 percent polymorphonuclear), low sugar and elevated protein. Although gram-positive cocci were identified in the spinal fluid, culture showed the organism to be *Proteus vulgaris*. The patient was treated with large doses of several antibiotics, including intrathecal kanamycin and colistin. Bilateral mastoidectomies were done. Erosion of the tegmen was found in the right ear and the dura had granulations on its surface. The patient continued to have meningeal signs and positive cerebrospinal fluid cultures. He died six weeks after admission. At necropsy, basilar meningitis was the prominent finding.

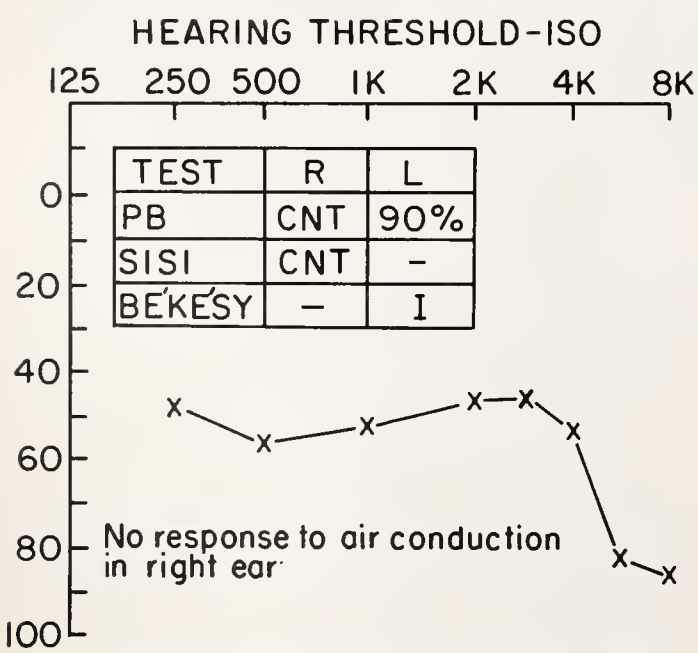

Fig. 1. Hearing threshold 23 days after onset of meningitis in a 67-year-old man.

In acute otitis media with meningitis, surgery for the ear disease should be postponed until chemotherapeutic control of the infection is attained. At that time the usual indications for mastoid surgery should prevail; however, in patients who have the preformed pathways of skull fracture with cerebrospinal fluid leak, or of fistula of the labyrinth, surgery should be done for correction of these defects. The patient with chronic otitis media who develops meningitis should have surgical treatment for the middle ear and mastoid disease whenever his general condition permits. Often, associated bone erosion with extradural abscess or petrous bone abscess is responsible for the development of meningitis.

Sinus Thrombophlebitis.—The clinical features of sinus thrombophlebitis are often characterized by a progression of the symptoms and signs until papilledema, seizures, interference in cerebral circulation, and posterior mastoid swelling are apparent. When otitis media is recurrent or fails to respond to therapy, or when headache persists, this diagnosis should be considered. Early headache, fever, and malaise are the only symptoms. These are often modified when antibiotics have been administered. As the phlebitic process continues, an infected thrombus

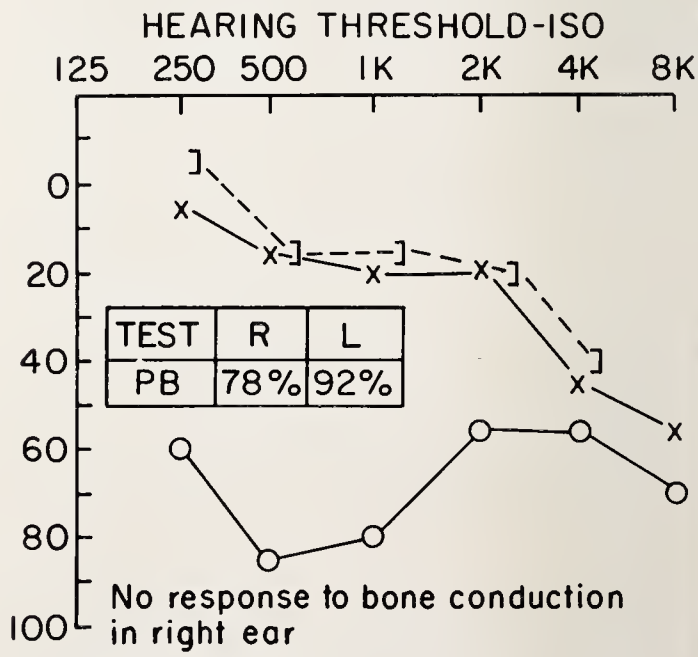

Fig. 2. Hearing threshold 95 days after onset of meningitis in the same patient.

may develop within the sinus and septicemia or obstruction to venous return may ensue, or both may occur. The obstruction to cerebral circulation results in increased intracranial pressure and edema over the posterior aspect of the mastoid process. The edema over the mastoid (Griesinger's sign) is associated with thrombosis of the mastoid emissary vein. Examination of the cerebrospinal fluid reveals an elevated pressure, but no organisms or biochemical abnormality.

This seven-year-old boy had a history of recurrent acute otitis media. Five days before admission to Methodist Hospital, he had onset of an intensifying right otalgia, lethargy, and irritability. Temperature was 104 F., and weakness of the right leg and arm appeared one day prior to admission. Right sided seizures began the day of admission. Cerebrospinal fluid studies disclosed no cells, normal protein and sugar levels, but elevated pressure. A carotid arteriogram demonstrated an impairment in cerebral circulation due to obstruction to outflow of the dominant right lateral sinus. Mastoiditis on the right side was apparent radiologically. Myringotomy was done, large doses of antibiotics given, and anticoagulants administered. Griesinger's sign appeared on the third day. Mastoidectomy was performed six days after admission. At surgery, the lateral sinus was involved with the inflammatory process. A slow clinical improvement followed.

Surgical exploration of the mastoid is valuable in patients with lateral sinus thrombophlebitis because diseased mastoid cells over the sinus can be removed and the phlebitic process curtailed. Unless patients have embolic phenomena, neither anticoagulants nor internal jugular vein ligation is recommended. At surgery, obliteration of the sinus is not performed as this prevents recanalization that may occur after the thrombophlebitic process subsides.

Brain Abscess.—In patients with otitis media, brain abscesses often develop subtly. The initial stage of encephalitis may go unnoticed; however, the onset of persistent fever, leucocytosis, headache, nausea, or vomiting should alert the clinician to the possibility. In cerebral abscesses, a convulsion may be one of the first signs. Ataxia may be the first clinical sign in a cerebellar abscess. Cerebrospinal fluid findings are elevated cells and protein, but normal sugar. The spinal fluid usually has no organisms in it. A quiescent stage often occurs in the clinical course of patients with brain abscesses. During this quiescent stage, which may last 10 to 14 days, there may be no symptoms. The third stage is associated with an expanding abscess. During this stage the patient has symptoms characteristic of increased intracranial pressure, such as violent headache and projectile vomiting. The pulse rate is reduced and respirations become irregular due to pressure on brain stem centers. Disorientation occurs. Later, convulsions, ocular paralysis, papilledema, and pupillary changes appear.

This 17-year-old boy had otitis media for four weeks prior to death. Ten days before admission to Methodist Hospital, he was hospitalized elsewhere for headache, vomiting, nuchal rigidity, and otorrhea. Cerebrospinal fluid studies at that time revealed elevated protein but normal sugar and no leucocytes. He was treated with antibiotics, became asymptomatic, and was discharged six days later. On the day of admission to Methodist Hospital, he had severe headache and fever, became delirious, developed moderate nuchal rigidity, and vomited. The otorrhea recurred. Babinski's sign was positive and a right gaze nystagmus and right facial weakness were present. Cerebrospinal fluid contained 26,800 leucocytes, and gram-positive cocci were seen on smear. The spinal fluid pressure was elevated. Large doses of antibiotics were administered, but the patient died of cardiorespiratory failure 12 hours after admission. Necropsy revealed a right cerebellopontine angle abscess, right cerebellar abscess, and basilar meningitis. Temporal bone specimens revealed a right petrous abscess with perforation into the posterior fossa, and hyperplastic middle ear mucosa with inflammatory disease in the middle ear and mastoid air cells (Figs. 3 and 4).

The quiescent phase in the clinical course of brain abscess must be recognized. Whenever transient symptoms and signs suggest encephalitis or localized meningitis, careful clinical and laboratory evaluation is necessary. Any abnormality on neurological examination or cerebrospinal fluid study (especially increased protein) deserves investigation. In this patient, the quiescent phase was prominent. Death was caused by rupture of the brain abscess with resultant overwhelm-

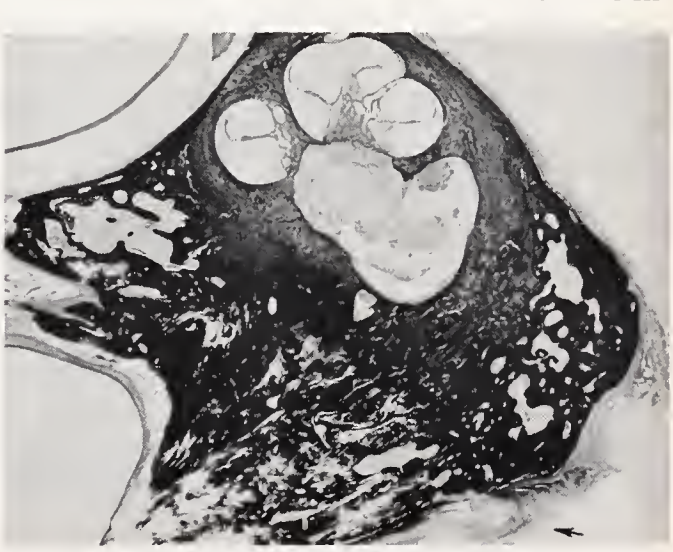

Fig. 3. Temporal bone section shows inflammatory cells, internal auditory meatus. Arrow points to posterior fossa abscess. X 2.66.

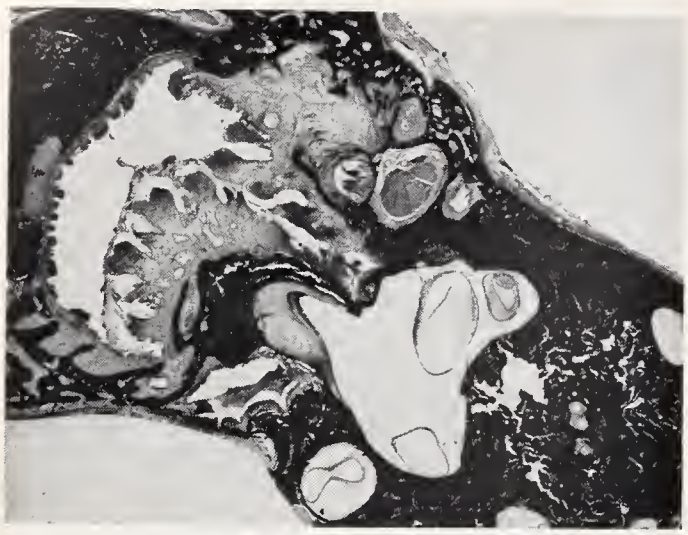

Fig. 4. Temporal bone section shows inflammatory disease of the mucous membrane, middle ear. Section is through the stapes footplate-vestibular area labyrinth. X 3.

ing meningitis. Neurosurgical treatment for brain abscess offers the only chance for cure.

Otitic Hydrocephalus.—The most common symptom in otitic hydrocephalus is headache. Papilledema is often present. Diplopia due to involvement of the sixth cranial nerve is reported as an associated symptom. Cerebrospinal fluid studies are cytologically and biochemically normal but pressure is elevated. Often, the otologic disease process has improved considerably when otitic hydrocephalus appears.

This 39-year-old man, with a history of chronic ear disease of many years' duration and previous mastoidectomy, had onset of severe headache, blurred vision, and otorrhea. Examination also revealed papilledema. In spite of specific chemotherapeutic treatment, his symptoms persisted. Cerebrospinal fluid pressure was elevated to 400 mm., but sugar and protein values were normal and there were no leucocytes in the fluid. Arteriograms were normal. A right mastoid exploration was performed five days after admission to Methodist Hospital. A cholesteatoma was found in the mastoid antrum; however, the tegmen was intact. The lateral sinus was not thrombosed. Postoperatively the symptoms disappeared. Papilledema gradually subsided and spinal fluid pressure decreased.

Otologic surgery for otitic hydrocephalus may not be indicated. However, when otitis media is present, the differentiation of otitic hydrocephalus from lateral sinus thrombophlebitis is often difficult. The use of antibiotics in middle ear and mastoid infections may mask the symptoms of thrombophlebitis. Thus, surgical exploration is often necessary to establish the diagnosis.

Conclusions

The introduction of chemotherapeutic agents for infection has reduced the incidence and mortality of intracranial complications of otitic origin. The physician must remember, however, that these agents may mask the classical symptoms and signs of the various complications.

The key to successful management is, in part, the alertness of the clinician, who must be on guard for the subtle signs and symptoms that are suggestive of these complications. Careful clinical observation of each patient with acute or chronic otitis media or mastoiditis is necessary in order to diagnose an intracranial complication early.

In following the clinical course of patients with otitis media the clinician should pay special attention to elevation of temperature, irritability or drowsiness, persistent headache, nuchal rigidity, or any positive neurological signs. Abnormal cerebrospinal fluid pressure, cytology or chemistry deserves thorough investigation. Also, it is imperative that the physician realize that more than one intracranial complication may occur in the same patient.

In treating patients with acute or chronic otitis media, an adequate therapeutic program must be carried out. This will consist of antibiotic administration and often otologic or neurosurgical operative procedures. Systemic bactericidal drugs are more effective than bacteriostatic ones and may reduce the incidence of otogenic intracranial complications. When an associated systemic illness accompanies otitis media, adequate therapy of that condition is extremely important. Surgical therapy for ear disease is an important consideration when these complications appear imminent. Otologic surgical procedures are necessary when lateral sinus thrombophlebitis, extradural abscess, or subdural abscess is suspected. Neurosurgical exploration is necessary in suspected brain abscesses. The need for surgery in the patient with meningitis must depend upon the clinician's judgment; however, in certain instances surgery is of value, especially in controlling the local ear disease.

ACKNOWLEDGMENT

The authors wish to thank Dr. Ben M. Nail, Jr., who permitted us to report the case of otitic hydrocephalus.

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Orbital Blowout Fracture

A "Black Eye"

C. Roger DeHaven, M.D., and Thomas S. Harle, M.D.

Patients treated for black eyes should also be examined carefully for evidence of an associated blowout fracture of the floor of the orbit. Failure to detect and repair these fractures constitutes a "black eye" for the physician as well.

CASE REPORTS

BLOWOUT FRACTURES of the floor of the orbit have been giving black eyes to many people for many years. Unfortunately blowout fractures are also giving a "black eye" to many physicians as well, simply because they are not aware of the blowout fracture entity. Only in the past ten years has the medical profession realized the importance of making this diagnosis early and instituting early operative correction in order to prevent later complications. Many such fractures are overlooked during examinations and treatment in emergency rooms. The clinical findings in seven cases of blowout fractures treated during the past two years are described.

Mechanism of Injury

This fracture is almost invariably caused by a large blunt object, such as a fist or baseball, striking the eye and creating a rapid increase in pressure within the orbit (Fig. 1). This phenomenon was clearly demonstrated by Smith and Regan[1] who, using a cadaver, placed a baseball over one orbit and produced a blowout fracture by striking the ball with a hammer. The opposite bone could not be fractured after the orbital contents had been exenterated and the soft tissue surrounding the orbital rim had been removed. This supports the hypothesis that orbital blowout fractures are more a "hydraulic" fracture than a direct bone transmission type. The fracture usually occurs in the thin bone of the orbital floor in the area of the infraorbital canal, and frequently is quite small. Orbital contents usually are incarcerated firmly into the fracture site.

Clinical Diagnosis

In most cases the clinical diagnosis is simple. The patient usually comes to the emergency room with a black eye and any of the following:

1. A black eye with subconjunctival hemorrhage, edema, and periorbital ecchymosis are usually the first objective signs.
2. Diplopia and limitation of vertical ocular movements are classically present because of limitation of movement of the incarcerated inferior rectus or inferior oblique ocular muscles or both.
3. Anesthesia in the distribution of the infraorbital nerve is a frequent complaint, and, if not noted by patient it should be sought out by the physician. This area extends from the cheek bone to the lateral side of the nose down to the upper portion of the lip on the involved side.
4. Roentgenographic evidence of fracture.

Any of the first three findings is an indication for x-ray studies. Obviously, routine

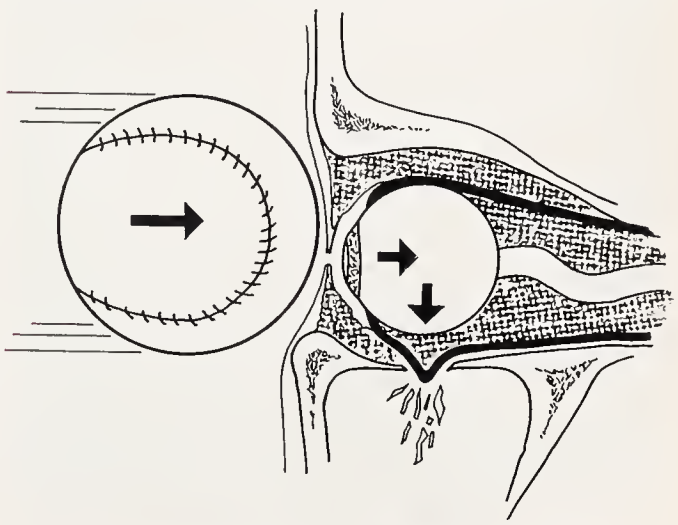

Fig. 1. Mechanism of injury. A sudden increase in intraorbital pressure produces blawaut in the weakest partion af the orbital floor.

ocular examination should also be carried out with particular attention to the globe, vision and ophthalmoscopic findings.

Roentgenographic Findings

Waters' views permit good evaluation of the orbital floor, and also give a good look at the zygoma, maxillary antrum and temporal arches. The Caldwell projection gives a clearer view of the orbital margin and frontal and ethmoid sinuses. Roentgenograms usually show a cloudy maxillary sinus or thickening of the mucosa in the superior portion of the antrum (Fig. 2). Laminagrams of this area usually delineate the herniated orbital structures more clearly (Fig. 3). It is particularly important for the clinician to be aware that a fracture may exist and then provide the radiologist with sufficient information for an adequate diagnostic study.

Importance of Early Diagnosis

Early diagnosis depends upon the alertness of the physician. When examining any black eye, the physician should look for any of the common clinical signs of orbital blowout. Early diagnosis is of the utmost importance since surgical correction is much simpler if performed within seven to ten days after injury. If the diagnosis is missed, the patient frequently develops irreversible cosmetic and functional defects.[2] The globe becomes sunken and frequently the eyelid droops on the enophthalmic side. Diplopia is a serious problem because the eyes are no longer on the same horizontal plane, and frequently there is considerable loss of orbital fat around the globe, secondary to herniation and atrophy.

Fig. 2. Case 2. Waters' view reveals a broad, round prolapse of orbital soft tissues into the upper half of the left antrum through a fracture of the orbital floor.

Treatment

It is not the purpose of this article to dictate treatment. Converse and Smith[3] believe that some type of permanent orbital floor fixation is superior to temporary support attained by packing the maxillary antrum. They state that although the antral pack restores the level of the sagging floor, orbital contents will continue to adhere to the fracture site unless a bone graft or similar structure is utilized to cover the defect.[4] Others feel that simple antral packing is sufficient. We think that each case must be evaluated and treated accordingly. In cases where a small fracture is combined with slight restriction of vertical movement and minimal antral clouding, simple reduction of the frac-

Fig. 3. Case 2. Laminagram reveals the same soft tissue density of the orbital structures.

ture and support from either an autogenous bone fragment or a synthetic material is sufficient. In more severe injuries where fat and orbital contents have herniated through a large comminuted defect, support from the maxillary antrum will be necessary in addition to an orbital implant. We have utilized a silicone rubber buttress in several cases, with excellent postoperative results.

Case Reports

Case 1.—A 25-year-old white man was admitted May 25, 1963, several hours after being struck in the right eye with a baseball. He had considerable edema and ecchymosis around the periorbital tissues, with mild subconjunctival hemorrhage, but no anesthesia in the distribution of the infraorbital nerve. Visual acuity was 20/20 in each eye without correction. Funduscopic examination showed no abnormalities. Two prism diopters of right hypotropia in the primary position accentuated in the upward position was noted. Traumatic iritis was present. Roentgenograms revealed clouding of the right maxillary sinus with a small area of increased density, which had appearance of bone, in the upper portion of the right maxillary sinus. Surgery was performed six days later; general anesthesia was used. A small blowout was found medial to the infraorbital groove. The fractured bone chips were easily elevated into normal position; however, they were unstable after reduction, and a 15 mm. square piece of silicone rubber, 2-3 mm. thick, was used to bridge the fracture site. It was held in place by suturing the periosteum at the orbital margin and closing subcutaneous tissues and overlying skin. Postoperatively the vertical deviation resolved, and one week after surgery roentgenograms revealed reduction of mucosal swelling in the upper part of the maxillary sinus.

Subsequently, the silicone implant dislodged; the patient felt a hard, palpable foreign body in the right infraorbital area, and was readmitted on Sept. 4, 1964 (4 months later). The silicone was permanently removed. At this time, the orbital floor was healed, with apparent bony union and overlying fibrous tissue beneath the silicone. There was minimal reaction around the silicone, and the sheet slipped out without difficulty. Sinus roentgenograms at this time were entirely within normal limits.

Case 2.—This 24-year-old white man was attacked and beaten on June 16, 1963. He was hospitalized elsewhere and treated with hot packs, x-rays, and an oral enzyme to reduce swelling around his left eye. When I saw him two weeks later, he had blurry vision and numbness in the left eye, cheek, and left side of his face. The blurry vision was caused by poor accommodation in the left eye, probably due to prior use of cycloplegics. Visual acuity was 20/20 in each eye without correction. Ocular motility was normal in primary position, but the red glass cover test revealed diplopia due to left hypotropia in upward gaze. Anesthesia in the distribution of the left infraorbital nerve was present. Laminagrams (Figs. 2 and 3) revealed well delineated depression of the orbital floor. At surgery on July 5, 1963, we found a large defect in the orbital floor which involved the infraorbital groove. A silicone rubber buttress was used as in case 1. Convalescence was normal and the haze in the left maxillary sinus gradually cleared.

Two months after surgery the patient complained of a pressure sensation in his left orbit whenever he lifted weights, and he also felt a popping sensation when he blew his nose. We thought that there was poor bony union in the floor of the orbit, and operated again on Oct. 3, 1963. The silicone was removed. An opening between the orbit and the left maxillary antrum approximately 2 mm. in diameter was noted. The silicone implant was easily removed, and the hole in the orbital floor was bridged with a piece of autogenous iliac crest bone. The postoperative course was normal and the symptoms have not recurred. If the patient had been given another two to three months prior to the second operation, the bony defect probably would have healed spontaneously. Roentgenograms in December, 1963, revealed satisfactory reduction of the left orbital floor fracture with a slight haziness in the upper portion of the left maxillary sinus probably representing residual mucosal reaction.

Case 3.—A 23-year-old white man had double vision after he got a black eye (right) in a fist fight on Sept. 14, 1963. Contusion and ecchymosis were present in the periorbital tissues about the right eye, but no anesthesia in the distribution of the infraorbital nerve was found. One diopter of right hypotropia was found in primary position, and red lens test revealed increasing amounts of right hypotropia in upper gaze. Preoperative roentgenograms showed depression of the right orbital floor into the upper portion of the maxillary sinus. Tomograms confirmed the blowout fracture. Four days after injury, a small trap-door fracture, medial to the infraorbital groove and with incarceration of muscle, was repaired. The incarceration was reduced and the fracture segments snapped back into place. Only a small skinhook was used for elevation; no supporting implant was necessary. Postoperatively, transient infraorbital anesthesia was present, but cleared one month after surgery. The patient had no vertical imbalance in November, 1963, and roentgenograms on the same date revealed satisfactory reduction of the fracture and a clear, normal maxillary sinus.

Case 4.—This 18-year-old white boy was struck in the left eye with a fist on Nov. 30, 1963. The following day he had moderate periorbital ecchymosis and edema, and could not move his left eye upward or downward. There was anesthesia in the distribution of the left infraorbital nerve. Visual acuity in the right eye was 20/20, and in the left, 20/30 without correction. Roentgenographic density in the left maxillary sinus, superiorly, was thought to represent a depressed fracture of the floor of the orbit. Laminagrams were not obtained. On Dec. 10, 1963, direct approach through the lower lid exposed the orbital floor. A moderate sized fracture through the infraorbital groove extended toward the nose. The soft tissues were partly incarcerated into the maxillary antrum and could not be reduced from above; therefore, a Caldwell-Luc approach was used. Manipulation from both sides of the fracture site enabled us to replace the orbital tissues. The maxillary antrum was packed with a tight petroleum jelly

gauze dressing which was brought out through a nasoantral window. The pack was removed one week after surgery. The patient continued to have weakness in upward and downward gaze, however, restriction of gaze was considerably less than prior to surgery. One year later the patient was free of symptoms, the eyes were orthophoric, and roentgenograms were normal.

Case 5.—This 20-year-old white man was struck in his left eye with a fist on Feb. 9, 1963. When seen three days later, the left side of his face was numb and he had double vision. Visual acuity was 20/20 in each eye without correction; only slight ecchymosis of the left lower eyelid was apparent. He had two prism diopters of left hypotropia in primary position. This became more severe when he gazed upward during the red glass test. Anesthesia in the distribution of the left intraorbital nerve was noted. Increased density in the superior portion of the left maxillary sinus, seen on roentgenograms, was thought to represent a depression of the floor of the left orbit. On Feb. 14, 1963, an orbital floor fracture through the infraorbital groove area was found at surgery. It was a trap-door fracture which was easily swung back into place after reduction of incarcerated orbital tissue. However, the fracture was unstable, and a small piece of iliac crest bone was placed over the fracture site for support. Postoperatively the patient did well and no vertical imbalance could be identified six weeks after surgery. Roentgenograms obtained in April, 1963, revealed a slight area of increased density in the roof of the left maxillary sinus. There was no depression of the orbital floor and sinuses were otherwise clear. Normal sensation returned to the left side of the face.

Case 6.—A white man, 21, was struck in the left eye in April, 1960, while he was stationed in Germany. Orbital floor fracture was not diagnosed, however, until he was evacuated to the continental United States several months later. At that time the orbital floor was exposed through an incision in the inferior fornix, and incarcerated tissues were found and supposedly replaced in normal position. The orbital floor fracture, however, was not repaired and no orbital floor support was utilized. Immediately after the operation, it was noted that the patient continued to have total inability to look upward with his left eye. The ophthalmologist advised that the patient should be returned to the operating room for repair of the fracture. However, for some reason the patient was transferred to another installation and surgery was never done. I first saw him in June, 1963; at that time he was totally unable to look upward, but there was no deviation in primary position. On June 27, 1963, surgery was performed to repair the orbital floor. Approach was through the skin fold below the left eyelid. Considerable incarceration of orbital tissue extended down through a large fracture in the orbital floor. Another approach through the inferior fornix was also used. The inferior rectus was grasped with a muscle hook in order to keep it taut while dissection through the skin incision was carried out. Considerable scarring was encountered between conjunctiva and globe and between the inferior rectus muscle and the orbital floor. The muscle was also adherent to the globe. The orbital contents adhered to the contents of the left maxillary antrum and could not be pulled out of the sinus cavity without sharp dissection. A Caldwell-Luc approach was utilized and the depressed orbital floor was refractured, elevated into normal position, and held in place by a petroleum jelly gauze pack which was brought out through the nasal antrum window. The bony defects on the orbital site were bridged with a layer of silicone rubber to prevent re-attachment of the inferior rectus muscle to this area. Four hours after surgery, the patient ripped out the packing and pulled the bandages from his eye. Within 48 hours, considerable conjunctival edema was observed. Approximately six weeks after surgery he was reexamined and was no better than he was prior to surgery. He was still unable to elevate his left eye above primary position. Amazingly, no enophthalmos occurred in this patient.

Case 7—A 13-year-old boy was struck in the right eye by a fist on April 21, 1965. Moderate swelling was present. No anesthesia was present, but six prism diopters of right hypotropia in primary position was measured, and increased in upward gaze. Roentgenograms indicated a blowout fracture. One week later the fracture was repaired and a silicone buttress was inserted. At the last visit, June 8, 1965, only two prism diopters of left hyperphoria could be measured; it was asymptomatic.

Summary

Blowout fracture of the floor of the orbit should always be suspected in any patient who has a black eye. A routine check for (1) numbness of the cheek on the involved side, (2) double vision in upward gaze, and (3) clouding of the maxillary antrum on Waters' view strongly suggest this entity, and early referral to an ophthalmologist for definitive care is indicated. The treatment is always surgical restoration of the level of the orbital floor and releasing the adherent orbital contents. Occasionally good correction can be accomplished by a direct orbital approach alone; frequently, a combined orbital and Caldwell-Luc approach with antral packing is necessary in order to maintain the reduction.

Early diagnosis is the most important step necessary for complete restoration of ocular function and preservation of normal cosmetic appearance.

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Tracheostomy
A Major Consideration

R. Maurice Hood, M.D.

In five years, 187 tracheostomies were performed in one hospital; mortality was 4.8 percent. The corresponding figure for appendectomy was .78 percent. On this basis, tracheostomy is a major operation; its consideration as such also is warranted by the finding that most resultant deaths were preventable. Indications, contraindications, technique, and alternative measures are reviewed.

REVIEW ARTICLES

TRACHEOSTOMY is an operation the origins of which are lost in antiquity. It was described by the Egyptians and was well known in the age of Hippocrates. Its use in ancient times, in fact, until less than a quarter-century ago, was reserved for relief of obstruction of the larynx or pharynx. At the latter period, tracheostomy was usually resorted to as a dramatic emergency procedure to relieve obstruction of the larynx from diphtheria and occasionally from foreign body aspiration.

In sharp contrast, the current status of this operation is such that it is utilized frequently and performed under almost any circumstances by any physician. The indications for its use have become legion and ill-defined. One may question, at times, whether there is any justifiable indication at all.

Having become increasingly aware of the number of fatalities and major complications associated with this procedure[1-4], I studied the records of patients who had tracheostomies at Brackenridge Hospital during five years. The cases in which death or significant complications occurred were critically evaluated.

Indications and certain contraindications for tracheostomy are reviewed, and surgical principles which can be applied to this operation are reiterated.

In five years, 187 tracheostomies were performed. No attempt was made to determine the indications for surgery. Charts of 81 patients who died during the hospital stay established that 9 patients died as a result of tracheostomy or of complications thereof. To give the figures some perspective, compare them with the number and the mortality rates for appendectomies done during the same period (Table 1). Appendectomy was chosen because it is always assumed to be a major operation and is so managed.

Operative mishaps have occurred in several instances as is shown in Table 2. The following reports are representative:

Case Reports

Case 1.—A 28-year-old white woman received a major head injury in an automobile collision; she remained comatose for ten days. Atelectasis and a

TABLE 1.—*Mortality.*

	Total	Deaths	%
Tracheostomy	187	9*	4.8
Appendectomy	1,540	12	0.78

*Attributable to operation.

TABLE 2.—*Major Technical Complications.*

	No.	Deaths
Injury of innominate artery	1	1
Injury of innominate vein	1	
Pneumothorax	2	
Pneumomediastinum	3	1
Injury of thyroid gland	1	
Postoperative bleeding	1	

pulmonary abscess developed, and a tracheostomy was done. Three days later there was sudden massive hemorrhage from the wound. At emergency operation the posterior wall of the innominate artery was found to be torn. Only temporary control was effected and the patient died.

Comment: The time interval indicates that the wall of the innominate artery was injured, but not completely ruptured, at the time of surgery.

Case 2.—A 36-year-old man sustained multiple rib fractures, producing instability of the chest wall. The day after injury, tracheostomy was performed while the patient was in his bed in the ward. During approach to the trachea, major bleeding occurred; it was controlled by pack. The injured vessel could not be identified until the patient was transferred to the operating room where adequate lighting and an experienced surgeon were available. The left innominate vein was torn and was repaired. Transfusion was required.

Comment: Inadequate lighting in the ward resulted in a major complication. The reason for not taking the patient to the operating room originally concerned convenience for the physician, since the tracheostomy was not urgently required.

Case 3.—A one-year-old boy had tracheostomy performed because laryngeal papillomatosis was producing respiratory obstruction. Respiratory distress continued and became worse after the operative procedure. A chest x-ray (Fig. 1) showed bilateral pneumothorax with almost complete collapse.

Comment: The intimate relation of the apex of the pleura to the midline structures of the neck in children is not always appreciated, particularly by the inexperienced surgeon.

In addition to technical accidents, a number of serious and lethal postoperative complications were encountered (Table 3).

Case 4.—A three-month-old infant, acutely ill, manifested severe respiratory stridor and cyanosis. There was a four-day history of fever and cough. A clinical diagnosis of acute tracheobronchitis was made and tracheostomy was done the same day. Postoperatively, the child did not improve. Irreversible atelectasis of the left lung developed, and the child died ten days after admission.

Fig. 1. Case 3. Chest roentgenogram shows bilateral pneumothorax and subcutaneous emphysema immediately following tracheostomy.

TABLE 3.—*Major Postoperative Complications.*

	No.	Deaths
Erosion of trachea and innominate artery	1	1
Atelectasis (infant)	3	3
Atelectasis: inspissated secretions	12	3
Cellulitis of neck	1	
Ulcerative tracheitis	1	

Comment: Even though a correct diagnosis was made, tracheostomy was incorrectly instituted as part of the treatment. As might be expected, the bronchiolar obstruction of the tracheobronchitis was not relieved and the resultant atelectasis was related to the presence of the tracheostomy tube. Both the diagnosis and age represented contraindications to surgery.

Case 5.—A 71-year-old white man was operated upon for bronchogenic carcinoma of the right main bronchus. Moderate diffuse emphysema was a complicating factor. Pneumonectomy was performed. Postoperatively, severe respiratory insufficiency was immediately apparent, manifested by dyspnea and cyanosis. Tracheostomy was done to reduce dead space, but produced no clinical improvement. The patient was then placed on the Mörch respirator and striking improvement was immediately noted. He was maintained on the respirator for nine days and therapy was being gradually withdrawn when on the 11th day, about 300 cc. of blood suddenly came through the tracheostomy tube. About six hours later the patient suddenly exsanguinated from the tracheostomy opening. Autopsy showed that the tip of the tracheostomy tube had eroded the anterior wall of the trachea and into the innominate artery.

Comment: The prolonged pulsation of the tracheostomy tube from the recoil of the respirator was responsible for the erosion. This accident may not have been avoidable.

Case 6.—A 50-year-old man underwent tracheostomy in the emergency room to provide access for aspiration of blood from the tracheobronchial tree. The patient had sustained a head injury and facial injuries. Four days later he was febrile and had edema and erythema of the entire cervical region. From purulent drainage material, a hemolytic streptococcus was cultured. Antibiotic therapy and drainage were required.

Comment: Although the trachea is not a sterile field, it is likely that this patient's infection was caused by poor sterile technique outside the operating suite. From the patient's clinical course as related to the injury, it is doubtful that the procedure was necessary.

A previous report[5] described the fatal consequence of obstruction of the trachea and bronchi by dried, encrusted bronchial secretions. Since that report, two additional cases of massive atelectasis due to this complication have been encountered. This brings to six the number who developed tracheal obstruction from encrusted secretions. There were three deaths from this cause.

TABLE 4.—*Indications.*

Obstruction
Access to secretions
To remove normal obstruction of larynx
To reduce dead space
To prevent tussive pressure
Postoperatively in patients with decreased compliance

TABLE 5.—*Contraindications.*

Tracheobronchitis
Infants under six months old
Acute respiratory decompensation (asthma-emphysema)
Apnea
Inadequate experience, lighting, or instruments
Lack of positive indication

Indications for Tracheostomy

Table 4 gives a practical classification of the indications for operation. Of those listed, obstructive lesions of the larynx or pharynx require little comment. The mechanism and extent of obstruction determine the need for operation and its urgency.

The need to aspirate tracheobronchial secretions is the most common indication for tracheostomy. Sometimes the need may be anticipated, for example, when an extensive operation about the head and neck is required. Here, tracheostomy should be done immediately. Except in such instances, other methods such as intratracheal aspiration, heated mist inhalation, and the use of mucolytic agents should be utilized until it is obvious that these measures will not suffice. Tracheostomy should not be used merely as a substitute for adequate professional and nursing care.

Tracheostomy to avoid the normal obstruction of the larynx and to reduce dead space has long been recognized as important in the management of the unstable chest wall resulting from multiple rib fractures. The use of positive pressure breathing devices has supplemented and even increased the number of tracheostomies performed for this condition.

Patients who have operative procedures on, or injuries involving, the trachea and major bronchi frequently need tracheostomy to prevent tussive pressure from disrupting suture lines or injured areas.

Spencer and others[7] have pointed out the advantages and indeed the lifesaving value of tracheostomy supplemented by one of several positive pressure devices in patients with decrease in pulmonary compliance due to pulmonary hypertension in the postoperative period following open heart surgery.

Contraindications for Tracheostomy

As in most areas of surgery, operative deaths and complications most often occur when tracheostomy is done either unnecessarily or despite actual contraindications. Several absolute or relative contraindications are obvious (Table 5).

Tracheobronchial infection with obstruction of small bronchi resembles laryngeal obstruction. Differential diagnosis may be difficult. Tracheostomy performed in acute tracheobronchial infection does not relieve the obstructive lesion, and by adding the dehydrating effect to already viscous and tenacious secretions, the problem may be made worse.

Although, rarely, it is necessary and even lifesaving, tracheostomy is generally contraindicated in small infants. The size and length of the tube usually do not match the anatomy of the trachea. Obstruction of main or lobar bronchi is common. Adequate aspiration is difficult because of the tiny lumen. Merely keeping the tube in place becomes a major problem. It is frequently necessary to maintain the tracheostomy for months because of the disproportionately small trachea and larynx. Mortality from the operative procedure and its complications in this age group may rise as high as 30 to 40 percent.[4,6]

Patients who are in severe respiratory distress from asthma or emphysema and who have elevation of carbon dioxide tension and are hypoxic should be considered with care before the normal airway system is disturbed. Carbon dioxide narcosis and resultant apnea are real threats, and unless a cuffed tube is utilized and positive pressure equipment with suitable adaptation to the tracheostomy tube is available, death may ensue. If retention of bronchial secretions is a major factor in the illness, tracheostomy may be helpful, but it should be done only when proper respirator equipment is at hand.

The same situation prevails when apnea has been produced by some means other than airway obstruction. Tracheostomy will not be beneficial and may make adequate resuscitation difficult unless the positive pressure equipment is at hand. Mouth-to-mouth respiration followed by the insertion of an endotracheal tube provides a more practical solution.

Inadequacy of experience, lighting, instruments or assistance should serve as contraindications to operations. Far too often tracheostomy is done in the emergency room by interns and others with little experience in handling patients who have intracranial and other major injuries, and occasionally it is done for no better reason than that the patient is unconscious. Again, other methods of resuscitation which leave an intact airway will be more beneficial to the patient, at least on an emergency basis.

Basic Principles

The following principles, if observed, would do much to reduce the mortality and incidence of complications in this operation:

1. The decision to operate should be made by an experienced surgeon if possible, and if practical, he should be in attendance at operation.
2. Assistance should be procured.
3. Tracheostomy should be performed in the operating room as an elective procedure, except in the most urgent situations.
4. Adequate lighting, instruments, and assistance should be secured.
5. The "emergency" tracheostomy should be avoided.
6. Adequate postoperative care should be given, including aspiration, humidification and precautionary sterile techniques insofar as practical.

With regard to the fifth principle, there are surgeons with a flair for the dramatic, who seem to revel in creating emergencies where none in reality exist. Also, most surgeons recognize that when they operate with a sense of urgency, they are more prone to technical errors. Therefore, if possible, the operation should be anticipated in order to prevent the real emergency.

A few practical suggestions are justified.

Tracheostomy trays should be examined periodically to assure completeness and availability. They should be strategically located in wards, recovery areas, emergency rooms, and intensive care units and should not be kept in central supply. Portable lights of sufficient quality should be available to all areas of the hospital. Resuscitation equipment of proper variety and in functioning condition should also be widespread in the hospital. Formal instruction of members of the house staff and of nonsurgeons in matters of resuscitation, intratracheal aspiration, tracheal intubation and in technical aspects of the emergency tracheostomy should also serve to lower the existing mortality and morbidity figures.

Conclusion

From the data presented it is apparent that tracheostomy is an operation which results in a significant number of operative deaths and major complications.

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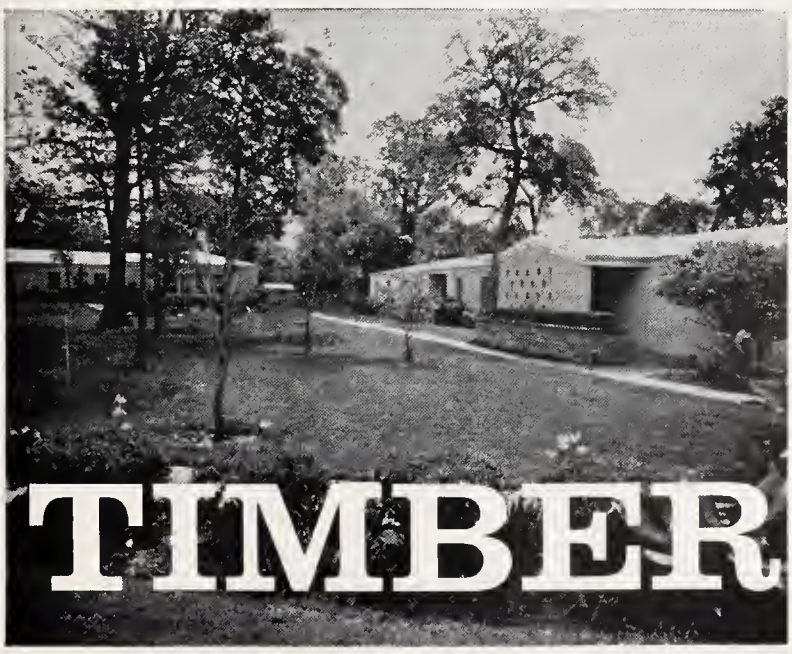

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Clinicopathologic Conference

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Three months before death, a 64-year-old woman known to have had tuberculosis for 21 years developed nausea, vomiting, spiking temperatures, tingling and loss of touch sensation in the lower extremities which progressed to loss of touch and pain up to the nipple level. She also developed a jerking or Parkinsonian-like tremor in both upper extremities and flaccid paralysis in one arm and the lower extremities and blindness in one eye. She was resuscitated after one cardiac arrest but died two hours later.

DR. SOTERES: A 64-year-old white woman was admitted to Woodlawn Hospital for the sixth and last time on Sept. 23, 1965. She was transferred from Tyler State Hospital, where she had been under treatment for active advanced pulmonary tuberculosis due to atypical tubercle bacilli (Runyon III, nonphotochromogenic). She was known to have had tuberculosis since 1944, when she was first treated. Follow-up at Woodlawn Hospital revealed negative sputums from 1959 to 1962, when the sputum once again became positive. Multiple drug regimens had been tried but had to be stopped because of sensitivity or intolerance, although she was able to take INH alone through most of this time. Her last sensitivity studies were in January, 1965, and the mycobacterium was sensitive only to kanamycin and streptomycin.

On Aug. 13, 1965, at Tyler Hospital, the patient had been started on INH and Rezipas. She developed nausea and vomiting and spiking temperature. Administration of Rezipas was stopped on Aug. 17, 1965. On Aug. 19, 1965, she developed tingling and loss of touch sensation of the lower extremities. This progressed over a two to three day period to loss of the sensations of touch and pain up to the nipple level, and her lower limbs became completely paralyzed with loss of deep tendon reflexes. No Babinski response was found. She developed what was described as a jerking or Parkinsonian-like tremor in both upper extremities with hyperactive deep tendon reflexes. All facial, buccal, and eye reflexes were normal. Data obtained from lumbar puncture at that time were glucose 62 mg. per 100 ml., protein 90 mg. per 100 ml., and cell count of three lymphocytes. On Aug. 26, 1965, the lumbar puncture was repeated with a cell count of six lymphocytes and three red blood cells, protein 54 mg. per 100 ml., and a normal colloidal gold curve. A diagnosis of Guillain-Barré syndrome was made.

On Aug. 15, 1965, the patient developed a flaccid paralysis of the right arm and deviation of the tongue to the right. On Sept. 10, 1965, she was found to be blind in the right eye. She was transferred to Woodlawn Hospital with the diagnosis of far advanced, active cavitary pulmonary tuberculosis due to atypical tubercle bacilli (Runyon III) and Guillain-Barré syndrome.

The past history included allergy to penicillin, several of the -mycins including streptomycin, para-aminosalicylic acid (PAS), and several sleeping pills and tranquilizers. Review of systems revealed no further com-

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This is the fourth in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School, Dallas. Guest editor for the series is Dr. Bruce D. Fallis, professor of pathology. Participants this month are Dr. Soteres, resident in internal medicine; Dr. Christensen, assistant professor of radiology; Drs. Foster, Pierce, and Nicholson, assistant professors of internal medicine; Dr. Johnson, associate professor of internal medicine; Dr. Seldin, professor and chairman, Department of Internal Medicine; and Dr. Reynolds, assistant professor of pathology.

plaints. The family history was negative for tuberculosis.

On admission she was a mildly cachectic, morose, chronically ill appearing white woman with blood pressure of 110/60, pulse 88 and regular, respiration 20, and temperature 99 F. She had questionable paralysis of upward gaze. The pupils were equal and reacted to light consensually; only the right pupil reacted to light directly. Bilateral cataracts were present. The fundi were poorly seen, but the disc margins were well-defined. The left eye was blind. The neck was normal. Examination of the heart was unremarkable. There was dullness to percussion with moist rales and egophony at the right lung apex. Coarse moist rales were present at the right and left lung bases. The diaphragm moved poorly. The abdomen was normal. She had an indwelling Foley catheter in her bladder. At neurological examination she was oriented as to time, place, and person. There was loss of light and heavy touch, pinprick, and temperature sensation below the second thoracic nerve on the right and the third thoracic nerve on the left, except in the left first sacral nerve distribution. Deep pain was intact in the left leg and both upper extremities. Position sense was lost in the toes. All cranial nerves were intact except for blindness of the left eye. There was flaccid paralysis of both lower extremities. The upper extremities, especially the right, were weak. Deep tendon reflexes were absent in the lower extremities but were normal in both upper extremities. There were no pathologic reflexes. There was no clubbing, cyanosis, or edema.

On admission to the hospital all drug treatment was withheld, and frequent vital signs and daily vitalometry were obtained. From Sept. 24, 1965, to Nov. 4, 1965, she showed some improvement in ability to dorsiflex her feet, and a 2+ knee jerk appeared transiently on the left. There was no change in the cranial nerves or sensory loss distribution. Urine cultures repeatedly grew *Klebsiella* and *Aerobacter* organisms in moderate numbers. On Nov. 4, 1965, she complained of excessive sweating on the right side of the body only and paresthesias in the hands. There was excessive sweating and increased warmth on the right and loss of discriminatory sense in both hands. The hyperhidrosis spontaneously remitted on Nov. 6, 1965. On Nov. 10, 1965, decreased vision of the right eye was noted; the left eye remained blind. The left disc showed optic atrophy. On Nov. 16, 1965, bronchoscopy was negative. On Nov. 22, 1965, respirations became extremely feeble. The patient began spiking temperatures and became disoriented. Rales were heard in both lung bases. Sputum cultures were taken, and a tracheostomy was performed. Keflin was started. On Nov. 24, 1965, she suffered a cardiac arrest and was resuscitated. Two hours later she again suffered cardiac arrest, and attempts at resuscitation were not successful. Representative laboratory data are shown in Table 1. Urinalysis was essentially normal on several occasions. Lumbar puncture was done on five occasions; in each instance cell counts were less than 10, protein varied from 50 to 68 mg. per 100 ml., and other tests were normal. Blood electrolyte levels were essentially normal. Results of tests of pulmonary function are shown in Table 2.

TABLE 1.—*Laboratory Data.*

	9-27	10-12	11-10	11-21
Hemoglobin (gm./100 ml.)	11.5		10.9	10.1
Hematocrit (vol. %)	37	37	36	33
White blood count x 10^3	14.1		11.1	10.0
BUN (Blood urea nitrogen)	18		14	
Carbon dioxide content mEq./liter	29	28		
Total serum protein (gm./100 ml.)	5.8		6.9	
Albumin	2.6		3.3	
Globulin	3.2		3.6	

Radiologic Findings

DR. CHRISTENSEN: The patient had innumerable x-ray examinations over a period of about six years. The initial chest films were taken in the second half of 1959 and showed a small cavitary lesion, about 1.5 cm. in diameter, in the right apex, and slight linear stranding in the left apical and midlung field. From then on there was progressive deterioration. The cavity was slightly larger in 1960 and still larger in 1961, with more reaction around it. By 1962, the cavity had become multilocular, and there was a great deal of reaction around it. By 1964, the cavity occupied the entire apical region on the right, the trachea and mediastinum were shifted to the right, and the right hilum was elevated. Progression continued until the patient died. In chest films taken during her final hospitalization, there was extensive fibronodular disease throughout

TABLE 2.—*Pulmonary Function.*

Vitalometry				
		Admission to:		
	Admission (ml.)	11-10 (ml.)	11-16 (ml.)	11-21 (ml.)
Total vital capacity	600	500-600	500	300
½ sec.	400	400-500	300	300
1 sec.	500	400-600	400	800

Blood Gases				
Test	9-23	11-22 (RAR)*	11-22 (40% O_2)	11-23 (30% O_2)
O_2	90%	71.5%	94%	85%
pH	7.47	7.36	7.40	7.49
pCO_2	39 mm. Hg.	61 mm. Hg.	59 mm. Hg.	47 mm. Hg.
pO_2	60 mm. Hg.	39 mm. Hg.	73 mm. Hg.	50 mm. Hg.

*Room air at rest.

both lung fields, with the right lung appearing almost completely destroyed. There also may have been some complicating nontuberculous bronchopneumonia. A cervical myelogram was done Aug. 24, 1965, at the Tyler State Hospital, and there were no significant abnormalities apparent on the films.

Clinical Discussion

DR. DANIEL W. FOSTER: This patient had far advanced and longstanding pulmonary tuberculosis and had been on antituberculous drugs, primarily isoniazid, for long periods of time without, as far as we know, concomitant pyridoxine therapy. Her final admission was for a subacute, progressive neurological disease characterized by both sensory and motor deficits. In a patient with this type of extensive neurological deficit, two general categories of disease must be considered: widespread polyneuritis or one of the large group of so-called demyelinating diseases. Let us consider multiple sclerosis and similar demyelinating diseases first. Whenever one has spotty neurological lesions and involvement of the eyes, the possibility of acute multiple sclerosis or related disease immediately comes to mind. Several features of the present history suggest that she does not have multiple sclerosis. In the first place, her age is against this diagnosis. Secondly, the course was progressive without remissions. While an occasional patient with multiple sclerosis may have a progressive course without remissions, this is very unusual. Thirdly, and the most important thing against the diagnosis of multiple sclerosis, is that the patient, throughout the development and the course of her disease, had flaccid paralysis with no evidence of hyperactive deep tendon reflexes in the lower extremities. One can have flaccid paralysis with absent deep tendon reflexes and no Babinski reaction in multiple sclerosis, but it is exceedingly rare. For these reasons I would tend to rule out a demyelinating syndrome of the multiple sclerosis type.

Fig. 1. A chest x-ray taken on Nov. 11, 1965, shows complete destruction of the right upper lobe by tuberculosis. Multiple cavities have replaced the lung tissue, and the hilum and mediastinum are pulled toward the right upper lung field. There is linear scarring and stranding in the right lung base, and the peripheral lung fields show a diffuse nodular infiltration.

As a general rule, widespread motor and sensory changes, extensive flaccid paralysis, and absent deep tendon reflexes should suggest a diagnosis of polyneuritis. There are many conditions which can be associated with polyneuritis: heavy metal toxicity, organic poisons like methyl alcohol and carbon disulfide, certain deficiency diseases such as beriberi and pellagra, metabolic disorders such as diabetes mellitus, porphyria, and amyloidosis, vascular diseases such as polyarteritis nodosa, and infections such as leprosy and diphtheria. None of these is

suggested by the history of the present case. In addition to these known etiologies for polyneuropathy there is the familiar idiopathic polyneuritis known as the Guillain-Barré syndrome. There is nothing in our patient's history which is not compatible with acute idiopathic polyneuritis of the Guillain-Barré type. She had an elevated protein in the spinal fluid associated with slight pleocytosis, the paralysis tended to be progressive and ascending, and there was extensive motor loss to the point of paralysis. However, at the time that she developed blindness and optic atrophy, I think that one could effectively rule out the possibility of the Guillain-Barré syndrome. While one can get extensive cranial nerve involvement in idiopathic polyneuritis, I don't believe that the second nerve is ever involved.

The obvious diagnosis that comes to mind in a patient who has tuberculosis and who has been on long-term antituberculosis therapy is INH toxicity, and I believe that this case represents a fatal polyneuritis associated with this drug. There are several diseases associated with reaction to INH.[3] In the first place, there can be a hypersensitivity reaction of the usual type, with fever, skin rashes, and associated symptoms. Two major types of neurological diseases occur. By far the more common is a peripheral polyneuropathy, such as this patient had, with widespread sensory and motor changes over much of the body. This type of neurotoxicity is usually associated with large doses of INH, but it is clear that it can develop on ordinary dosage. There is only about a 2 percent incidence of peripheral neuropathy on the usual 300 mg. daily dose of INH, that is, about 5 mg. per kilogram, but the incidence of neurotoxicity goes up very rapidly if one increases the dose to 10 mg. per kilogram, and by 20 mg. per kilogram, the incidence of polyneuropathy may reach 40 percent. The polyneuropathy usually begins with paresthesias accompanied by sensory loss and progresses to a flaccid paralysis with loss of reflexes. There is no Babinski response. There may also be bladder paralysis, as this patient apparently had, since we were told she had an indwelling Foley catheter. Involvement of the respiratory muscles may occur, and finally in association with the peripheral neuropathy, one may get an optic atrophy. In other words, the picture presented by this patient had been associated with INH intake, and I would consider this the most likely diagnosis.

The second major neurological abnormality that can occur with INH treatment is a toxic encephalopathy with a peculiar psychosis.[1,4] Most people who have the toxic encephalopathy also have some degree of peripheral neuropathy.

Nobody knows exactly how INH produces neurotoxicity. INH interferes with a ubiquitous coenzyme in the body, pyridoxal-5-phosphate. This coenzyme is a prosthetic group on many enzymes involved in protein metabolism such as decarboxylases and transaminases. Perhaps the peripheral neurotoxicity of INH represents an acute or subacute pyridoxine deficiency. At any rate one can probably prevent the peripheral neurotoxicity of INH by the concomitant administration of pyridoxine. The encephalopathy, in contrast, cannot be prevented from developing by giving pyridoxine, at least in customary doses. It should be noted that longstanding and frequently permanent damage occurs with INH neurotoxicity, even with prompt removal of the drug and treatment with pyridoxine. For example, in a recent case reported by Adams and White,[1] a patient who developed both a toxic encephalopathy and a peripheral neuropathy after a relatively brief treatment with INH was still unable to walk one year after cessation of the drug.

In summary, I believe that the patient had advanced pulmonary tuberculosis, presumably due to the atypical organism found on culture, and that the neurological disease represented INH toxicity of the peripheral type.

DR. ROBERT L. JOHNSON: I would generally agree with Dr. Foster's diagnosis. PAS may also cause neurological manifestations by virtue of hypersensitivity. However, the PAS was stopped quickly after its initiation—I think within about two or three days. The initial nausea, vomiting, anorexia, and fever may have been due to the PAS, but the neurological manifestations were probably not related.

The cause of death in this patient was obviously cardiorespiratory, and I will confine my discussion to this aspect of the case. This patient had extensive pulmonary tuberculosis. Tuberculous granulomas are avascular and as they grow, spread, and heal by fibrosis they destroy alveolar capillaries; if such

areas of involvement continue to be ventilated, they constitute alveolar dead space. The final results of extensive pulmonary tuberculosis are a restrictive ventilatory defect due to the pulmonary fibrosis, an increased alveolar dead space due to destruction of alveolar capillaries, and a reduced diffusing capacity of the lung, due to the reduced area and increased thickness of the alveolar capillary membranes. Thus, if the tuberculous involvement has been extensive, gas exchange in the lungs becomes exceedingly inefficient. To maintain normal arterial blood gases, such patients must sustain a larger total ventilation and must work harder than normal to achieve this ventilation.

In this particular patient, superimposed upon a background of extensive tuberculosis, we have respiratory failure due to what amounts to a transection of the cervical cord. She had use of both arms, and I would presume that her diaphragm was intact. Normally, a transection of the cord below the fifth or sixth cervical vertebra leaves the phrenic nerves intact, and you would expect a reduction in her vital capacity and maximum breathing capacity to only about 50 to 70 percent of normal. Obviously this patient's lung volumes and her ventilatory capacity were reduced well below this point. She had a vital capacity on admission of about 600 ml. with a half-second of 400 ml. and one second of 500 ml., an extremely restrictive ventilatory defect. So we must assume that in addition to a neurological defect she probably had a considerable reduction in lung compliance, and I think that this is supported by the x-ray results. Now as time went on, her vital capacity progressively fell, especially in the last week of her illness. I presume that this was due to a superimposed bacterial pneumonia. In addition, in the last week, her arterial oxygen saturation fell to about 72 percent when breathing room air; and even when on 40 percent oxygen, she was still remarkably desaturated. The low arterial oxygen saturation when she breathed 40 percent oxygen suggests that 25 to 30 percent of her cardiac output was going through atelectatic lung. This shunt could explain only about half of the arterial oxygen desaturation measured when she breathed room air; thus, in addition to atelectasis she also must have had extensive unequal distribution of ventilation with respect to perfusion in those areas of lung which were still ventilated. Undoubtedly these abnormalities of blood gas exchange were due to the presence of extensive exudate causing atelectasis of some areas of lung which still had blood flow and relative hypoventilation in other areas. I think she died primarily of respiratory failure and I imagine that the cardiac arrest was a consequence of a period of sustained anoxia.

DR. ALAN K. PIERCE: She had infection by group III atypical mycobacteria, the so-called Battey strain. This is an unusual infection for our area, although it is a common infection in the southeastern United States. In this area we usually see group I or photochromogens. Either can cause a progressive disease with ultimate lung destruction.

Let us now direct our attention to the management of patients with respiratory failure due to a progressive neurological deficit. One must first establish a baseline for pulmonary function. As Dr. Johnson has pointed out, intact diaphragms are the best evidence of fair performance by physical examination. However, one should then proceed with vitalometry testing to tell how much ventilatory defect is present at the time of admission. If the neurological disease has been rapidly progressive, by the time the patient's vital capacity falls to about 50 percent of normal, a tracheostomy should be considered. If the disease is more slowly developing, tracheostomy may be deferred until vitalometry is even more severely restricted. The amount of airway obstruction present is also important. If the patient is able to exhale 60 percent of his predicted vital capacity, but he cannot move it rapidly (for example, in a half second, he can exhale only 20 percent of the total), the patient is far more ill than one who can exhale rapidly (for example, with a half second of 75 percent of the total vital capacity). Such slowing indicates either asymmetrical involvement of the diaphragm or obstruction with mucus that the patient has been unable to clear. A tracheostomy must be performed in the presence of a less severe degree of total restriction. One obtains the baseline measurements and then observes the patient carefully. The frequency of vitalometry testing is determined by the rapidity of the onset of the neurological disease. In the usual patient with the Guillain-Barré syndrome, who presents with a fulminating course, the patient should probably have vitalometry every hour. On the other hand, in a patient

who has a more chronic course that has already stabilized by the time of admission, less frequent vitalometry would be reasonable.

If the patient is thought to need some form of ventilatory assistance, but it is thought that he may recover rapidly from his paralysis, we may utilize some form of body respiratory assistance, that is, external shell respiratory ventilation. Assistance with this type of ventilator is not as efficient as it is with intermittent positive pressure. Thus, if there is reason to believe that the paralysis will be longstanding, we think that intermittent positive pressure is preferable. If one plans to ventilate the patient with positive pressure, a tracheostomy must usually be performed.

DR. DONALD W. SELDIN: Now, Dr. Pierce, do I understand that you regard the decision to delay a tracheostomy to the 22nd following a vital capacity on admission of 600 ml. with a progressive pulmonary infiltrate as a wise one?

DR. PIERCE: Based on the information in the protocol, I would probably have performed a tracheostomy on admission. The patient was hypoxic and this hypoxia was, of course, detrimental to her nervous system. Whether she would have recovered her neurological function or not, no one can say, but certainly hypoxia would at least delay recovery. The pH was alkalotic, and the carbon dioxide pressure (pCO_2) was low normal. An alkalotic pH shifts the oxygen dissociation curve in such a manner that the hemoglobin has more affinity for oxygen, and thus oxygen is not readily available to the tissues. Tissue oxygenation is not as good as indicated by the arterial oxygen saturation in the presence of alkalosis. In the presence of a low pCO_2, blood flow to the brain is reduced, and cerebral oxygenation is further impeded. In such a situation the patient needs virtually continuous oxygenation to prevent tissue hypoxia. In addition, correction of the hypoxia would remove part of her drive to respiration, and her pCO_2 might slip up a little; if her pH then came down, she would be benefited. One would have to be careful, of course, to be sure that the better oxygenation didn't remove her drive for breathing altogether, causing her pCO_2 to rise too much. I believe that this patient should have had continual oxygenation of a sufficient magnitude to keep her arterial oxygen saturation normal but not so much that it would tend to promote more atelectasis than she already had. The best manner to give continuous oxygen is not by nasal cannula which gives virtually dry oxygen, but by continuous heated nebulization, so that as much liquid as possible is carried by the gas. Thereby two things are accomplished: she will be oxygenated, and her secretions will be loosened so that she can bring them up. If she had a lot of secretions, as she probably did, a tracheostomy would be mandatory early in the course.

DR. JOHNSON: In assisting the ventilation of a person such as this, I think that an attempt should be made to follow at regular intervals the tidal volume that is being provided. This is important because it is going to determine whether the ventilation is continuously adequate or not. Now to start with, you can make a simple calculation of what an adequate ventilation would be if the patient's lungs were normal. The alveolar ventilation in ml./min. should equal the carbon dioxide production in ml./min. times a constant, 863 mm. of mercury at sea level, divided by the arterial carbon dioxide tension in mm. of mercury that you wish to maintain. Carbon dioxide production can be estimated as about 100 ml./min. per square meter of body area, adding 10 percent for each degree of temperature elevation. Knowing the alveolar ventilation you want to provide the patient, the respiratory rate and tidal volume required to achieve it can be estimated as follows:

$$\text{Tidal volume required (ml.)} = \frac{\text{alveolar ventilation (ml.)}}{\text{respiratory rate}} + \text{estimated anatomical dead space (ml.)}$$

(Equation 1)

The anatomical dead space in milliliters in a normally proportioned person is approximately equal to the body weight in pounds. However, in this individual, the estimate from the preceding formula will probably be inadequate, because her lungs are abnormal and gas exchange is inefficient, that is, there is an increase in alveolar dead space. Consequently, an arterial pCO_2 must be drawn to find out whether the predicted ventilation is adequate with regard to CO_2 excretion. From the discrepancy between the arterial pCO_2 predicted and that measured, we can estimate the fraction (f) of the alveolar ventilation which is effective. Then we can appropriately correct the tidal

volume according to the following modification of equation 1:

$$\text{Tidal volume} = \frac{\text{alveolar ventilation} \times f}{\text{respiratory rate}} + \text{estimated anatomical dead space (ml.)}$$

(Equation 2)

This patient obviously at times was not being ventilated adequately; for example, she had a pCO_2 of 59. It was perhaps adequate ventilation to maintain a normal oxygenation since she was receiving an oxygen-rich mixture, but not adequate to maintain a normal pCO_2; furthermore, her tidal volumes may have been too small to prevent progressive development of patchy atelectasis.

DR. DAVID NICHOLSON: By and large, I agree with Dr. Foster. There are several possible neurological disorders of the peripheral and central nervous systems that could arise. The central effects of isoniazid vary from somnolence, through apprehension and convulsion, to an acute toxic psychosis. These effects are reversible on withdrawal of the drug. The peripheral neuropathy is prevented by the use of pyridoxine, but this vitamin is not of therapeutic benefit. In view of the sudden onset of an apparent peripheral polyneuropathy which would be consistent with INH toxicity, proceeding to a demyelinating disease, I would speculate that INH toxicity led to a peripheral neuritis, the release of neural components into the circulation, and the subsequent development of autoimmune sensitization of the central nervous system.

The patient had received pyridoxine until a few days prior to the onset of her neurological disease, and at that time too PAS was given despite a history of previous hypersensitivity.

DR. SELDIN: In view of the previous comments about pyridoxine, was this patient given pyridoxine along with her INH?

DR. NICHOLSON: Yes, but she did not receive pyridoxine at Tyler State Hospital. In the INH and PAS compound we use, pyridoxine is incorporated.

Pathological Findings

DR. R. C. REYNOLDS: The major anatomic lesions were found in the lungs and the central nervous system.

The lungs weighed 2,300 gm. Gross examination revealed a massive tuberculous cavity in the upper lobe of the right lung. This cavity communicated with a medium-sized bronchus, which was partially filled with caseous material. Other medium-sized and large bronchi were filled with a mucopurulent exudate. Multiple small cavities and foci of caseous necrosis measuring up to 2 cm. in diameter were found scattered throughout the middle and lower lobes of the right lung. There was severe pulmonary congestion and edema.

Microscopic examination of the lungs revealed active nodular-caseous and cavitary tuberculosis with extensive endobronchial dissemination. A tuberculous bronchitis was found in the bronchus communicating with the large cavity in the right upper lobe. A number of the smaller cavities were found to communicate with small bronchi. Scattered throughout both lungs, particularly in the right lower lobe, there were multiple miliary granulomata arising in the respiratory bronchioles. The lesions in the respiratory bronchioles varied in age from an early acute focal tuberculous pneumonia to well-developed tubercules showing a collar of dense fibrous tissue, caseous necrosis, and Langhans' giant cells. The fully developed lesions frequently involved the pulmonary arteriole accompanying the respiratory bronchiole causing thrombosis and obstruction of the blood vessel lumen. Acid-fast stains of the lung revealed numerous acid-fast bacilli.

The patient's second disease process was found in the central nervous system, involving primarily the spinal cord and the optic nerves. Gross examination of the optic nerves disclosed that they were essentially normal in appearance. A transverse myelitis was found in the lumbar segments of the spinal cord. In this area the spinal cord tissue was granular and hemorrhagic, and all normal landmarks were obliterated. In the thoracic portions of the spinal cord the dorsal tracts were yellowish-white, indicating extension of the disease process upward from the lower segments of the cord.

Apparent on microscopic examination of the spinal cord and the optic nerves was severe demyelinating process associated with destruction of the nerve fibers in the most severely involved areas. There were few identifiable structures in sections taken from the lumbar segments of the spinal cord. The lesion in the lumbar spinal cord varied in its microscopic appearance. A portion of the cord was characterized by an active, acute demyelinating process with a moder-

ate infiltrate of lipid-laden macrophages and other inflammatory cells. In other portions of the spinal cord the disease process was no longer active. In these areas there was gliosis with virtually complete disappearance of myelin. The axon fibers were destroyed, and there was minimal inflammation. With myelin sheath stains, the demyelinating process in the dorsal columns could be traced microscopically as high as the lower medulla.

A similar demyelinating process (Fig. 2) was evident upon microscopic examination of the optic nerves. The lesion was most severe in the left optic nerve and extended from the globe back to the optic chiasm. Gliosis and fibrosis were present. Virtually no myelin remained in the left optic nerve. There was a moderate infiltrate of chronic inflammatory cells. The right optic nerve was less severely involved. The demyelinating process was patchy in distribution. The early lesions were characterized by swelling and fragmentation of the myelin sheaths and an infiltrate of lipid-laden microglial cells. Microscopic examination of the optic tracts revealed a few focal lesions. No lesions were found in the occipital area of the brain.

In summary, the patient had active adult-type pulmonary tuberculosis with multiple cavities and extensive endobronchial dissemination due to atypical tubercle bacilli. In addition, there was a demyelinating disease of the central nervous system, primarily involving the optic nerves and the lower segments of the spinal cord. The gross and microscopic appearance of this lesion is consistent with that described for Devic's disease for neuromyelitis optica. This disease has long been considered an atypical form of multiple sclerosis. Devic's disease occurs more frequently in middle-aged people than does the usual form of multiple sclerosis. An age range from 5 to 60 years has been described by one author[6].

The etiology of the central nervous system disease in this patient remains obscure. That it represents a demyelinating process produced by INH is an intriguing possibility, and Dr. Nicholson's comments in this regard are appropriate. INH therapy has characteristically been associated with peripheral polyneuropathy rather than with central nervous system disease. However, there is experimental evidence suggesting that in the duct[2] and in dogs[5], INH may produce a demyelinating central nervous system disease.

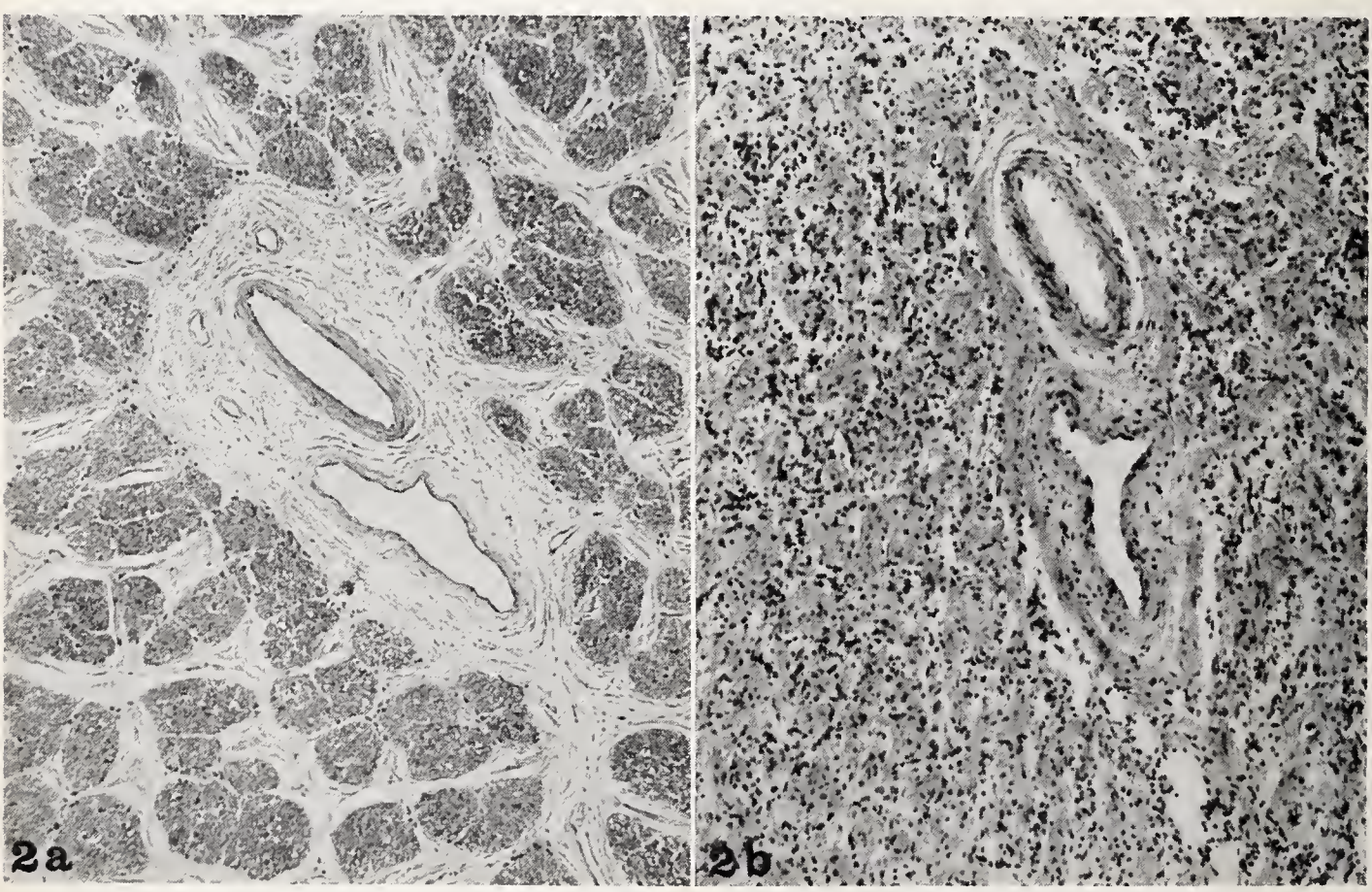

Fig. 2. a. Photomicrograph of a myelin sheath stain showing an uninvolved portion of the right optic nerve. b. Complete demyelination and destruction of axon fibers which characterized the entire left optic nerve. x 100.

DR. FOSTER: Pathologically this case, despite the objections I mentioned earlier, appears to represent a fulminant demyelinating disease with a classic picture of neuromyelitis optica, thought by many to be a variant of the multiple sclerosis syndrome. I do not believe it likely to have been due to INH toxicity, because INH does not, to my knowledge, produce this type of lesion.

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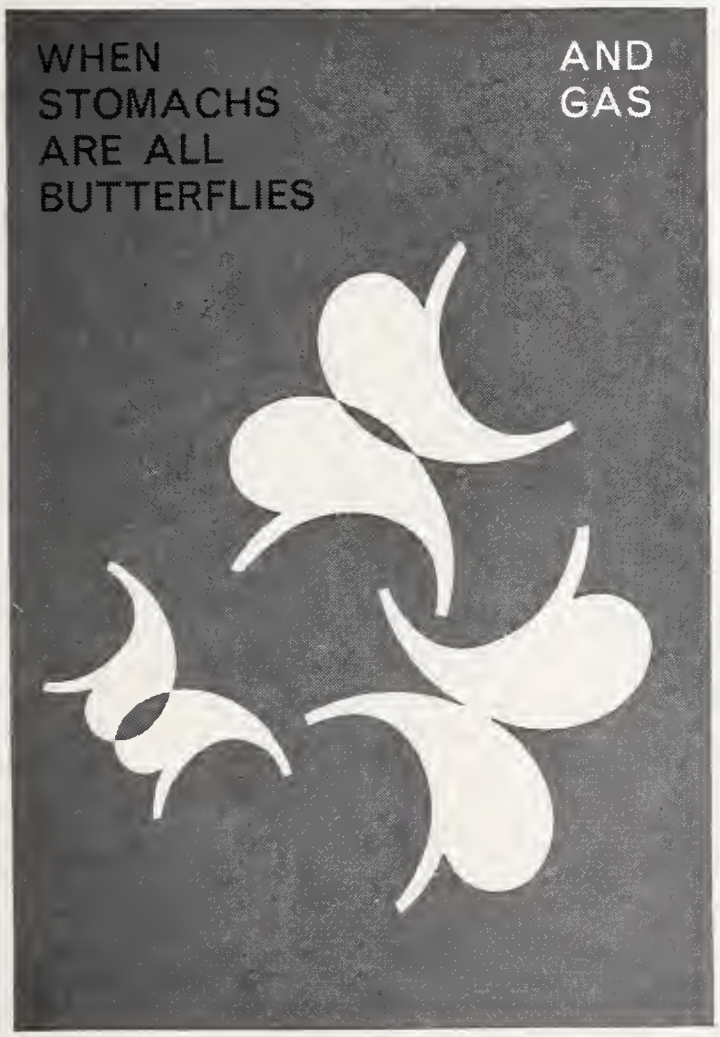

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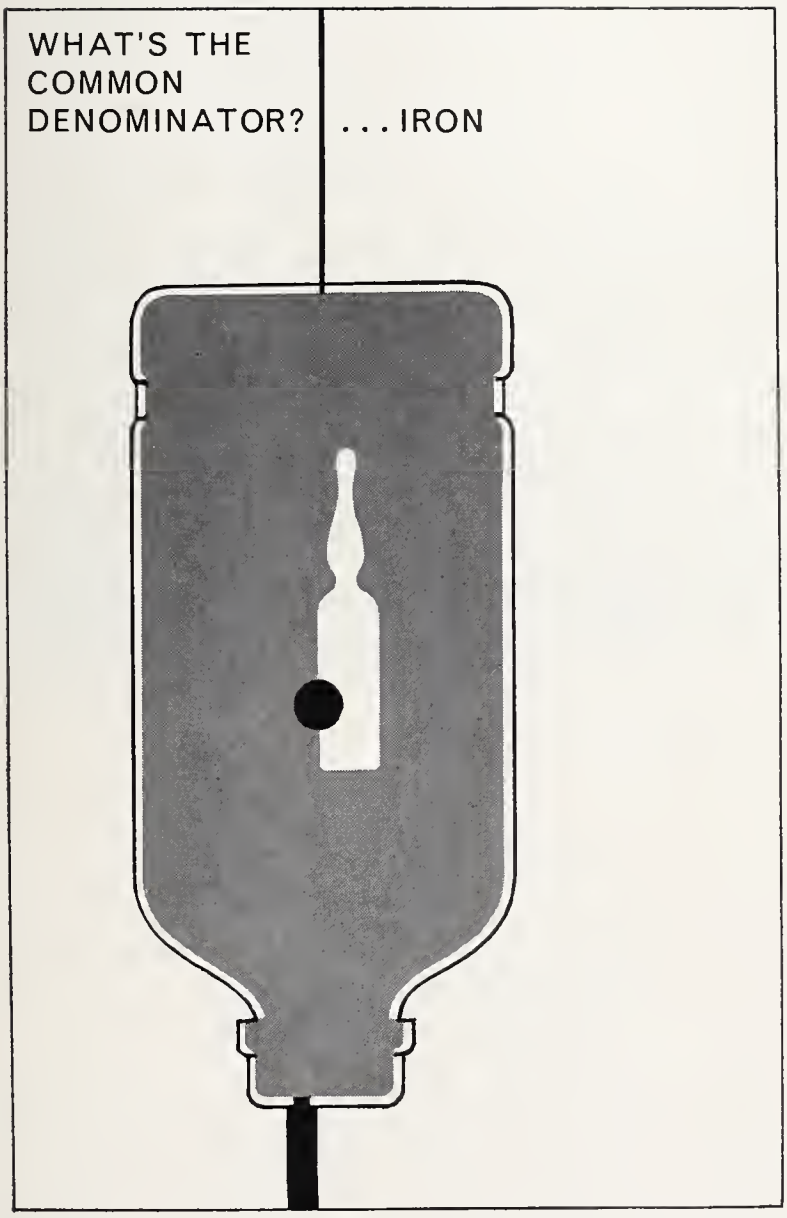

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IN BRIEF: ACTION AND USES: A single dose of Imferon (iron dextran injection) will measurably begin to raise hemoglobin and a complete course of therapy will effectively rebuild iron reserves. The drug is indicated only for specifically-diagnosed cases of iron deficiency anemia and then only when oral administration of iron is ineffective or impractical. Such iron deficiency may include: patients in the last trimester of pregnancy; patients with gastrointestinal disease or those recovering from gastrointestinal surgery; patients with chronic bleeding with continual and extensive iron losses not rapidly replenishable with oral iron; patients intolerant of blood transfusion as a *source of iron;* infants with hypochromic anemia; patients who cannot be relied upon to take oral iron.

COMPOSITION: Imferon (iron dextran injection) is a well-tolerated solution of iron dextran complex providing an equivalent of 50 mg. in each cc. The solution contains 0.9% sodium chloride and has a pH of 5.2-6.0. The 10 cc. vial contains 0.5% phenol as a preservative.

ADMINISTRATION AND DOSAGE: Dosage, based upon body weight and Gm. Hb/100 cc. of blood, ranges from 0.5 cc. in infants to 5.0 cc. in adults, daily, every other day, or weekly. Initial test doses are advisable. The total iron requirement for the individual patient is readily obtainable from the dosage chart in the package insert. Deep intramuscular injection in the upper outer quadrant of the buttock, using a Z-track technique, (with displacement of the skin laterally prior to injection), insures absorption and will help avoid staining of the skin. A 2-inch needle is recommended for the adult of average size.

SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylatoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

CONTRAINDICATIONS: Imferon (iron dextran injection) is contraindicated in patients sensitive to iron dextran complex. Since its use is intended for the treatment of iron deficiency anemia only it is contraindicated in other anemias.

CARCINOGENICITY POTENTIAL: Using relatively massive doses. Imferon (iron dextran injection) has been shown to produce sarcoma in rats, mice and rabbits and possibly in hamsters, but not in guinea pigs. The risk of carcinogenesis, if any in man, following recommended therapy with Imferon (iron dextran injection) appears to be extremely small.

SUPPLIED: 2 cc. ampuls, boxes of 10; 5 cc. ampuls, boxes of 4; 10 cc. multiple dose vials.

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SAIGON
MY THO
CAN THO
RACH GIA
GULF
OF
SIAM
SOUTH CHINA
SEA

Texas physicians, arriving for service in South Vietnam, were keenly impressed with the war's reality there, surpassing what they had imagined at home. Five of ten Project Viet-Nam civilian volunteers describe their experiences and personal reactions during two-month tours of duty in this war-weary land on the far side of the globe. Exotic Saigon, once the Pearl of the East, was found to be a city of contrasts — amid filth, there was evidence of rich, natural beauty; amid sounds of artillery, there was gaiety and laughter. Almost unanimously, however, the emotions of the Texans were aroused by the Vietnamese people who were found to be courageous, generous, and grateful for medical help. Their need for help in many areas was found to be great.

VIET-NAM

TEN TEXAS doctors have spent two months in voluntary service in a civilian capacity in South Vietnam since Project Viet-Nam began operations in August, 1965.

The project, an attempt to bring better medical care to the civilian population of Vietnam, was initially sponsored by the People-to-People Health Foundation and became a permanent program under the auspices of the American Medical Association on July 1 of this year.

Texas doctors who have participated in the project so far will be honored at the Texas Medical Association Public Relations Conference in Austin on Oct. 1.

The ten Texas physician-participants are, in order of departure date, (1) Dr. Roscoe Gene A. Schulze, Flatonia general practitioner and psychiatrist, September, 1965; (2) Dr. John V. Connolly, Ingleside general practitioner, and (3) Dr. Kurt Lekisch, Midland internist, October, 1965; (4) Dr. Otto H. Heins, Raymondville general practitioner, December, 1965; (5) Dr. Billy Jim Blankenship, Galveston surgeon, and (6) Dr. Lumir F. Novak, retired San Antonio surgeon, January, 1966; (7) Dr. Edward B. Singleton, Houston radiologist, and (8) Dr. Jerry W. Bains, Galveston surgeon, February, 1966; and (9) Dr. Wayne Truax, Galveston internist, April, 1966; and (10) Dr. Milton S. Thompson, San Antonio orthopedic surgeon.

President Johnson initiated the idea for Project Viet-Nam in a speech on May 13, 1965. He said, "The third face of war in Viet-Nam is, at once, the most tragic and most helpful. It is the face of human need. It is the untended sick, the hungry family, and the illiterate child."

At the time of the President's address, the need for trained medical personnel to care for Vietnamese civilians had reached emergency proportions. There were only 700 Vietnamese physicians to care for 16 million people. Five hundred of these doctors were serving with the armed forces. The doctor-patient ratio was 1 to 23,000 in Vietnam; 1 to 780 in the United States.

The Vietnamese government initiated the request for United States government help in the medical area. The Vietnamese asked for some means to be provided to encourage American physicians to volunteer to help in Vietnam. The country had experienced a quarter of a century of military upheaval. The people were sick, injured, weary, and without an adequate number of medical personnel to serve their needs.

Project Viet-Nam was a pilot program, administered by the People-To-People Health Foundation. Before the project was concluded on June 30, it had sent 119 US doctors to Vietnam.

President of the People-to-People foundation and chairman of Project Viet-Nam, Dr. William B. Walsh, made the following statement concerning the project's success. "The response of American doctors to the call for volunteers has been both enthusiastic and heartening. When President Johnson asked us to undertake this program on a trial basis, we felt certain that doctors would be ready and willing to serve. We were right. Within two weeks our first volunteer was in the field and in a month many more were serving. Better than 1,000 doctors have directed written inquiries to our office."

After the successful trial period, Project Viet-Nam has now become a permanent program under the auspices of the American Medical Association. Operating funds are provided by the government Agency for International Development (USAID). The AMA Department of Government Medical Services will administer the project. Dr. Charles H. Moseley is director of this department. When the AMA assumed control of the project on July 1, the work was renamed AMA Volunteer Physicians for Vietnam.

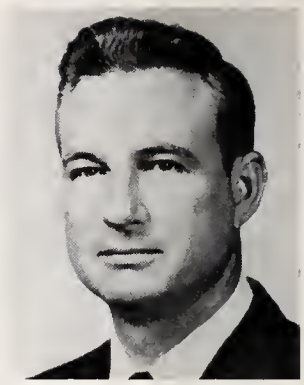
Under the program 20 American doctors, volunteering for a minimum of two months' service, serve on a rotating basis. These physicians are to treat primarily the medical needs of the civilian population of South Vietnam. Generally, the most pressing need has been for orthopedic surgeons, general surgeons, and general practitioners. Project Viet-Nam has also been concerned with making assignments of long-term medical personnel for whom some salary arrangements are made.

Doctors serve in civilian provincial hospitals where long-term US medical personnel are working. They work under the supervision of the Health Division Chief for the United States Operations Mission (USOM now is the same organization as USAID) and within the structure of the government of the Vietnam USAID Provincial Hospital Development Program. The hospitals where American USAID medical teams work are in Da Nang and Nha Trang on the coast, and Can Tho and Rach Gia in the Mekong Delta. Each team is made up of two or three physicians, a like number of nurses, one laboratory technician, and one hospital administrator. In addition, six Army doctors are on loan to the Agency for International Development for service in half a dozen other cities.

DR. LEKISCH

DR. SINGLETON

DR. HEINS



DR. BLANKENSHIP

Other volunteer medical workers in Vietnam are teams from the International Rescue Committee (IRC) which provide medical care for Vietnamese refugees.

An IRC team is composed of two physicians and two nurses. The IRC Vietnam Medical Program also is being conducted under a contract with USAID.

The IRC is a voluntary, non-sectarian organization which has been active in refugee work for more than 30 years. The program includes doctors from other countries besides the United States. Project Viet-Nam employed only American physicians to serve in Vietnam.

Doctors volunteering for assignment in Vietnam requested application forms from Project Viet-Nam headquarters. Upon acceptance, the usual routine of securing passport, visas, immunization shots, and transportation began. Expense connected with all of these activities was paid by Project Viet-Nam.

Housing was provided for volunteer doctors in available hotels or apartments in Vietnam. Each volunteer was covered while in Vietnam by an all-risk insurance policy at no expense to the doctor. Each physician also received a $300 a month living allowance but was not otherwise paid.

In order to preserve continuity of medical care, the volunteer doctors worked closely with the long-term medical workers. These workers were from Vietnam, the United States, and other free world countries.

Power Struggles in Vietnam Rooted Deep in Area's History

Kurt Lekisch, M.D.

VIETNAM has one of the world's oldest living civilizations. It dates back to hundreds of years before the birth of Christ, with roots in Asian religions and philosophies. The Hong Bang dynasty apparently ruled Vietnam for 26 centuries. This is particularly interesting to me, as the hospital in Saigon at which I was stationed bore the name of this dynasty.

In 111 BC, Chinese armies conquered and occupied Vietnam and for a thousand years the Chinese Han dynasty ruled Vietnam. However, the Vietnamese retained their cultural and ethnical individuality. Apparently they had their own customs and were not absorbed into the Chinese society.

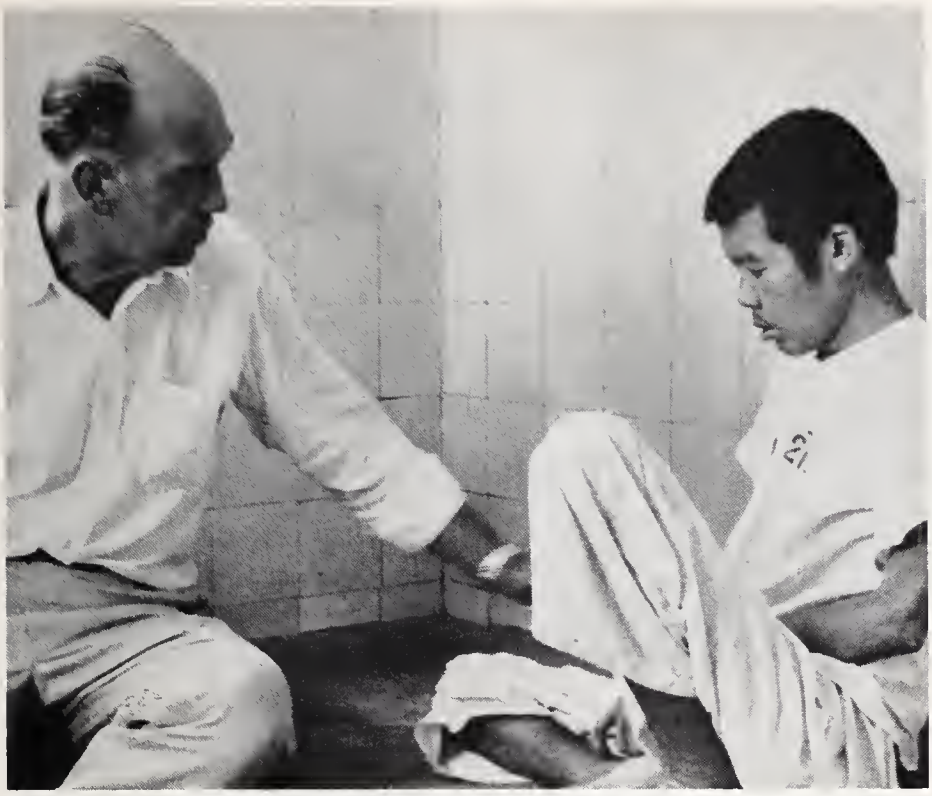
In about 939 AD, the Chinese army was defeated by Vietnamese forces under Nto Quyen.

During Vietnam's Golden Age, from 940 AD until approximately 1600 AD the country enjoyed a period of brilliant progress under Emperor Le and his dynasty. In the 15th century, the Viets pushed southward to conquer the once great kingdom of Champa which occupied much of what is now South Vietnam. The Chams, the people of Champa, were an Indonesian people with a Hindu civilization. Their kingdom had capitals at first near Da Nang and later near Qui Nhon.

Vietnam has been known to the West since the first century AD, as evidenced by the Roman coins excavated in South Vietnam. However, it was in 1535 that Dutch, Portuguese, French, and Japanese merchants settled along the coast to trade spices, aloe wood, and other products.

In 1615 the French and Portuguese merchants were followed by Catholic missionaries. In 1626 a group of highly educated Jesuit fathers were expelled from Japan; the most famous of these was Alexander of Rhodes, a linguist, who introduced the Latin alphabet and changed the Chinese script to a Latin script. This Latin script, known as *quoc-ngu*, became from then on out the written language and was responsible for closer ties between Vietnam and the Western world—from an economic and cultural aspect.

The Catholic mission work was extremely successful in Vietnam; consequently a rivalry developed among the Catholics, the Buddhists and the believers in the Confucian ethics. On one occasion, a Catholic bishop lost his life; and this was the beginning of the French po-



Dr. Roscoe Gene A. Schulze talks with patient in the admissions ward of Bien Hoa Mental Hospital. Dr. Schulze was sponsored by Project Viet-Nam and AMDOC, Inc. The Bien Hoa Hospital is the only mental institution in South Vietnam. Two Vietnamese doctors care for 2,000 patients.

Barbed booby traps, contaminated with human excrement and capable of producing serious infection, are placed on the ground by Viet Cong.

litical control of Vietnam shortly after the turn of the 18th century. Finally, in 1862, the French had political power in their hands and called this part of the world French Indochina, which also included territories now known as Laos and Cambodia.

The French realized that people in Vietnam, proud but cynical, could be handled only by the "divide and rule" principle. Consequently, Vietnam was divided: the northern portion was known as Tonkin, now mainly North Vietnam; the central portion was known as Annam, part of it now belonging to North Vietnam and part belonging to South Vietnam; and Cochin China was the main portion and included Saigon and the Mekong Delta region south of Saigon.

There was continuous unrest while the French had political power. The educated Vietnamese had learned "liberty, equality, and fraternity" as principles but not to be practiced in their homeland. About 1914 the French recruited 100,000 Vietnamese for service in France during World War I, overlooking the obvious danger that these men would return from the West with modern ideas and a desire for change in their homeland.

In general, Asian leaders considered World War I as a European civil war; and this demolished the moral foundation of Europe's prestige.

During and at the close of World War I, Woodrow Wilson taught, "Self-determination of peoples everywhere has to be the dominant political philosophy of the 20th century." This political foresight was recognized by a man, unknown in 1919, who made contact with his Vietnamese fellow citizens in France and presented to the Versailles Peace Conference a plan for the independence of Vietnam. Ho Chi Minh (meaning "he who enlightens") was this unknown figure. He was not heard.

He organized a few socialistic Vietnamese organizations in France about 1919 and then vanished for revolutionary political training in Russia. He returned to North Vietnam about 1923, and that year has come to be considered the year of the beginning of the Liberation Wars in Vietnam. Terrorist activities against the French in the early 1920's were limited to the northern section of Vietnam. Ho Chi Minh's financial support came from the communists.

In the spring of 1940 the French and British armies collapsed on the European continent; and, simultaneously, about one and a half years before the attack on Pearl Harbor, the Japanese occupied all of French Indochina. The French that were left behind cooperated with the Vichy regime but were only allowed to carry on cultural programs under the strict, but fair, Japanese occupation. This was the first time for more than a century that the peoples of Indochina could recognize that the yellow man could rule just as well as the white man. Their confidence in the white man was shattered.

Ho Chi Minh was operating from Chinese bases and actually had political and other support from the US Central Intelligence Agency. He helped facilitate the escape of American pilots shot down by the Japanese in that part of the world in addition to his other activities.

Ho Chi Minh tried to be heard, in a direct or indirect way, during World War II at high level conferences such as Casablanca, Teheran, and Yalta. The US State

Department had only 15 experts on Indochina; and, due to other disagreements among the Allies, the question of what should be done with Indochina after the defeat of Germany and Japan was never seriously considered.

In August, 1945, the Japanese surrendered, leaving all their military hardware in Indochina. Ho Chi Minh declared the Democratic Republic of Vietnam independent of France on Sept. 2, 1945.

The French, then, received military assistance from Chinese national forces in the north and British expeditionary forces in the south.

From 1946 until 1954, under the direction of Ho Chi Minh and Gen. Vo Nguyen Giap the French were attacked throughout Vietnam. The French lost approximately 140,000 soldiers. The war was supported 80 percent by 2.6 billion dollars in US aid; but, finally, in 1954, the terrorist guerrilla type of warfare was so detrimental to the French morale that a conference in Geneva was assembled. Other nations participated, but the treaty was signed only by the representatives of Ho Chi Minh and the French government.

The country was divided between North and South Vietnam at the 17th parallel. Free exchanges of those who wanted to go to a different section was agreed upon. One million went south and 150,000 went north. However, Ho Chi Minh stated that his goal was to govern all of Vietnam.

From 1951 to 1954, Mr. Ngo Dinh Diem was residing in the United States. He was able to make leaders of this country believe in his political abilities. He was appointed as premier of the Independent Republic of South Vietnam and until 1963 acted as dictator. He had the assistance of his brother Nhu, his sister-in-law, and another brother, the Archbishop of Saigon.

Throughout the Diem regime the political leaders of his circle, reinforced by many Catholics from North Vietnam, angered the Buddhists by showing continued favoritism to their own religious cohorts. The Diem regime had no understanding of land reforms in a country where 82 percent of the people were farm tenants. Actually, the important plantations (rice, rubber, tea, and coffee) had remained more or less in French hands.

The strategic hamlet project, seeking a self-defending fortified unit for farmers, was suggested and tried. This project was unsuccessful although it had worked in Malaya. Col. Edward Lansdale, a CIA official, tried to convince Diem to initiate practical land reforms; but, because it was thought that the white man's sug-

Top, opposite page. People gather in front of the police station in Hue immediately following an attack by the Viet Cong. Bottom, opposite page. Vietnamese military officers visit citizens of a small village in a northern province of South Vietnam. Immediate left. South Vietnamese soldier stands guard outside Viet Cong prisoner compound in a small provincial village. Thirty prisoners were housed in this small hut.

gestion might represent colonialism in the public mind, nothing was accomplished.

The Vietnamese army attempted to cope with Communist terror activities. In 1957 the Americans tried to assist by counterpart assignment in the military, a system which had worked well in Greece and Korea. Apparently a considerable lack of understanding of the Vietnamese people was responsible for the failure of this program.

Political movements against the regime developed in South Vietnam about 1959. These movements took place among a group of young men students educated since the independence of their country in 1954; the Bonzas who led the Buddhist movement and had their greatest support in the countryside; the Catholic group in power; and the military establishment. This led in 1960 to the first coup against the Diem regime led by Col. Nguyen Chanh Thi, commander of a parachute brigade (the same man who was removed from his duties as Commander of the First Corps on March 10, 1966). General Thi disappeared in Cambodia after his coup failed and returned in November, 1963, after Diem's death.

In the spring of 1963 the now well-known Buddhist leader and monk, Quang Duc, initiated political unrest, "Buddhist-induced anarchy," in the Hue–Da Nang area. Several monks burned themselves. The emergence of Buddhism as a political force has to be understood in the light of oppression for about 150 years, under the French and later under President Diem. We now see Buddhism as a political force in Vietnam. The Buddhist appetite for power is in the making. In November, 1963, it was mainly the military "Young Turks" who were responsible for the overthrow and death of Diem and his associates.

The power struggles between the different groups for political, economic, social, and technological leadership are the real revolutions in South Vietnam, in addition to the fight against the Communists. Religious animosities in South Vietnam have deep historical roots, and this is exploited in the climate of unrest so well demonstrated in recent months.

From 1963 until the present, eight different governments have tried to run South Vietnam and to fight a war. On June 12, 1965, finally the insurgents under General Nguyen Cao Ky took power. Ky's first actions were to sever diplomatic relations with France because of French statements about neutralization of Southeast Asia; and to move against corruption, hoarding and war-time profiteering. In February, 1966, President Johnson met with Premier Ky; for, at that point, it appeared clear that General Ky was not a great mover but, rather, a defender of the internal status quo. The meeting had held promise that immediate changes would be initiated.

The rivalry between General Thi and General Ky was responsible for the dismissal of General Thi. As the newspapers have explained, the supporters of the ambitious Buddhist monk, Quang Duc, took advantage of this political unrest.

It is understood that American influence behind the scenes has tried to save the Ky government by negotiation with General Thi's supporters and with the powerful Buddhist movement. Considering the deep political, religious, social, and regional divisions existing in South Vietnam, combined with the cynical and sensitive character of the Vietnamese people, the outcome of internal struggle will require a great deal of diplomacy from the American point of view. Any direct interference will be considered colonialism.

Recently, Secretary of State Dean Rusk stated: "The war in Vietnam is a war about the future of Asia and may be remembered as a historic turning point. The loss of Southeast Asia would constitute a serious shift in the balance of power against the interests of the Free World. The loss of South Vietnam would make the defense of the rest of Southeast Asia much more costly and difficult."

A small village in a northern province of South Vietnam is viewed from a helicopter. From the air one can often see that the country is at war. Highways fail to go any where, and railroads end in a snarl of twisted track. Bones of convoys are visible: they never made their destination. Bridgespans can be seen dropped in midstream.

Faraway Places With Strange Sounding Names Lure Physician to Volunteer Work in Vietnam

B. J. Blankenship, M.D.

VIETNAM, SAIGON, DA NANG —names of far-away places— strange sounding names. Vietnam, a strange land, a land of paradoxes and contrasts; the East at its most inscrutable and also at its most gracious and exciting! Saigon, once a gay city, the Pearl of the East, the Paris of the Orient.

Project Viet-Nam first came to my attention in September, 1965, when I read an article in the *AMA News* giving a resume of the program and asking for volunteers.

I wrote to Project Viet-Nam headquarters and received a reply to my inquiries in early October, 1965, from the program director.

After paper work was completed, on Nov. 9, 1965, I received a letter stating that "as yet, there is no opportunity for utilizing the services of a maxillofacial surgeon in Vietnam."

Naturally, I was disappointed, but I told myself that maybe it was best that I did not go. After all, I had just finished 19 years of college and residency training, was trying to start a private practice. There were plenty of things for me to do in Galveston, and I felt that I probably had already done my part for my country when, as a youth, I spent six years in the US Navy and Merchant Marines, most of the time overseas and much of it in the Pacific and the Orient. Then, too, most important of all, I had not as yet told my wife of my intentions.

In late November, 1965, Dr. Roy A. Stout and I were in Washington to attend a meeting with the consultants to the Army Surgeon General. I decided to stop by the Project Viet-Nam office and learned at that time that there was a critical shortage of general practitioner volunteers. Although trained in oral and maxillofacial surgery, there was a chance that I could be of some service as a general practitioner and still serve as a surgeon when needed. The processing of my application for assignment in general medicine began.

On Dec. 7, 1965, a letter from Washington assured me of a January departure.

The very next day I received another letter from Washington stating that "the Government of Vietnam is likely to question your credentials in utilizing you as a general practitioner" and that I "should think less positively of the tentative mid-January assignment."

Then on Dec. 16, 1965, I received final approval of a 60-day assignment in Vietnam, beginning Jan. 17, 1966, with a departure date from Galveston on Jan. 13. I also received additional papers to complete.

Most of my friends, and especially my family, wanted to know my reasons for volunteering to serve without pay in a war-torn country far from home. There were many reasons, three of which I related to the Project Viet-Nam office:

1. I believe that my training, knowledge, personality, and ability to get along with and like people will make me a good representative of the American public in general and the American doctor specifically to the people of South Vietnam.

2. Because of the critical need for doctors, I want to personally do all I can to treat and help the oppressed people of South Vietnam.

3. This experience abroad will increase my knowledge and insights of the practice of medicine in general and make me a better doctor and medical educator in the United States when I return home.

Army jeep stops in front of Project Viet-Nam Guest House, Saigon.

Vain or not, I still hold to my original reasons for going to Vietnam. Of course, there were other reasons, some of which are not valid. Even though I did not express these reasons to anyone before, I will relate them now:

4. I was becoming aware of our foreign policy in Vietnam, and I wanted to see for myself what was going on over there.

5. I had been studying and training for nearly 19 years. I was tired and wanted a change of scenery.

6. Although I had already spent several years overseas and had circumnavigated the globe five times, I still had a yearning for "those faraway places with strange sounding names."

7. A classmate and one of my best friends, Capt. Glenn R. Capps, was serving in Vietnam as a US Army doctor and adviser to the Vietnamese First Infantry Division. I am a great believer in true friends.

With less than 30 days left before departure, I began a frantic pace to get things in order and to make final preparations.

My last night at home was spent packing bags, and I finished just in time for my wife and two girls (four months and three years old) to drive me to the airport in Houston.

Finally, in the luxurious Los Angeles International Hotel, I met the other five doctors who would make up the Project Viet-Nam group. At a farewell dinner we were briefed by Dr. Edwin W. Brown, Jr., and told that 59 doctors from 29 states had preceded us. Early the next morning, newspaper photographs were taken. Then we boarded an airplane with our around-the-world tickets, a $50,000 life insurance policy, and $300 expense money, furnished by Project Viet-Nam.

After four hours of flying, we stopped briefly at Honolulu, and two hours later we were airborne for the 2,300-mile flight to Wake. The other doctors were L. O. Carpenter, an obstetrician from Point Arena, Calif.; Henry Ricci, an ophthalmologist on leave of absence from the Veterans Administration and a former San Angelo practitioner for many years; Lumir F. Novak, a retired general surgeon from San Antonio; William E. Barratt, an otolaryngologist from Painesville, Ohio; and Edward Sheris, a general practitioner from Wilton, N. H., a former instructor in infectious diseases at Johns Hopkins University.

Wake island, an area of seven square miles, half of which is lagoon, was our next stop for about one hour.

Four hours and 2,000 miles later our plane landed at Tokyo, and 1,800 miles later, Hong Kong.

Long renowned as the market place of the Orient, the British Crown Colony and free port of Hong Kong offers the judicious shopper bargains to be found nowhere else in the world. We spent one day and two nights in this city of a million lights.

On Jan. 17 we boarded a four-engined turboprop plane for the 950-mile, three-hour flight to Saigon. The plane was heavily loaded with Orientals, and there was only a handful of anxious Americans on board. One hour out of Hong Kong and high over the South China Sea, our No. 2 port engine had to be feathered, and our arrival in Saigon was delayed 30 minutes. The last half hour was spent flying over the rugged, mountainous terrain of South Vietnam, before landing at Saigon's Ton San Nhut airport. The pilot remained at a high altitude until over the airfield and then began a fast, circling letdown onto the third busiest airport in the world. A war zone was definitely in evidence: fighter-bombers were lined up and down the airfield in sandbagged and concrete bunkers.

We were quickly processed through immigration and customs officials by the friendly USOM representatives who met us. On the drive from the airport to our quarters, it was easy to see how Saigon, with its chic shops and shady tree-lined streets has been considered one of the most beautiful cities in the Orient.

The Project Viet-Nam guest house was a two-story stucco building of French design surrounded by a high concrete wall and steel gate. The guard opened the gate, and an old Vietnamese man helped us unload our luggage.

The high ceilinged house had electric fans and was relatively cool. We were introduced to Vietnam's "33" beer and cautioned not to drink any water in Vietnam unless it had been boiled.

That afternoon, after a cold shower, we were transported to USOM Headquarters downtown. Even though the weather was very hot and humid, the windows of the car were raised three-fourths of the way to prevent the Viet Cong from throwing bombs into the car. Also, at the entrance of all US compounds, a guard searched the automobile and stuck a large mirror under the car to check for hidden explosives. We were briefed by Maj. Gen. James Humphreys, USAID Public Health Division director. That night a reception was given by Dr. Anthony Brown, an English USOM agent and anesthesi-

ologist of the Saigon office for Project Viet-Nam.

Saigon still tries to keep up gaiety and laughter. Artillery rumbles are heard all night long on the outskirts of town. Flare ships and giant searchlights bathe the lovely Saigon River in an eerie light to protect the line of freighters waiting to be unloaded. Army convoys rush through the narrow streets carrying troops, supplies, and ammunition. And yet, Saigon swings! The streets are full of castor oil-burning motorbikes, three-wheeled bicycles, taxis, and throngs of women shoppers wearing the colorful formfitting native Ow-dias.

It was a lovely tropical night with an enormous delta moon silhouetting the tall coconut palms and stretching a silvery path across the swift-flowing Saigon River. A dance was in progress on the roof of the Rex Hotel, an American officer's billet, and we heard music, laughter, and the cranking of the slot machines. Down the river, the artillery had begun pounding and the armed helicopters were going in to attack. Tracer bullets streaking across the sky could be seen, and the flare ships were turning night into day. We returned to our guest house and prepared to spend our first night in Vietnam.

The next morning, after a breakfast of canned ham and powdered eggs, we walked down Cong-Ly Avenue, crossed over to Pasteur Avenue, and visited the heavily armed headquarters of Gen. William Westmoreland, commander of American military forces in Vietnam. After that we went up the street a few blocks to the old Pasteur Institute, still supported and operated by the French.

Drs. Ricci and Barratt were assigned to the medical school in Saigon. They were the only doctors recruited as straight specialists. The other four of us, while specialists, were recruited as general practitioners.

Dr. Novak was assigned to Long Xuyen, a delta hospital with an Australian surgical team commander.

Dr. Carpenter was assigned as a general practitioner to the Province Hospital in Da Nang, 350 miles north of Saigon and an area of tremendous Viet Cong and US Marine Corps fighting.

Dr. Sheris was assigned as a general practitioner to Phan Rang, a small hospital on the coast about

Vietnamese women buy and sell fish in marketplace.

175 miles from Saigon. And I was assigned as a general practitioner to the Khan Hoa Hospital at Nha Trang, a coastal resort city about two hundred miles northeast of Saigon on the South China Sea.

I was very enthusiastic over my assignment, but I had hoped for Hue. I had hoped that I could see my friend, Dr. Capps. I did get to visit with him, however, during Vietnamese New Year's holidays.

At noon on Jan. 18 Dr. Sheris and I were taken to Ton San Nhut airfield to board a US government twin-engined Beechcraft for our journey to Phan Rang and Nha Trang. The plane was overloaded and the temperature exceedingly hot as we climbed steeply out of Ton San Nhut. Soon we passed over Bien Hoa airfield; and on further, American jets and skyraiders were making napalm attacks on the Vietnamese countryside. From the air, it was easy to see that a war was on. The highways don't go anywhere, and the railroads end in a snarl of twisted track just outside of town. The Viet Cong have blown up the bridges, chopped up the pavement, and discouraged travel with electrically detonated mines and masterfully camouflaged ambuscades. Throughout the 700-mile length of Vietnam, one flies; and when he does, he can look down and see the bones of convoys that never made it, the twisted wreckage of the trucks, and the bridgespans dropped in midstream. The roads are cut and cratered and fraught with the danger of ambush, yet the rickety, rattling, incredibly overloaded tourist buses, loaded with Vietnamese natives, still rumble into the provinces, passing scenes of battle and blown-up trucks all the way.

High over the mountainous terrain of South Vietnam, it was easy to see how armies become confused.

The war goes on at many levels, in many varied terrains, with varying degrees of fervor and intensity.

There is the "old war" in the Mekong Delta, with Vietnamese troops doing most of the fighting. There is the "new war," north of Saigon all the way to the 17th parallel, where massive operations by American Marines, cavalrymen, and infantrymen are engaging the Viet Cong.

Last year there were more than 1,000 Americans killed in Vietnam, and during the first four months of 1966, the figure had tripled. Yet the South Vietnamese had 12,000 men killed last year, and I have been told that they have had more than 1,000,000 men killed so far in their battle with the Communists. They have been at this war a long time, some of them for more than 20 years. There are stories that the South Vietnamese won't fight, that he has no heart for the war; this is understandable. The individual American is only here for a year, and he wants to get on with the war. One can't blame the Vietnamese soldier for having a placid, eternally patient Oriental view of things. He will be fighting and dying long after his American counterpart has gone home. You cannot blame him for getting discouraged, disgusted, and just plain tired.

The next two and one-half months of my life would be spent in South Vietnam, practicing medicine in circumstances that I had never encountered before. I had a nostalgic feeling as my plane circled the airfield at Nha Trang.

Top. Saigon streets remain busy amid sounds of gunfire only a few miles distant. Women shoppers, horse-drawn carts, vendors, and motorcycles fill the thoroughfares. Bottom. Project Viet-Nam doctors pausing during stopover in Honolulu are, left to right, Drs. Barratt, Blankenship, Novak, Ricci, Carpenter, and Sheris.

Difficult Conditions in Vietnam Test Resources of Physicians

Dr. Heins: My Tho

MY 60 DAYS in Vietnam were spent at My Tho—42 miles southwest of Saigon in the Mekong Delta. I was the only American civilian doctor in the province of Dinh-Tuong with a population of over 600,000. Most of my time was spent at the Provincial Hospital where I worked with a very efficient Filipino surgical team, taking care of civilian casualties.

The patients were mostly women and children cruelly mutilated by shell fire and bombs. Others had limbs chopped off by the Viet Cong for not conforming to communist ideology.

Our hospital had all the essential supplies and drugs — furnished by USAID. However, we had no blood bank. Because most of the wounded were seen from 6 to 12 hours after injury, many were in shock and died during or after operations.

Sanitation and hygiene as we know it does not exist—not even in the hospital. We had a very efficient American nurse to teach them, but the results were very frustrating.

The Americans are referred to as foreigners, not as friends or allies. Most of the people I believe resent our being there, though the poor and sick were very appreciative.

Our town was in Viet Cong territory, and every night we were surrounded by them. The natives would pay a tax to leave and enter. The outposts that I called on were reached by US helicopter.

Although my work seemed often futile and frustrating, I enjoyed doing it.

I consider my tour as a duty and a privilege.

Dr. Schulze: Bien Hoa

I WAS CONFUSED about Vietnam. Should we be there? Should we get out? So I went to find out. I've never had a trip before where someone else paid the bill.* The idea of the government spending the money on me instead of vice-versa was too good to turn down.

Medically, I was impressed by the large number of unusual (to me) diseases such as cholera, beriberi, plague, strychnine and morphine poisoning, tropic ulcer, anthrax, Pott's disease, malaria (some resistant to all medication including quinine), worms, and trypanosomes. But especially was I impressed by the war casualties.

These civilian casualties accounted for 20 to 30 percent of our work in the province hospital. I still feel that people here do not know or have forgotten what we are fighting for in Vietnam. But if they saw the children, women, and old men brought to the hospital hacked and chopped by hatchets, knives, and clubs; or if they saw these people blown apart by hand grenades and land mines placed where only civilians could possibly be injured; or if they saw pungee stick wounds and crude attempts at decapitation, it would force them to face what happens along the edge where freedom ends and communist methods begin. One sees a clear picture of what communist rule would be like.

Being a psychiatrist—as well as a general practitioner—I worked mornings at the only mental hospital in the country at Bien Hoa. I was surprised at the large number of paranoid symptoms (most unusual here), and at the mildness of violent symptoms among the patients. Handcuffs, straitjackets and other forms of physical restraint which are essential equipment of any mental hospital in the United States were never used. Shock treatments were not given as freely as they are in the United States.

It appears when a Vietnamese becomes psychotic, he does it rather mildly and in moderation. Of more than 2,000 mental patients, only one needed actual restraint—and he was a Frenchman. The Buddhist religion (embraced by more than 70 percent of the population) advises against eating meat "because this practice may encourage the sanguine nature." Perhaps this is part of the explanation?

I was enchanted with the people—so small, so polite, so intelligent, so hard working, so stoic. And although I was there only two months and have been back home for several months, I am still receiving letters and gifts from them. They are extremely poor—as their high rate of tuberculosis would indicate. But even so, they are most generous.

It is true that a little deaf and dumb boy on my ward of brain-damaged children is now named after me. He was the only one with no name, so I asked if he could be called Schulze in Vietnamese. He could: almost exactly the same sound is spelled "Xuan," which means "spring" in Vietnamese. So he is Xuan van Gene. (All boys have the middle name "van" and all girls are "thi.")

Dr. Singleton: Hue

THE CITY OF SAIGON was unbelievable and teeming with all forms of humanity. The streets were filthy, and the gutters were filled with garbage. It was not an uncommon sight to see a person voiding in the gutter, but no one seemed to mind, this being the way of life there.

The filth and mass of confusion which I saw in Saigon was not evident in Hue. Hue was, by contrast, a delightful city and also delightfully cool.

The first item on the agenda my first morning was to meet with the dean of the medical school, an affable middle-aged man who fortunately spoke fluent English. That afternoon I had a meeting with the dean, the professor of internal medicine, and the professor of pediatrics. As time went on it be-

*Dr. Schulze was sponsored by both Project Viet-Nam and AMDOC, Inc.

Vietnamese deaf-mute boy gets a warm greeting from Dr. Schulze. The boy, who had no name before the doctor arrived, was subsequently named Xuan van Gene —Gene Schulze in Vietnamese—in honor of the Project Viet-Nam doctor.

came more apparent that my position there was more political than academic—and it was thought that I could judge the merits of this very new medical college and determine if, in the future, additional support should be given to it.

The curriculum there consisted of two pre-clinical years and four clinical years. All the basic sciences, as well as clinical subjects were included, but the structural components as well as faculty were substandard. There was little financial support for the school from the Vietnamese government, and USOM had not supported the University of Hue because of commitments to the other medical school at the University of Saigon. There is no question that this environment would be much more conducive to an academic atmosphere than the mass confusion which I encountered in Saigon.

I visited one of the hospitals in Saigon, and I believe that Mr. Jan de Hartog (author of the book, *The Hospital*) should visit this area. It was quite an experience to see the unbelievable patient accommodations. Practically every bed had two patients in it, and some beds, three patients. In addition, the entire hospital was filthy. All of the patients appeared to be critically ill, and I suppose that in their condition it did not make a great deal of difference whether they had a bed partner or not. A visit to the x-ray department was also a depressing experience. The quality of the films was unbelievably bad, and the radiologic interpretation was appallingly incorrect.

The main bit of medical news which I received on my arrival was that there was a bubonic plague epidemic, including a number of cases of pneumonic plague. This, in addition to the sound of mortar fire in the distance, added to the unbelievable adventure in which I found myself. The country was unquestionably in a deplorable condition, but the Vietnamese people appeared to be courageous, intelligent, and in dire need of all forms of aid.

One morning an English-speaking medical student picked me up, and for the first time I was able to communicate with the technicians in the x-ray department. They seemed extremely reluctant to change their ways.

The medical aspects of our trip were disappointing. The radiology department at the hospital was frustrating and depressing. The equipment was poor and the technical aspects miserable. Although I had an interpreter with me, the technicians and the local radiologists were reluctant to change the habits and procedures which they had followed for years. An intravenous pyelogram, for example, was a big procedure. The contrast media was old and comparable to the outmoded Diodrast. Compression was used in order to see the ureters, and a post-voiding film was something which they did not understand.

Only a few x-ray examinations were done each day, and the patients were brought from many of the surrounding buildings to the small x-ray department. The fluoroscopic examinations were inadequate, and there was no way to accommodate one's eyes except by sitting in a darkened room. In addition there was no x-ray protection for the technician when he took the radiographs.

The medical students I met were very eager and enthusiastic, but their overall knowledge at their respective levels in medical school seemed inadequate and below our standards. So much more could be accomplished in an educational way if Vietnamese doctors were brought to the United States for training than for a handful of American doctors to go over there.

The political situation is interesting. Dr. Quyen, the dean of the school, had apparently requested aid from USAID, but had not received this support because of

Dr. Gene Schulze shakes hands with a guitarist who is entertaining patients while they paint in the occupational therapy wing of Bien Hoa Mental Hospital. The wing was a gift from nations of the free world.

Top. Attendont shows obsolete x-roy equipment ot the rodiology deportment of the University of Hue. Bottom. Medicol student works in the University's rodiology deportment. Inodequote focilities mode the proctice of rodiology difficult.

other USAID obligations to the University of Saigon Medical School. In addition, Gen. James Humphreys, who is the Public Health Division director of USAID in South Vietnam had refused to give assistance to the University of Hue unless the Minister of Education in Saigon requested such assistance. This had been slow in coming; and in spite of several pleas from the dean, assistance had been denied.

Concomitant with my arrival on the scene there was a request from the Minister of Education for a Project Viet-Nam doctor to go to the University of Hue. A part of the reluctance on the part of the minister of education may have been due to the past association of Dr. Quyen with Premier Diem.

My experience with the radiology department was disappointing. The equipment was obsolete, and there was only one fluoroscopic-radiographic unit. Consequently, the examinations which one can perform adequately were limited. This resulted in no more than three gastrointestinal examinations being scheduled three times a week. In addition, on three days a week two intravenous pyelogram examinations might be scheduled. The rest of the work consisted of a few scattered chest examinations and some work on extremities. Many more examinations could be performed, but the inertia of technical help made it almost impossible to accomplish anything.

One morning I decided to exert my authority by accepting more patients than had been scheduled but promptly received word from one of the doctors who did part-time radiology that I would have to have his permission in the future for such scheduling.

My criticism is not directed at Project Viet-Nam. A radiology assignment in several of the other cities might be much more useful, mainly because American physicians have been there previously. It apparently takes several months before any improvement in medical standards or appreciation of medical ability can be achieved.

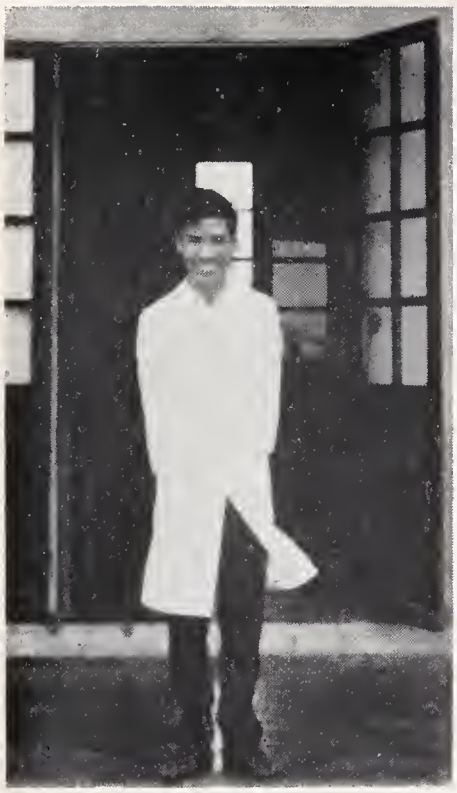
The experience was interesting and in many ways exciting, and I am grateful for the opportunity to have been a part of Project Viet-Nam.

Dr. Blankenship: Nha Trang

IN HAPPIER times Nha Trang would be an idyllic resort, thronged with visitors. Peaceful blue waters lap the curved white sandy beach touted by many as the best in Asia. Out in the bay, palm-covered islands stand sentinel, and at night the sparkling lights of fishing junks dance on the moon-washed purple waters. A curved peninsula holds the harbor in its embrace and beyond it rise the hills that lead into the jungles and to Qui Nhan and Da Nang, far to the north. Docked at the sandline were two large gray United States Navy landing craft with lowered ramps revealing their gaping interior. Along the nearby highway roared the military trucks, off for operations against the Viet Cong. Off to the northwest, a covey of helicopters droned over the hills.

The airfield at Nha Trang was home for the Vietnamese Air Force as well as headquarters for the US Fifth Special Forces, and the Army's Eighth Field Hospital. There were various US Air Force squadrons based there also.

I was met at the airfield by United States Operations Mission (USOM) representative, Sam Taylor, and I was taken to the USOM guest house where I was billeted in a pleasant room with air conditioner and private bath. We then drove the ten blocks to the Province Hospital where I met the Vietnamese medicine chief, Dr. Luc, and the American medical officer



Above. Medical official pauses in front of a dispensary in a small village. He is a high school graduate with a few weeks of first aid training and has returned to his village to help take care of medical needs. Upper Right. Members of US Public Health Service Surgical Team No. 35 pause in front of the Neptune Club at Nha Trang. Lower Right. Several weeks' refuse in garbage dump on Saigon street awaits tardy collection.

in charge, Dr. Richard Novack, a general surgeon from Livonia, Mich. Dr. Luc is a graduate of the medical school in Saigon and has had one year of training at the University of Pittsburgh and two years of surgery training in Australia. He is a capable surgeon and administrator, respected by the American doctors and nurses, and well-liked by the Vietnamese doctors and staff.

Most of the hospital buildings in Vietnam date back to the 1880's. At Nha Trang a new two-year-old hospital had been completed by USOM and additional two-story concrete wings are presently being built. The hospital is being constructed on a quadrangle occupying the equivalent of a city block. A separate building houses the medical, pediatric, surgical, and obstetrical wards. I would guess that the total capacity of the hospital would approach 400 beds.

The pediatric, obstetrical, and medicine wards were supervised almost entirely by capable Vietnamese doctors, working for and paid by the Vietnamese government. There was also a well-educated and likeable dental officer on the hospital staff, Dr. Truc, who had spent six years in the Vietnamese Army as an artillery officer before going to dental school in Saigon. The surgical service was staffed by US Public Health Service Surgical Team Number 35, composed of three medical officers, six registered nurses, a medical technologist, an x-ray technician, and a hospital administrator. One or two Project Viet-Nam doctor-volunteers who rotated in for two months' service also served with the team.

Unaware of my surgical qualifications, Dr. Novack assigned me as the first American medical adviser to the 90-bed medical ward. I was introduced to my American nurse adviser for the medical service, Miss Lynn Allan from Albany, N.Y., who came to Vietnam from Laos, where she had served two years with the Thomas Dooley Foundation. This petite and attractive young nurse was well versed in the practice of native medicine.

My first tour through the medical wards was most revealing. With the aid of an inexperienced Vietnamese interpreter, I saw my first cholera and bubonic plague

Above. Vietnamese waman, martally waunded by martar fire, was brought to hospital at Nha Trang. Right. Three share a bed at USOM hospital in Phan Thiet. The Vietnamese boy in back had received severe burns from mortar white phosphorus. Patients, even those in critical canditian, have bed partners.

patients. Then there were rows of debilitated patients lying on straw mats with such maladies as dysentery, typhoid fever, congestive heart failure, amoebic liver abscesses, parasitic gastrointestinal obstructions, malnutrition, rheumatic heart conditions, tuberculosis, and cancer. The stench, filth, and swarming of flies was nauseating, and I wondered if I would be able to contribute anything good to this deplorable situation. I noted that several of the beds were empty and was told that many of the patients were going home for the Tet holidays. Even though they were on their deathbeds, families had carried them home for the New Year's festivities, and there was little for me to do until after the New Year. My first night in Nha Trang was like the previous night in Saigon, and I was to have many sleepless nights until I adjusted to this new way of life, half way around the world.

The hospital day began at 7 a.m. with surgery ward rounds and then to the operating rooms until noon. The Vietnamese paramedical personnel are great believers in a noon hour rest, and work never resumes until 2:30 p.m. Most of the shops in town are likewise closed during this time of the day. From 2:30 to 5 p.m. the outpatients were seen in the clinic and only emergency surgery was performed. This usually meant that both of our operating rooms were functioning in the afternoon too. After the late afternoon ward rounds were completed, we were free until the next morning except when on call every third or fourth night.

Night call was taken at quarters and usually meant being called to the hospital once or twice each night. An ambulance driver and interpreter would arrive to take us to the hospital. After the emergency patients had been treated, the ambulance would bring us home. It was not uncommon for patients to arrive at the hospital after having been wounded 24 hours previously and having traveled 50 to 75 miles over dirt roads in a horse-drawn wagon or small three-wheeled vehicle. Many of the patients would usually be in profound hypovolemic shock, but would respond dramatically to intravenous fluid therapy.

There was a constant shortage of blood because the Vietnamese were reluctant to donate blood. Then, too, many potential donors were turned down because they were not physically well enough themselves or were possible carriers of blood transmitted infectious agents. Most of our blood was outdated and discarded blood from the US Army Eighth Field Hospital. However, we were most grateful for this 30-day-old blood because it still saved lives. Most of the surgical patients who died did so because of lack of blood.

The standard antibiotic order for all infections was procaine penicillin, 500,000 units intramuscularly twice a day, and streptomycin, 0.5 gm. intramuscularly twice a day. The nurses, as well as doctors, were permitted to order these. I was amazed at the ability of penicillin in small doses to combat severe infections, and I suppose this was due to the relatively recent introduction of antibiotic therapy to the masses in Vietnam and the consequent lack of penicillin-resistant strains of bacteria. I might also add that tetanus antitoxin was given routinely without skin testing. I did not see any allergic reactions to either antibiotics or tetanus antitoxin.

Most of the surgery in the provincial hospital is done under spinal anesthesia, including gastric resections and bowel cases. At my hospital we had a US nurse anesthetist and several Vietnamese anesthetists, all of whom were capable of administering a mixture of ether and air; but this was reserved for chest and head cases. Most trauma patients required orthopedic surgery, but a fair share were considered general surgery and head and neck surgery. Block anesthesia and procaine or Xylocaine Hcl were used extensively.

Not long after I arrived in Nha Trang, headquarters for II corps area, news got around that a maxillofacial surgeon was there. I began receiving official and non-official requests from other province hospitals and from US Army Special Forces camps to perform cheiloplasty on cleft lip children and young adults. There were frantic, pleading, handwritten notes by US Special Forces medics and MACV advisers working in civic action and rural pacification programs, requesting surgical aid "that would greatly enhance American and free world relations in a Viet Cong dominated village." I always tried to help them whenever possible.

A total of 34 aerial flights, in many different types of aircraft and under sometimes difficult flight conditions, were made to most of the important towns and hamlets. Some of the "airfields" were only short dirt strips, high in the mountains, while others were long, aluminum-planked jet bomber runways. Adverse weather conditions and sniper fire to low-flying aircraft were present many times. I will not soon forget the morning my plane was hit by Viet Cong groundfire as we were approaching Qui Nhon. The projectile hit the plane with a deafening impact only six feet from where I was sitting. I luckily escaped serious injury, but I did lose two teeth and suffered a sprained neck accompanied by chills and fever for three days afterwards.

Sanitary conditions in Vietnam are extremely poor. The hospital wards are hot and filthy. Insects predominate because of the filth and lack of window screens. Families accompany the patients to the hospital and sleep with them on straw mats, cook on the floor, and throw the debris out the window.

Brick on the end of a string provides traction for injured Viet Cong prisoner.

Hospital kitchen facilities are practically nonexistent, and food is prepared individually by the family. Water is always scarce and many times not available at all.

Postoperative nursing care was inadequate, and many patients died soon after a successful operation due to nursing neglect. Hospital personnel were among the lowest wage earners; and good, hard working people were hard to find and hold. Restaurant waitresses, bar maids, and taxi drivers made many times more money than a nurse or even a Vietnamese doctor. For instance, the pediatrician at the province hospital had a salary of 13,000 piasters per month, which amounted to $108 in American money. Even worse was a professional Army first lieutenant with six years of service and a salary of about 600 piasters per month or $41 American dollars. A good legitimate restaurant waitress made approximately 100,000 piasters per month or $833 American dollars.

My most thrilling experience in Vietnam was parachuting from Vietnamese aircraft onto Vietnamese drop zones with US Special Forces troops. I am indeed proud of my Vietnamese jump wings and wear them on my uniform knowing that only a few Americans are eligible to do so.

My most rewarding experience was seeing the parents' and children's faces after a cleft-lip repair. Few operations are so dramatic.

My most educational experience came while serving two weeks at the Navy Support Hospital near Da Nang treating wounded Marines. It was heartwarming to know that 99 out of 100 wounded Marines brought into the hospital would live. Yet, it is so disheartening to see just one American die in this far-off land.

It was a unique experience to see the Navy's frozen blood bank in operation. Massive transfusions were given without typing or crossmatching and without adverse blood reactions. War surgery can be shocking to a new service doctor. It gives a strange sensation to see wounded Marines wearing the same unform you are wearing, come into the battle dressing wards with legs torn off, dirt, sand, and blood all over them, maybe an eye gone or half a face missing. This scene tends to take the glory out of war and should make a "would-be-hero" think twice before throwing himself unnecessarily at enemy gunfire. A scene such as I have described makes a man think of his wife and kids back home, safe and sound because people like this are out sweating and bleeding and dying for a cause which we believe is right.

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Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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NEWS

October Public Relations Conference To Study Today's Medical Challenges

New developments and challenges facing the physician in his relations with his patients will be studied at the 1966 Texas Medical Association Public Relations Conference in Austin, Oct. 1. Five main speakers and two panels of speakers will focus attention of present and future problems the physician must deal with in changing society. The Conference will be held at TMA Headquarters Building, 1801 N. Lamar Blvd.

MR. POWERS

John J. Powers will be the first speaker at the Conference. He will speak on "Medical Research — The Years Ahead." The president and chief executive officer of Chas. Pfizer & Co., Inc., has worked closely with the medicinal products research of both the Pfizer company and its international subsidiary, Pfizer International. He has served the Pfizer firm both as president and as vice-chairman. He has also served as the foreign company's president and chairman.

Also a participant in activities outside his company, Mr. Powers is a member of the board of the Pharmaceutical Manufacturers Association and of the board of the association's foundation. He is a member of the board of trustees of the US Council of the International Chamber of Commerce.

The president of the Public Relations Society of America, Robert B. Wolcott, Jr., will speak to the Conference on "Public Relations Is Human Relations." Mr. Wolcott is president of Robert B. Wolcott Associates, a national public relations counseling firm. He established this firm in 1950. Previously, he had held various executive positions with Time, Inc.

MR. WOLCOTT

Active in PRSA affairs for 15 years, he served as vice-president, as a member of the Board of Directors, and as a delegate to the national assembly. He has twice been given the society's Presidential Citation for his leadership in founding the PRSA Counselors Section.

Closing out the opening session of the conference will be Dr. J. A. Sabatier, of Baton Rouge, La., Dr. Sabatier is a member of the American Medical Association Committee on Quackery and president-elect of the Louisiana State Medical Society. He has been for ten years chairman of the Louisiana society's Committee on Public Policy and Legislation. He has conducted a continuing study of the scope of chiropractic in order to evaluate and advise the Louisiana state legislature regarding licensure of chiropractic in Louisiana. He will deliver the address, "The Fight Against Quackery," to the Conference.

DR. SABATIER

The closing session will feature two other out-of-state speakers. John J. Henry of Alexandria, Va., and editor of *Babson's Washington Forecast Letter*, will speak on "Paternal Government — Can We Afford It Financially or Spiritually?" The other speaker will be Roy Pfautch of St. Louis, president of Civic Service, Inc. His speech is entitled "The Flag of Individual Liberty—A Challenge for Action."

MR. HENRY

Mr. Henry has been editor of *Babson's Washington Forecast Letter* since 1952. Before that time he had served as a newscaster and commentator for the American Broadcasting Company and as industrial relations director of the US Time Corporation in Waterbury, Conn. During World War II, he served as a special agent of

the Federal Bureau of Investigation. He has lectured professionally on communism.

For the past 14 years, Mr. Henry has been writer and producer of the "Navy Hour" on the ABC network. He is also the writer, producer, and narrator of the "Navy's Portraits in Patriotism," which has been awarded the national Freedom Foundation Award for the best governmental contribution to patriotism. He is also the originator of the Babson poll, the first national poll to use the switch-vote polling system.

MR. PFAUTCH

Mr. Pfautch, as president of Civic Service, Inc., has specialized in institutional, religious, educational, and political public relations, fund raising, and opinion research. He has managed congressional campaigns in the Midwest, Border States, and South. He has served as a special consultant to the Republican National Committee and to the American Medical Political Action Committee. In 1965 he was appointed editor-in-chief of the *Washington World*, a monthly magazine of political comment.

Mr. Pfautch is also an ordained minister of the United Presbyterian Church in the United States of America. He served as assistant to the president of Princeton Theological Seminary from 1961-63. In 1962 he was director of the Sesquicentennial Celebration of Presbyterian Theological Education and Princeton Seminary.

Nine To Conduct Panels

Besides the five main visiting speakers, the Conference program will feature two panel discussions, "New Federal and State Medical Programs and the Physician" during the second morning session, and "Reaching the Public Through Positive Public Relations" during the first afternoon session.

Four speakers will participate on the morning panel, which will be presided over by Dr. Howard R. Dudgeon, Jr., of Waco, President-elect of Texas Medical Association. Panel speakers and their titles include C. Lincoln Williston, executive secretary, Texas Medical Association, "The First 90 Days of Medicare in Texas"; Dr. Charles Max Cole of Dallas, Speaker of the House of Delegates of TMA, "Title XIX and Texas"; Dr. John F. Thomas of Austin, chairman of TMA's representatives to the State Coordinating Committee for Planning Regional Programs in Heart Disease, Cancer, and Stroke, "The Heart Disease, Cancer, and Stroke Program in Texas"; and Mr. Fred R. Higginbotham, director of public relations, Blue Cross-Blue Shield of Texas, "Kerr-Mills in Texas." A question and answer period will follow this panel presentation.

Dr. Joseph T. Ainsworth of Houston, chairman of the TMA Council on Public Relations and Public Service, will preside over the afternoon public relations panel. The three speakers participating are members of the American Medical Association staff in Chicago. Their names and speech titles are Mr. Jim Reed, director of the AMA Communications Division, "Developing Good News Media Relations"; Mr. Leo Brown, assistant to the executive vice-president of AMA, "Organizing An Effective Speakers Bureau"; and Mr. C. Darrell Coover, director of the AMA Community Relations Department. These speeches will also be followed by a question and discussion period.

Orientation Scheduled

An Orientation Program will be held for new and transfer TMA members at the same time as the Conference. This program will also take place at the Headquarters Building. Orientation program speakers and their subjects are Dr. Elliott Mendenhall of Dallas, vice-chairman of the TMA Board of Trustees, "An Accounting of Stewardship; What Happens to Your Dues"; Dr. James H. Sammons of Highlands, vice-chairman of the TMA Board of Councilors, "Medical Ethics—Obligations of the Physician to His Colleagues and to the Profession"; Dr. Walter Walthall of San Antonio, chairman of the TMA Board of Councilors, "Doctor-Patient Relations"; and Mr. Philip R. Overton of Austin, general legal counsel for Texas Medical Association, "Legal Aspects of Medicine; Malpractice—How To Avoid It."

Both the Conference and the Orientation Program, meeting jointly at some sessions, will run the full day on Oct. 1.

Other Activities Slated

Another of the activities to take place at the Conference will be a TEXPAC breakfast meeting at 7:15 a.m. Saturday in the Crystal Ballroom of the Driskill Hotel. Dr. Milford O. Rouse of Dallas, President-elect of the AMA, will speak.

Physicians, Auxiliary members, and guests will be served a luncheon in the basement of the Headquarters Building immediately following the morning sessions.

Forty TMA committees, councils, and boards will meet at various times and places throughout the Conference weekend. The Executive Board will meet at 9:30 a.m. Sunday, Oct. 2, to consider and act on reports from committee meetings.

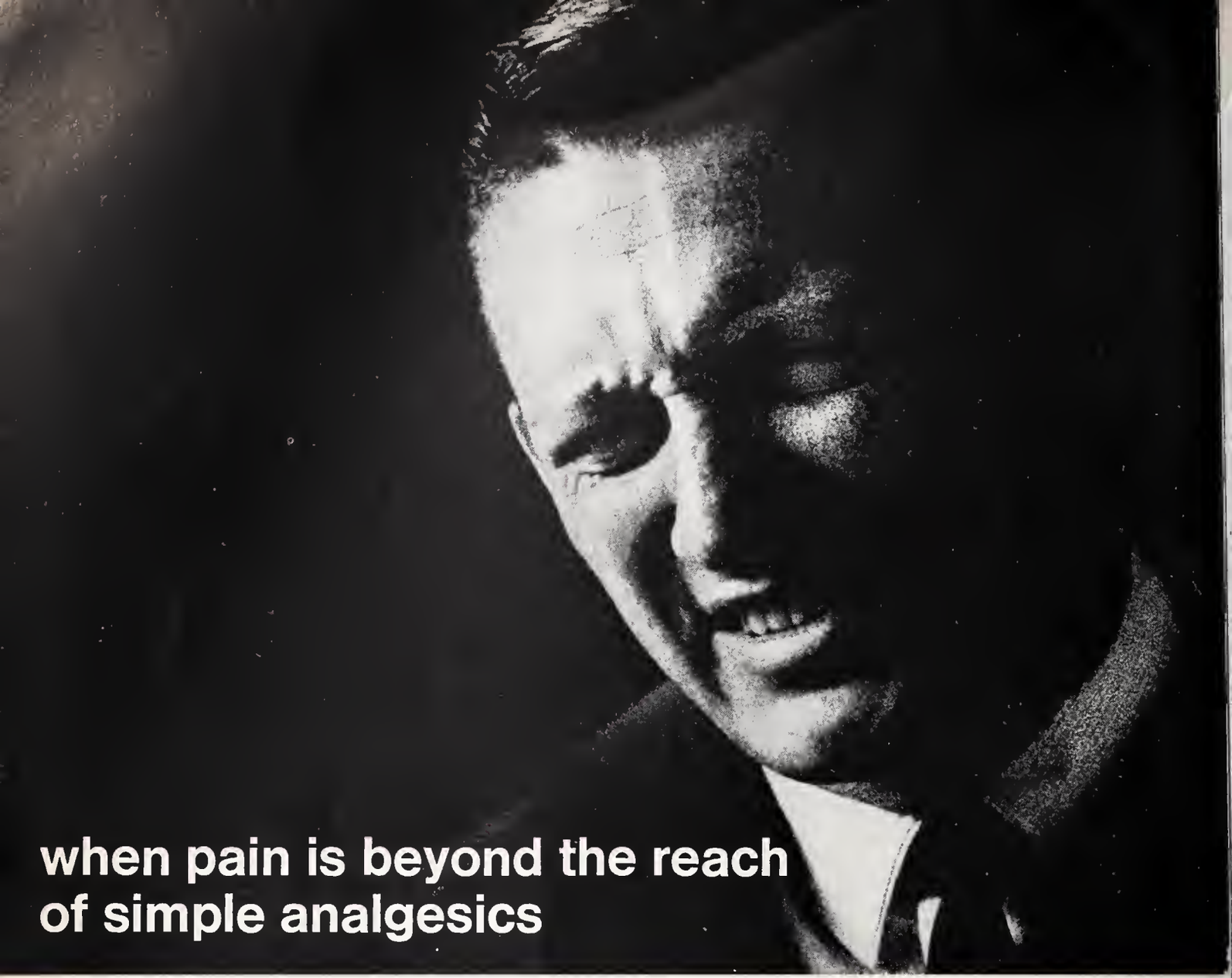
TMA physicians who have served in Vietnam in civilian capacities will be honored at a special presentation during the conference.

At 5:30 p.m. Saturday Charles O. Finley & Company will sponsor a hospitality hour at the Colonnade Room of the Commodore Perry Hotel. Following the hospitality hour at 6:45 p.m. Charles O. Finley & Company will sponsor bus transportation to the University of Texas-University of Indiana football game at Memorial Stadium. The game begins at 7:30. Tickets are $5 and can be purchased from TMA. TMA will furnish bus transportation from the downtown hotels to the Conference in the mornings on Saturday and back to the hotels at the conclusion of the Conference.

Physicians should write to one of the following facilities to make reservations for overnight accommodations: Stephen F. Austin Hotel, Commodore Perry Hotel, Crest Hotel, Driskill Hotel, Chariot Inn Motor Hotel, Terrace Motor Hotel, or Downtowner Motor Inn.

Auxiliary To Convene

The Woman's Auxiliary to the Texas Medical Association will hold a board meeting on Thursday, Sept. 29, and a school of instruc-



when pain is beyond the reach of simple analgesics

In the management of mild to moderate pain, give your patients comprehensive relief. TRANCO-GESIC extends the range of usefulness of aspirin by dimming pain perception and *also* reducing mental and muscle tension.

TRANCO-GESIC®

tablets

chlormezanone 100 mg. with aspirin 300 mg.

subdues the major contributors to pain:

- pain perception
- mental tension
- muscle tension-spasm

TRANCO-GESIC is so well tolerated it can be prescribed for anyone who can take aspirin. It is non-narcotic, and free from dangers of addiction, habituation, or dependence. TRANCO-GESIC is effective in all types of mild and moderate pain. Of 862 patients who were treated with chlormezanone and aspirin for various disorders, 88% reported excellent or good pain relief.[1]

Side effects have been minor. Occasionally gastric distress, weakness, sedation or dizziness occur. Reversible cholestatic jaundice has been reported on rare occasions. However, in 4,653 patients treated with chlormezanone, 97.7% had *no* side effects.[1] *Contraindication:* just one: sensitivity to aspirin. *Dosage:* Adults, usually 2 tablets three or four times daily. Children (from 5 to 12 years), 1 tablet three or four times daily.

1. Collective studies, Department of Medical Research, Winthrop Laboratories.

Winthrop WINTHROP LABORATORIES, NEW YORK, N. Y. 10016

tion on Friday, Sept. 30. Both of these functions will take place at TMA Headquarters Building.

Executive Board members will hear Paul Boesch speak on "The Womanly Art of Self-Defense" at 3 p.m. Thursday. Mr. Boesch is a professional wrestler who makes his home in Houston. He is a television and radio commentator on wrestling and is billed as the "Voice of Wrestling in Texas." He is also a writer. His work includes *The Road to Huertgen*, a chronicle of his experiences in the US Army during World War II, and *The Womanly Art of Self-Defense*, on which his talk will be based.

MR. BOESCH

Also on Thursday afternoon committee workshops will take place. Workshops will include that on recruitment conducted by Mrs. Grover Bynum of Austin, chairman of the Auxiliary Recruitment Committee; and on safety, conducted by Mrs. Phillip L. Day, safety committee chairman. The state treasurer will be available to answer questions on membership dues.

Thursday evening Mrs. James A. Hallmark of Fort Worth, Auxiliary President, will sponsor a hospitality hour at the Gondolier Motor Hotel. The Executive Board dinner will follow at The Barn.

The school of instruction on Friday will be highlighted by Col. Vincent I. Hack of Brooke Army Medical Center speaking on "The Psychology of Color." Col. Hack is chief of the Training Aids Division and officer-in-charge of the Army Medical Museum at the Medical Field Service School, and also Information Officer at the Medical center. He has done portrait painting, oils, etchings, pastels, water colors, and medical photography. Two of his works are in the permanent collection of the Smithsonian Institution in Washington, D.C. During World War II he served in the office of the Surgeon General designing and illustrating for Army medical publications. He has completed experimentations concerning the effects and uses of color.

Other speakers on the Friday program will be Dr. James D. Murphy, president of TMA; C. Lincoln Williston, executive secretary of TMA; and Dr. Robert D. Moreton of Houston, TMA Adviser to the Auxiliary.

Medical Quackery Congress To Be Sponsored by AMA

The Third National Congress on Medical Quackery will take place Oct. 7-8 in Chicago at the Pick-Congress Hotel.

The Congress will be sponsored by the American Medical Association and the National Health Council.

The Congress will again bring together major American groups concerned with efforts to safeguard the public against useless cures, mechanical gadgets, food fads and other quack devices, and worthless treatment.

Interest in public education concerning quackery has been translated into action at a state and community level. Quackery conferences have been held in many states, and more are planned.

The Congress will once again focus the full light of publicity and public opinion on quackery, which costs the American people an estimated billion dollars a year. The theme of the three half-day sessions will be "Medical Quackery: 1966." The final session on Oct. 8 will be devoted to chiropractic. One of the speakers for this session will be Dr. Joseph A. Sabatier, Jr., of Baton Rouge, La. who will speak at the Texas Medical Association's Public Relations Conference on Oct. 1.

Dr. William H. Gordon of Lubbock will also speak at the congress.

Texas Surgeons To Attend Houston Semi-Annual Meet

Members of the Texas Surgical Society will gather in Houston on Oct. 2-4 for the society's fall semi-annual meeting. The meeting will be headquartered at the Shamrock-Hilton Hotel.

Items on the program will include talks by guest speakers, presentations by members, films, council meeting, and various entertainment activities.

Guest speakers include Dr. Frank Glenn, professor of surgery at Cornell University Medical College in New York; Dr. Nihat Tasdemiroglu, surgeon at St. Joseph's Hospital in Houston; and Dr. K. E. Salyer, surgeon at Parkland Memorial Hospital in Dallas.

Entertainment activities will consist of a golf tournament at the Houston Country Club, a cocktail buffet at the Emerald Room of the Shamrock-Hilton Hotel, both on Oct. 2; a men's luncheon at the Continental Room of the Shamrock-Hilton, a ladies' sightseeing tour, and a banquet, all on Oct. 3.

The Texas Surgical Society Council will meet Sunday, Oct. 2.

Academy of General Practice Schedules Boston Assembly

"The Angry Adolescent," a frank exploration of why "kids do what they do," is one of the highlights awaiting family doctors attending the first fall scientific assembly of the American Academy of General Practice, Oct. 10-13 in Boston.

Sharing the Assembly spotlight with the adolescence panel will be other "in-depth" discussions on such topics as stroke, obstetrics and mental retardation, and heart disease; and two half-days of bedside refresher courses at Massachusetts General Hospital and 13 other hospitals.

The four-day program, to be held in Boston's War Memorial Auditorium, will present 31 medical authorities and some 115 scientific exhibits keyed to the program. The scientific program will follow the annual meeting of the Academy's Congress of Delegates Oct. 8-10 in the Sheraton-Boston Hotel which adjoins the auditorium.

Fourteen Boston hospitals will take part in the clinical refresher program. There will be nearly 60 courses offered each day on topics ranging from treatment of cardiac arrest to leukemia and office gynecology. The courses will be conducted for groups of five to 10 participants.

This year's assembly will feature two floors of scientific exhibits in War Memorial Auditorium, and more than 30 lectures in the auditorium. "Therapeutic Nuggets" will offer doctors five ten-minute reports of latest developments in therapy and procedures, ranging from nitrogen mustard injections for arthritis to mediastinoscopy.

Three generations of sportsmen

Dallas bankers Michaux Nash Sr., right, and his son have bagged North American big game trophies from Mexico to the Northwest Territories. And grandson, Michaux III, age 8, already has a fine whitetail buck to his credit! These Texas sportsmen and many others depend on Cullum & Boren for the finest in firearms and hunting clothes. Guns for every quarry...sports clothing for every climate. Next time you're in Dallas, stop in at the two new Cullum & Boren stores: 1609 Bryan, downtown, and in suburban NorthPark.

Cullum & Boren

1609 Bryan 620 NorthPark

12th Annual

PUBLIC RELATIONS CONFERENCE

Saturday, October 1
Headquarters Building
TEXAS MEDICAL ASSOCIATION
Austin, Texas

8:30 a.m. ***REGISTRATION AND COFFEE—READING ROOM***

OPENING SESSION
SAM THOMPSON ROOM

9:00 a.m. ***CALL TO ORDER AND WELCOME***
JAMES D. MURPHY, M.D., Fort Worth
President
Texas Medical Association

9:15 a.m. ***MEDICAL RESEARCH—THE YEARS AHEAD***
JOHN J. POWERS, New York
President
Chas. Pfizer & Co., Inc.

9:45 a.m. ***PUBLIC RELATIONS IS HUMAN RELATIONS***
ROBERT B. WOLCOTT, JR., Los Angeles
President
Public Relations Society of America

10:15 a.m. ***THE FIGHT AGAINST QUACKERY***
J. A. SABATIER, M.D., Baton Rouge
Member, Committee on Quackery
American Medical Association

10:45 a.m. ***COFFEE BREAK—READING ROOM***

SECOND MORNING SESSION
SAM THOMPSON ROOM

11:00 a.m. ***PANEL SESSION: NEW FEDERAL AND STATE MEDICAL PROGRAMS AND THE PHYSICIAN***

Presiding:
HOWARD R. DUDGEON, JR., M.D., Waco
President-Elect
Texas Medical Association

1. THE FIRST 90 DAYS OF MEDICARE IN TEXAS
C. LINCOLN WILLISTON, Austin
Executive Secretary
Texas Medical Association

2. TITLE XIX AND TEXAS
CHARLES MAX COLE, M.D., Dallas
Speaker of the House of Delegates
Texas Medical Association

3. THE HEART DISEASE, CANCER AND STROKE PROGRAM IN TEXAS
JOHN F. THOMAS, M.D., Austin
Chairman, Texas Medical Association's representatives to the State Coordinating Committee for Planning Regional Programs in Heart Disease, Cancer and Stroke

4. KERR-MILLS IN TEXAS
FRED R. HIGGINBOTHAM, Dallas
Director of Public Relations
Blue Cross-Blue Shield of Texas

QUESTIONS AND DISCUSSION

12:30 p.m. ***LUNCHEON—BASEMENT***
Catering by Walter Jetton, Fort Worth
Compliments of the Texas Medical Association

FIRST AFTERNOON SESSION
LIBRARY STACK AREA

1:30 p.m. ***PANEL SESSION: REACHING THE PUBLIC THROUGH POSITIVE PUBLIC RELATIONS***

Presiding:
JOSEPH T. AINSWORTH, M.D., Houston
Chairman
Council on Public Relations and Public Service
Texas Medical Association

1. DEVELOPING GOOD NEWS MEDIA RELATIONS
JIM REED, Chicago
Director
Communications Division
American Medical Association

2. ORGANIZING AN EFFECTIVE SPEAKERS BUREAU
LEO E. BROWN, Chicago
Assistant to Executive Vice-President
American Medical Association

3. EFFECTIVE PARTICIPATION IN THE COMMUNITY
C. DARRELL COOVER, Chicago
Director
Community Relations Department
American Medical Association

QUESTIONS AND DISCUSSION

3:00 p.m. ***COFFEE BREAK—READING ROOM***

CLOSING SESSION
SAM THOMPSON ROOM

Presiding:
JOHN K. GLEN, M.D., Houston
Vice-President
Texas Medical Association

3:15 p.m. ***PATERNAL GOVERNMENT—CAN WE AFFORD IT FINANCIALLY OR SPIRITUALLY?***
JOHN J. HENRY, Alexandria, Virginia
Editor
BABSON'S WASHINGTON FORECAST LETTER

3:45 p.m. ***THE FLAG OF INDIVIDUAL LIBERTY—A CHALLENGE FOR ACTION***
ROY PFAUTCH, St. Louis
President
Civic Service, Inc.

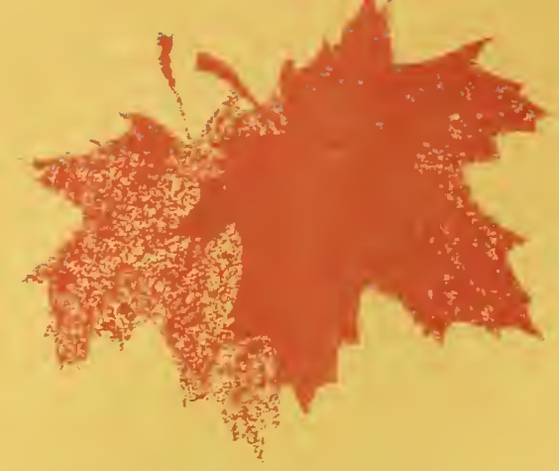
4:30 p.m. ***ADJOURNMENT***

EVENING ENTERTAINMENT

5:30 p.m. ***HOSPITALITY HOUR***
Charles O. Finley & Company, Host
Colonnade Room, Commodore Perry Hotel

6:45 p.m. ***CHARTERED BUSES LEAVE FOR MEMORIAL STADIUM***
Brazos Street Entrance, Commodore Perry Hotel
Courtesy of Charles O. Finley & Company

7:30 p.m. ***FOOTBALL: UNIVERSITY OF TEXAS VS. INDIANA UNIVERSITY***
Memorial Stadium



ORIENTATION PROGRAM

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Austin, Texas

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Compliments of the Texas Medical Association

FIRST AFTERNOON SESSION
SAM THOMPSON ROOM

Presiding:
WENDELL H. HAMRICK, M.D. Houston
Vice-Speaker, House of Delegates
Texas Medical Association

1:30 p.m. ***AN ACCOUNTING OF STEWARDSHIP; WHAT HAPPENS TO YOUR DUES***
ELLIOTT MENDENHALL, M.D., Dallas
Vice-Chairman, Board of Trustees
Texas Medical Association

1:50 p.m. ***MEDICAL ETHICS—OBLIGATIONS OF THE PHYSICIAN TO HIS COLLEAGUES AND TO THE PROFESSION***
JAMES H. SAMMONS, M.D., Highlands
Vice-Chairman, Board of Councilors
Texas Medical Association

2:10 p.m. ***DOCTOR-PATIENT RELATIONS***
WALTER WALTHALL, M.D., San Antonio
Chairman, Board of Councilors
Texas Medical Association

2:30 p.m. ***LEGAL ASPECTS OF MEDICINE; MALPRACTICE—HOW TO AVOID IT***
PHILIP R. OVERTON, LL.B., Austin
General Counsel
Texas Medical Association

3:00 p.m. ***COFFEE BREAK***

CLOSING SESSION
SAM THOMPSON ROOM

Presiding:
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Vice-President
Texas Medical Association

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Memorial Stadium

Medical Assistants Prepare For October Annual Session

Texas Medical Assistants Association members have elected delegates and alternate delegates to represent TMAA in the House of Delegates of the American Association of Medical Assistants. The AAMA annual meeting is scheduled for Oct. 19-23, 1966, at the Chase-Park Plaza Hotel in St. Louis.

Attending in an official capacity will be:

Delegates: Mrs. Gwen Martin, Potter-Randall County; Mrs. Jan Mustonen, Dallas County; Miss Josephine Estrada, Bexar County; and Mrs. Phyllis Goodrich, El Paso County. Alternates are Mrs. Bobbie Smith, Jefferson County; Mrs. Thelma Dahm, Tarrant County; Mrs. Jan Coffman, Tom Green Eight Counties; and Mrs. Yvonne Pace, Harris County.

The entire Texas delegation will host a campaign party for Dr. Travis Smith of Abilene, whom Texas Medical Assistants are nominating as Advisor to the American Association of Medical Assistants. Dr. Smith currently is serving as Advisor to TMAA.

The role of the medical assistant in medicare and the drug abuse law will be discussed at the 10th annual meeting of the American Association of Medical Assistants. Approximately 600 physicians' employees are expected to attend the meeting.

The discussion of the new laws, a symposium sponsored by Wyeth Laboratories, is part of a two-day educational program.

The House of Delegates, the association's policy-making body, will meet Oct. 19 and 20. New officers will be installed Oct. 22, when Mrs. Elvera Fischer, Chicago, is inaugurated as president.

American College of Surgeons Sets October Clinical Congress

More than 12,000 fellows of the American College of Surgeons and guests from many countries are expected to attend the College's 52nd annual Clinical Congress in San Francisco, Oct. 10-14.

Surgical developments in every phase of surgery will be presented during the five-day program through 261 research-in-progress reports; nine postgraduate courses; 42 panel discussions in general surgery and surgical specialties; 107 medical films; 14 operative telecasts from Palo Alto-Stanford Hospital Center in Palo Alto, Calif.; and 425 scientific and industrial exhibits. Approximately 1,100 doctors will be participants in the program.

Major addresses during the week include: Dr. Walter C. MacKenzie, Edmonton, Alberta, Canada, incoming president, "Our Destiny Is To Build"; Dr. Paul A. Weiss, professor emeritus at The Rockefeller University, New York, "The Cellular Mechanics of Wound Healing"; Dr. Tord G. Skoog, professor of plastic surgery, University of Uppsala, Sweden, "The Surgical Treatment of Burns"; and former Arizona governor, Howard A. Pyle, president, National Safety Council, Chicago, "Safety—Preventive Medicine."

US-Mexico Society to Meet

The Medical Society of the United States and Mexico will hold its 11th annual meeting at the Pioneer Hotel in Tucson, Ariz., Oct. 4-7.

For further information write Dr. James Nauman, Chairman, 1603 N. Tucson Blvd., Tucson, Ariz. 85716.

Three-day Annual Conference Planned by Oklahoma Society

The Oklahoma City Clinical Society will hold its 36th annual three-day conference at the Sheraton-Oklahoma Hotel, Oct. 24-26.

The program will include lectures and discussions by 14 speakers selected from various medical and teaching centers throughout the United States. Closed-circuit color television presentations will feature practical office procedures and techniques.

The society has extended invitations to attend to members of county medical societies. Registration fee is $25. For further information write Mrs. Alma O'Donnell, Executive Secretary, Oklahoma City Clinical Society, 2809 NW Expressway, Oklahoma City, Oklahoma 73112.

Endocrine Society Members To Gather in Oklahoma City

The Endocrine Society's Eighteenth Annual Postgraduate Assembly will take place at the Skirvin Hotel in Oklahoma City, Oct. 3-7.

The assembly will be co-sponsored by the University of Oklahoma Medical Center. Recent advances in all phases of endocrinology and metabolism will be discussed with emphasis on diagnostic procedures, pathologic physiology, and clinical management. The program will consist of formal presentations, panel discussions, and question-and-answer periods. A syllabus summarizing the salient features of these presentations will be provided.

Attendance is limited to 250, and each application must include the entire tuition fee of $100 in US currency or money order.

For further information and for applications write to Dr. Henry H. Turner, The Endocrine Society, 1200 N. Walker St., Oklahoma City, Okla. 73103.

Medical Writers to Explore Changes in Communication

"The Changing World of Medical Communication," will be developed in a series of lecture programs, panel discussions, roundtables, and workshop sessions at the American Medical Writers' Association national meeting Sept. 29-Oct. 2. The meeting will be held at the Waldorf-Astoria Hotel in New York City.

Information concerning pre-registration at the AMWA meeting, or membership in the Association may be obtained from the national office, 2000 P Street NW, Washington, D.C. 20036. Executive Secretary of the AMWA is James E. Bryan.

Blood Bank Association To Meet in Los Angeles

Four distinguished figures in blood banking abroad will address the 19th annual meeting of the American Association of Blood

Night Leg Cramps...Frequent Bedfellow

In Diabetes,[1] Arthritis,[2] and Peripheral Vascular Disorders[2]

now...specific therapy for night leg cramps

QUINAMM™

Consistently effective, QUINAMM provided complete relief in 94% of 200 patients studied, many of whom were severe cases refractory to other medication.[3] Your prescription for one tablet at bedtime often controls painful night cramps with the initial dose . . . helps restore restful sleep.

QUINAMM Prescribing Information: Composition: Each white, beveled, compressed tablet contains: Quinine Sulfate 4 grains (250 mg.) and Aminophylline 3 grains (200 mg.). **Precautions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptams of cinchanism, such as tinnitus, dizziness, and gastraintestinal disturbance. Discantinue use if ringing in the ears, deafness, skin rash, ar visual disturbances accur. **Contraindication:** QUINAMM is contraindicated in pregnancy because af its quinine content. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased ta ane tablet following the evening meal and one tablet upon retiring. **Supplied:** Battles af 100 and 500 tablets. **Caution:** Federal law prohibits dispensing without prescriptian.

References: 1. Shumon, C.: Am. J. Med. Sci., 225:54, 1953. 2. Perchuk, E., et al.: Angialagy, 12:102, 1961. 3. Rawls, W., et al.: Med. Times, 87:818, 1959.

WALKER *Laboratories* Division of Richardson-Merrell Inc., Mount Vernon, New York 10551

Banks in Los Angeles, Calif., Oct. 25-28.

The four scholars, who will appear on the scientific section of the program are Dr. W. T. J. Morgan, Lister Institute of Preventive Medicine, University of London, England; Dr. John Murray, Bernhard Baron Memorial Research Laboratories, Queen Charlotte's Maternity Hospital, London, England; Dr. Herbert Pfisterer, Institute of Hematology, Munich, Germany; and Dr. Maurice Shapiro, South African Blood Transfusion Service, Johannesburg, South Africa.

More than 2,000 physicians, technicians, and administrators of community and hospital blood banks will attend the four-day sessions. About 80 papers, dealing with scientific, administrative, and technical subjects will be presented. The papers will cover many of the latest developments in blood banking and modern concepts in blood group genetics.

Texas Pediatric Society To Hold Annual Meeting

The Texas Pediatric Society will hold its annual meeting at the Inn of the Six Flags, Arlington, Sept. 29-Oct. 2.

Speakers for the scientific sessions will be Dr. Louis K. Diamond and Dr. Thomas E. Cone, Jr., both of Harvard Medical School, Boston; Dr. Heinz F. Eichenwald, Southwestern Medical School, Dallas; Dr. Earl J. Brewer, Jr., Baylor University College of Medicine, Houston; Dr. Armond S. Goldman, University of Texas Medical Branch, Galveston; and Dr. Glen E. Austin, Los Altos, Calif.

Anesthesiology Convention Set for Philadelphia, Oct. 1-5

The 1966 Annual Meeting of the American Society of Anesthesiologists will take place Oct. 1-5 at the Sheraton Hotel in Philadelphia.

The meeting schedule will include presentations of papers on scientific work completed, presentations of papers on scientific work in progress, panel discussions, cracker barrel sessions, refresher courses, a closed-circuit television program on "Monitoring for Anesthesiologists," motion pictures on anesthesiology, scientific exhibits, organizational meetings, and a special scientific program for ladies attending the meeting with their husbands. This ladies' program will be moderated by Dr. Thomas K. Burnap, professor of anesthesiology at the University of Texas Southwestern Medical School in Dallas.

May 1-7, 1967, Designated Texas Community Health Week

Texas will be officially observing Community Health Week the week of May 1-7, 1967, in order that the dates may coincide with the 100th Annual Session of TMA.

The Board of Trustees of the American Medical Association has officially designated the week of Oct. 16-22 as National Community Health Week.

Community Health Week is a time for all local members of health professions and health organizations—public, private, and voluntary—to conduct community wide activities emphasizing the continuing theme of the observance—"Teaming Up for Better Health."

Dr. F. J. L. Blasingame, executive vice-president of AMA, said, "The primary objectives of this nationwide observance are to stimulate greater awareness and appreciation of the wealth of health facilities and services which are available at the community level and to stress the health progress and medical advances which have been made locally through the united efforts of all members of the community health team."

The AMA is sending promotional aids to local societies concentrating on two appropriate local themes—the expanding career opportunities in medicine and its allied fields and overcoming preventable disease, with special emphasis on measles immunization and control of venereal disease. These AMA materials should be held for use during the Community Health Week in Texas in May, 1967.

Hospital Benefit To Feature Bob Hope, Fred Waring Show

Comedian Bob Hope, musician Fred Waring, and an Arabian Horse Show will be three feature highlights of the centennial celebration of the Sisters of Charity of the Incarnate Word, founders of Houston's St. Joseph Hospital.

The Sisters of Charity operate several public service organizations in Houston and in other cities. They have been doing their charitable work since 1866.

The entertainment personalities will participate in a benefit show at the Jones Hall for the Performing Arts on Oct. 29. Proceeds will go to St. Joseph Hospital for support of charity patients.

The Second Annual St. Joseph Hospital Charity Arabian Horse Show will be held at Pin Oaks Stables on Oct. 7-9. The show is under the sponsorship of the Gulf Coast Arabian Horse Club.

Three Cardinals will visit the celebration, and a Pontifical Mass will be held. The hospital's St. Anthony Geriatric Center will also be dedicated as part of centennial activities.

Society Regional Conference To Study Cancer in Children

The American Cancer Society's 20th Annual Southwest Regional Cancer Conference will be held Saturday, Oct. 1, at the Inn of the Six Flags, Arlington. The list of speakers will include Dr. Guido Currarino, chief of radiology, Children's Medical Center, Dallas; and Dr. John T. Mallams, professor of radiology, University of Texas Southwestern Medical School, Dallas.

The meeting is co-sponsored by the Tarrant County Medical Society and the American Cancer Society. It runs from 1:30-5:30 p.m. Saturday, Oct. 1, immediately following the Texas Pediatric Society meeting.

This year's conference will deal with the clinical aspects of cancer in children. Subjects to be discussed include potential value of presurgical irradiation in tumors in children, prognosis of the child with cancer, and lumps in children.

Centennial Annual Session Taking Shape

Plans for the program of the Centennial Annual Session to be held in Dallas on May 4-7, 1967, are rapidly materializing. Twenty-seven outstanding out-of-state speakers have already been engaged for the meeting.

In addition to the speakers, closed-circuit television presentations will be sponsored during the Annual Session by Smith Kline & French Laboratories. The Council on Annual Session and Dr. Vernie A. Stembridge of Dallas are supervising the scientific content of the programs to be offered.

Dr. Von Braun To Speak

Three nationally known speakers who will be on the Annual Session program will be Dr. Wernher Von Braun, director of the George C. Marshall Space Flight Center in Huntsville, Ala.; Mr. Bud Wilkinson, president of Lifetime Sports Foundation in Washington, D.C.; and Dr. Charles L. Hudson, president of the American Medical Association. Dr. Von Braun will speak at the May 5 General Meeting Luncheon, and Dr. Hudson will speak at the May 6 Annual Luncheon. Mr. Wilkinson will speak to the Texas Society of Athletic Team Physicians.

Out-of-state guest speakers will also include Dr. Oliver H. Beahrs, head of section, Division of Surgery, Mayo Clinic, Rochester, Minn.; Dr. R. G. Bell, executive director, the Donwood Foundation, Willowdale, Ontario, Canada; Dr. Hugh R. Butt, professor of medicine, Mayo Foundation, University of Minnesota, Rochester, Minn.; Dr. Ramon Castroviejo, New York City; Dr. Robert J. O. Coffey, professor of surgery and chairman, Department of Surgery, Georgetown University School of Medicine, Washington, D.C.; and Dr. Frank D. Costenbader, Washington, D.C.

Other out-of-state speakers will be Dr. Anthony R. Curreri, professor of surgery, University of Wisconsin School of Medicine, Madison, Wis.; Dr. Charles L. Dunham, director, Division of Biology and Medicine, Atomic Energy Commission, Washington, D.C.; Dr. Sydney S. Gellis, professor and chairman, Department of Pediatrics, Tufts University School of Medicine, Boston; Dr. Zeph J. R. Hollenbeck, professor of obstetrics and gynecology, Ohio State University College of Medicine, Columbus, Ohio; Dr. Raymond E. Jordan, professor of otolaryngology and chairman, Department of Otolaryngology, University of Pittsburgh School of Medicine, Pittsburgh, Pa.; and Dr. Albert M. Kligman, Department of Dermatology, Hospital of the University of Pennsylvania School of Medicine, Philadelphia.

Also speaking at Annual Session will be Dr. Chauncey D. Leake, senior lecturer in pharmacology, University of California School of Medicine, San Francisco; Dr. Lee W. Milford, Jr., Campbell Clinic, Memphis, Tenn.; Dr. William B. Ober, Knickerbocker Hospital, New York City; Dr. John D. Porterfield, director, Joint Commission on Accreditation of Hospitals, Chicago; Dr. William H. Saunders, professor and chairman, Department of Otolaryngology, Ohio State University College of Medicine, Columbus, Ohio; Dr. J. G. Scadding, Institute of Diseases of the Chest, Brompton Hospital, London, England; and Dr. Donald B. Slocum, chief of orthopedic sur-

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FOR COMPLETE INFORMATION, FILL OUT AND MAIL THIS FORM

☐ Please send me complete information and application forms for exhibiting at the 100th Annual Session of the Texas Medical Association, May 4-7, 1967.

☐ I am interested in presenting a paper at a TMA Annual Session

on the topic _____

before the Section on _____

Name _____

Address _____

City, State, Zip Code _____

gery, Sacred Heart Hospital, Eugene, Ore.

Additional speakers from out-of-state are Dr. Robert I. Wise, Magee professor of medicine and head, Department of Medicine, Jefferson Medical College, Philadelphia; Dr. Reginald S. Lourie, Department of Psychiatry, The Children's Hospital of the District of Columbia, Washington, D.C.; Dr. Joseph H. Ransohoff, professor and chairman, Department of Neurosurgery, New York University School of Medicine, New York City; Dr. P. V. Siegel, federal air surgeon, Federal Aviation Agency, Washington, D.C.; and Dr. James A. Ferguson, consulting surgeon, Michigan Veterans Administration Hospital, Grand Rapids, Mich.

Manuscript Proposals Due

Deadline for submitting proposals of papers to be presented at the 1967 Annual Session has been set for Oct. 15. Doctors who have papers which they wish considered for presentation at the meeting in May, 1967, should return the form on the preceding page before the deadline date.

Officers of the various sections will select the manuscripts to be read. Manuscripts in the following fields will be considered: digestive diseases, general practice, internal medicine, nervous and mental diseases, obstetrics and gynecology, ophthalmology, otolaryngology, pathology, pediatrics, public health, radiology, and surgery.

Those wishing to display exhibits should also fill out and return the accompanying form.

Scientific exhibits are divided into four categories: (1) individual, (2) group, (3) institutional, and (4) educational-promotional. First, second, and honorable mention awards are given in each of the four categories. A $200 cash prize, the Aesculapius Award for the most outstanding scientific exhibit, will be presented by Mead Johnson Laboratories.

All exhibits will be located in the Dallas Memorial Auditorium. There is no charge for exhibit space. Exhibit applications will be reviewed by the TMA Committee on Scientific Exhibits.

Texas Polio Cases Reach 50; Immunization Program Urged

Texas has reported 50 cases of paralytic polio thus far in 1966. Only three of the cases reported had been completely immunized; but two others had received Type I vaccine, the polio with which they were stricken. None of the five had received booster immunization. Many are infants and preschoolers. On Aug. 20, there were 59 cases in the nation, 45 in Texas.

All persons who have not been properly immunized against polio are still vulnerable to attack. This includes the majority of pre-school children, who have not received full protection. The success of Salk and Sabin polio vaccines have lulled many persons into a false sense of security. They do not realize that vaccines have not eradicated the viruses which cause polio but rather build up antibodies to combat viruses entering the body.

Massive polio immunization programs conducted by county medical societies several years ago immunized the majority of Texas residents. Many persons, however, took only the first dose, and do not have full protection from the dread disease. It is especially important that pre-school children receive the oral polio vaccine.

County medical societies in affected areas, primarily in South Texas, are taking the lead in stepped up immunization programs. The vaccine may, of course, be administered on an individual basis in a physician's office.

Meeting Speakers Available From Bureau of Blue Shield

Members of the Blue Shield Speakers Bureau will discuss health care and its cost and related subjects before church, civic or fraternal groups.

Speakers can also be provided to medical society and medical specialty organizations, with subjects specially tailored for medical audiences.

The National Association of Blue Shield Plans, which coordinates 84 Blue Shield Plans, will handle the details concerning the speakers. Speakers are available without cost; however, in some cases, a contribution toward travel and incidental expenses may be requested, but not demanded. Speakers are based in almost all states and in most major cities.

Four topics of possible interest to lay groups are "What Is Blue Shield's Role in Medicare?"; "How To Conserve the Health Care Dollar"; "What's the Future of Health Care in America?"; and "What Is Unique about Blue Shield?"

Two topics of possible interest to medical audiences are "What's Ahead for Blue Shield?" and "The Physician's Role in Promoting Blue Shield's Growth and Development."

Coronary Diseases To Be Hospital Lecture Subject

The Seventh Annual Methodist Hospital Lectureship in Medicine to be held on Sept. 24 in Lubbock, will study coronary diseases.

Guest lecturers for the event, to be held at the KoKo Palace Convention Center, are Drs. F. Mason Sones, Jr., and Donald B. Effler of the Cleveland (Ohio) Clinic Hospital.

The lectureship will be held in conjunction with the annual Lubbock Surgical Society Lectureship.

TWU Appoints Director In Graduate Nursing Field

Mrs. Opal H. White has been appointed as director of graduate work in the field of psychiatric nursing at the Texas Woman's University College of Nursing. The appointment became effective on Sept. 1.

Mrs. White has been on the faculty of the University of Colorado School of Nursing since 1955. She has served as coordinator of psychiatric-mental health nursing for nine years and assisted in the development of a master's degree program in the field. She is presently completing a dissertation for the Doctor of Nursing Science Degree to be awarded by Boston University.

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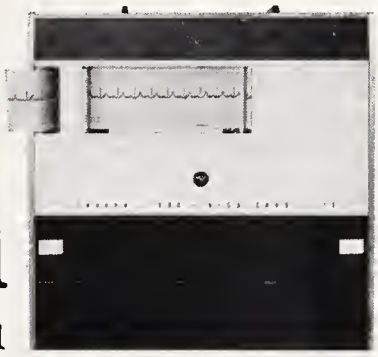
then 

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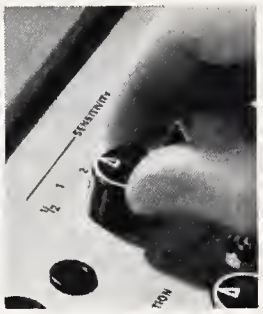

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A Preliminary Report: Insect-Borne Viral Encephalitis in Texas

[Editor's Note: Four officials of the Texas State Department of Health have prepared a current report on outbreaks of encephalitis cases in Texas for *Texas Medicine*. Collaborating to provide the report are Dr. James E. Peavy, Texas State Commissioner of Health; Dr. Hal J. Dewlett, director of Dallas City Health Department; Dr. W. R. Metzger, director of Corpus Christi-Nueces County Health Department; and J. V. Irons, Sc.D., director of Texas State Department of Health Laboratory.]

Dallas Outbreak

DURING THE first week of August, Dr. Jay Sanford, chief of infectious diseases at Parkland Memorial Hospital in Dallas, reported to the city health officer a possible occurrence of encephalitis in Parkland hospital patients. The officer, in turn, reported to the Texas State Department of Health; and blood specimens were submitted.

The first case was confirmed as St. Louis encephalitis by the state health department laboratory on Aug. 9, and the city health officer asked the state health commissioner for assistance from both the state health department and the Communicable Disease Center (CDC) in Atlanta. State personnel were dispatched immediately, and specialists from CDC arrived in a few hours.

Prior to this time, land spraying had been intensified during early July because of flooding caused by spring rains in that area. State health department *Aedes aegypti* eradication crews assisted with the spraying, using DDT.

Specialized teams from Atlanta identified the encephalitis vector as *Culex quinquefasciatus* and recommended aerial spraying. The city health officer then recommended that the state commissioner of health request a special team of US Air Force spray planes through official channels. Six planes were sent immediately to Dallas. Although previous spraying with the specially equipped planes had been done in Puerto Rico, Florida, South Carolina, and on several air force bases, this was the first use of the modernized aerial spraying technique in a metropolitan area for the control of an epidemic of mosquito-borne disease. Spraying began on Aug. 19 and was concluded Aug. 27. The residential area of Dallas, Dallas County, and surrounding area were included.

The insecticide was a technical solution of malathion, distributed in an aerosol spray, using three ounces per acre of land. The particle size with the new technique was adjusted for a range of from 30 to 90 microns. The effectiveness of the insecticide was immediately apparent because of the insect kill and the use of dye cards as coverage indicators.

Because of the incubation period of 5 to 15 days, it was anticipated that additional cases would occur until the expiration of the time period, even though the vector had been controlled.

As of Sept. 1, 120 cases had been reported from Dallas, 13 of which were confirmed, 59 presumptive, and 48 pending. Epidemiological investigators found in retrospect that some cases had occurred as early as July, with the maximum number occurring during mid-August. Most cases were observed among adults, and although the cases first were thought to be concentrated along the Trinity River, it soon became apparent that cases were occurring throughout Dallas County. There was no racial distribution of cases.

The clinical picture presented by typical cases consisted of headache, fever, stiff neck, disorientation and mental confusion, lethargy ranging from mild to semicomatose, nystagmus, and tremors. The older patients were the most severely ill. Spinal fluid showed pleocytosis averaging 250-300 cells, predominantly lymphocytes, with an increase in protein.

Through Sept. 1, eight deaths had been attributed to the epidemic.

Corpus Christi Outbreak

Paralleling the outbreak of encephalitis in Dallas, Corpus Christi also was experiencing an epidemic situation. During the week of Aug. 15, suspected cases on a clinical basis were first reported to the city health officer. Because of the increasing number of cases, assistance was requested of the State Department of Health and the Communicable Disease Center on Aug. 18. Epidemiologists arrived the following day. On Aug. 19, the state health department laboratory reported significant titers of St. Louis encephalitis using hemagglutination inhibition (HI) tests.

Land spraying had been intensified during the week of Aug. 15, converting from a "spraying on complaint" to a routine schedule. Benzene hexachloride (BHC) was used as the insecticide. After consultation with state and federal officials, the Corpus Christi city health officer requested the Air Force also to begin aerial spraying of Nueces County and environment.

Again in Nueces County, the aerial spraying was highly effec-

tive, with almost 100 percent elimination of adult mosquitoes. While cases were continuing to occur as of Sept. 1, it is thought that the peak was reached in mid-August.

As of Sept. 1, there were 13 confirmed cases in Nueces County with 4-fold titer rise with complement fixation tests. Thirty cases were considered presumptive and 56 probable. Two deaths have been reported since the onset of the epidemic. Specialists from the Communicable Disease Center also identified the probable vector in the Corpus Christi outbreak as *Culex quinquefaseiatus.*

Sporadic cases of Western encephalitis have also been confirmed by the state health department laboratory. As of Aug. 30, Hale County in West Texas reported eight cases, four of which were confirmed, with no deaths. Lamb County reported five cases, two of which were confirmed, with one fatal unconfirmed case. A Swisher County resident died of clinical encephalitis, without laboratory confirmation. Lubbock County reported seven suspected cases. A single confirmed case occurred in Tom Green County. The vector of Western encephalitis cases has been identified as *Culex tarsalis.*

As of Sept. 1, the number of cases occurring in Dallas and Corpus Christi had been abruptly reduced, and it was apparent that the control measures had aborted more explosive outbreaks.

Postgraduate Courses

Endocrine and Metabolic Disorders, Brooklyn, N.Y., Nov. 7-11 — The American College of Physicians (ACP) will present a five-day course through the State University of New York Downstate Medical Center.

The purpose of the course is to correlate in a dynamic manner recent advances in the basic sciences and new concepts in the fields of endocrinology and metabolism with clinical endocrine and metabolic disorders.

The course will be divided into 10 half-day symposia devoted to inborn errors of metabolism, lipid metabolism, carbohydrate metabolism, metabolism of electrolytes and water, steroid-metabolism, calcium-metabolism, uric acid metabolism and gout, endocrine interrelations, and disorders of metals and metalloproteins.

Topics within the symposia will include obesity, pre-diabetes, the hypoglycemias, ketotic and non-ketotic diabetic acidosis, and the lipidemias. Each symposium will be followed by a panel discussion conducted by essayists and a moderator. Course registrants will be able to ask questions and make comments.

Tuition fee for ACP members is $60; for nonmembers, $100. Only ACP members are accepted in courses immediately upon receipt of applications. The names of nonmembers are placed on waiting lists in the order received. Three weeks after the release of the course outline (if possible, earlier) nonmembers will be notified whether sufficient room for them remains in the course. They will be officially registered and notified of this registration at that time.

Minimum course registration will be 40; maximum registration, 100.

Government personnel wishing to participate in an ACP course should be registered in the course before requesting authorization from the government agency concerned.

Registration forms and requests for information should be directed to Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine St., Philadelphia 19104.

Newer Aspects of Experimental and Clinical Allergy, Boston, Nov. 14-18.—Another American College of Physicians postgraduate course will be presented through Harvard University Medical School at the Sheraton-Boston Hotel.

This course will center on the pathophysiologic consequences of allergic reactions and some related phenomena and the suppression or treatment of these by pharmacologic and immunological means.

Presentations will be planned so as to bring the experimental and clinical aspects together, with the major emphasis on allergic reactions in man. The atopic disease, anaphylaxis and allergic reactions to drugs will be particularly emphasized.

The course will include a review of the various types of allergic reactions, the role of the various types of antibodies and cells involved, the liberation of chemical mediators and what is known concerning the contribution of all these to the production of tissue injury.

Minimum course registration will be 50; maximum, 200. Information concerning registration and fees is the same as in *Endocrine and Metabolic Disorders* preceding.

Treatment of Common Neurosurgical Disorders, Galveston, Oct. 21.—The University of Texas Medical Branch will hold this second Annual Postgraduate Course in cooperation with Texas Medical Association and the Texas Academy of General Practice on Friday, Oct. 21, in Randall Pavilion.

Three guest speakers and the faculty of neurology, neurosurgery, and psychiatry will participate.

Further information can be obtained from the Department of Postgraduate Education, University of Texas Medical Branch, Galveston 77550.

Population Dynamics, Genetic Counseling, and Birth Control, Denver, Oct. 27-29.—The University of Colorado School of Medicine will offer a three-day postgraduate course. The course is designed to present the background and recent advances in the fields of population dynamics, genetic counseling, and birth control; to point out the implications of these advances that may affect the practice of preventive medicine and public health; and to provide background for a series of follow-up training courses in the techniques and clinical practice of genetic counseling and birth control.

Participation in the course is offered to physicians interested in all aspects of public birth control or genetic counseling clinics, for teachers of obstetrics and gynecology, resident physicians from hospitals where birth control or genetic counseling is not taught, foreign exchange doctors, and other interested persons by special arrangement.

The course will be sponsored by the departments of preventive medicine, obstetrics and gynecology, pediatrics, and biophysics, and the Office of Postgraduate Medical Education. Tuition is $50. A limited number of US Public Health Service Traineeships are available for tuition fees or for per diem expenses. Travel funds are available for foreign graduates in special circumstances.

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For further information, application, and detailed program, write to The Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver, Colo. 80220.

Industrial Medicine: The Doctor's Role in Occupational Health, St. Petersburg, Fla., Oct. 20-22.—A three-day postgraduate course will be sponsored by the Department of Medical Education of the Mound Park Hospital, the Bay Pines Veterans Administration Hospital, the University of South Florida Division of Continuing Education, and the Pinellas County Medical Society.

The course will be oriented to interest physicians particularly concerned with industrial and occupational health, either full or part-time.

Tuition is $40. Registration will be limited. The course may be taken for 18 credit hours acceptable by the American Academy of General Practice. Further information is available from Industrial Medicine, Mound Park Hospital Foundation, Inc., St. Petersburg, Fla. 33701.

Three Postgraduate Education Courses, Philadelphia. — The Department of Medicine of Hahnemann Medical College and Hospital, Philadelphia, is sponsoring several postgraduate courses to take place between October and April. Three of these courses, their directors, and dates are (1) *Rational Therapy and Control of Tuberculosis*, Dr. William S. Blumenthal, Oct. 3-5; *General Internal Medicine*, Dr. Wilbur W. Oaks, Oct. 5, 1966-March 29, 1967; and *Coronary Arteriography* (cosponsored with the American College of Cardiology), Dr. Hratch Kasparian, Nov. 11.

For further information, write the individual course directors at the college, 230 N. Broad St., Philadelphia 19102.

Texas State Department of Health Laboratory Workshops, Austin.—The Texas State Department of Health is sponsoring a number of workshops this fall. These workshops are designed to accommodate workers in public health, hospital, clinical, and other medical laboratories. In most cases, 4 to 12 persons can be accepted in a workshop.

Admittance to all workshops is by advance registration only. There is no registration fee.

Applications should be addressed to Dr. J. V. Irons, Director of Laboratories, Texas State Department of Health, 1100 W. 49th St., Austin 78756.

Workshops to be offered in November include Introductory Bacteriology, Nov. 2-4; and Miscellaneous Serology, Nov. 16-18.

Mycology Workshop, Carlsbad, Tex., Nov. 15-16.—The Texas State Department of Health laboratory personnel and personnel from the McKnight Tuberculosis Hospital in Carlsbad will present a cooperative program. The systemic fungi will be emphasized and the dermatophytes considered.

Applicants for tuberculosis hospital workshops should write to Dr. Robert B. Skinner, Director, Division of Tuberculosis Hospitals, Texas State Department of Health, 1100 W. 49th St., Austin, 78756.

Microbiology, Atlanta, Ga., Nov. 28-Dec. 2.—The Laboratory Branch of the Communicable Disease Center of the US Public Health Service will present a laboratory refresher course in "Preparation of Biological Reagents." The closing date for this one-week course is Oct. 17. The course is one of several being offered during a 12-month period.

Information and application forms may be obtained by writing the Training Office, Laboratory Branch, Communicable Disease Center, Atlanta, Ga. 30333.

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AMA Board Ruling Annuls 1966 Convention Resolution

The American Medical Association Board of Trustees has directed that medical societies should not implement a resolution which would make it unethical "for a physician to displace a hospital-based physician who is attempting to practice separate billing when the displacement is primarily designed to circumvent separate billing."

The Board warned that implementation of the resolution, adopted at the Annual Convention in June, would involve certain legal risks. "Even the successful defense of litigation of this nature is extremely costly and time-consuming, and the results are frequently unpredictable," the Board said.

An ethical principle for a physician could be superseded by a decision on the part of a court or jury that a particular situation caused unreasonable restraint of trade or was otherwise unlawful. The resolution carries with it risks of litigation, including damage claims by persons asserting economic injury.

The Board took this action under a provision of a 1955 resolution which allows the Board to investigate the legal implications of any adopted resolution before it is implemented.

Personals

Dr. Carleton B. Chapman, formerly a professor of medicine at the University of Texas Southwestern Medical School in Dallas and director of the Pauline and Adolph Weinberger Laboratory for Cardiovascular Research, will assume duties as dean of Dartmouth Medical School in Hanover, New Hampshire this month. He is a past president of the American Heart Association and of the American Federation of Clinical Research.

Dr. Louis W. Breck, El Paso orthopedic surgeon, was named Physician of the Year by the Texas Rehabilitation Association at the organization's annual conference awards banquet on July 21 in San Antonio.

Dr. William Wilkinson Taylor of Dallas has volunteered under the

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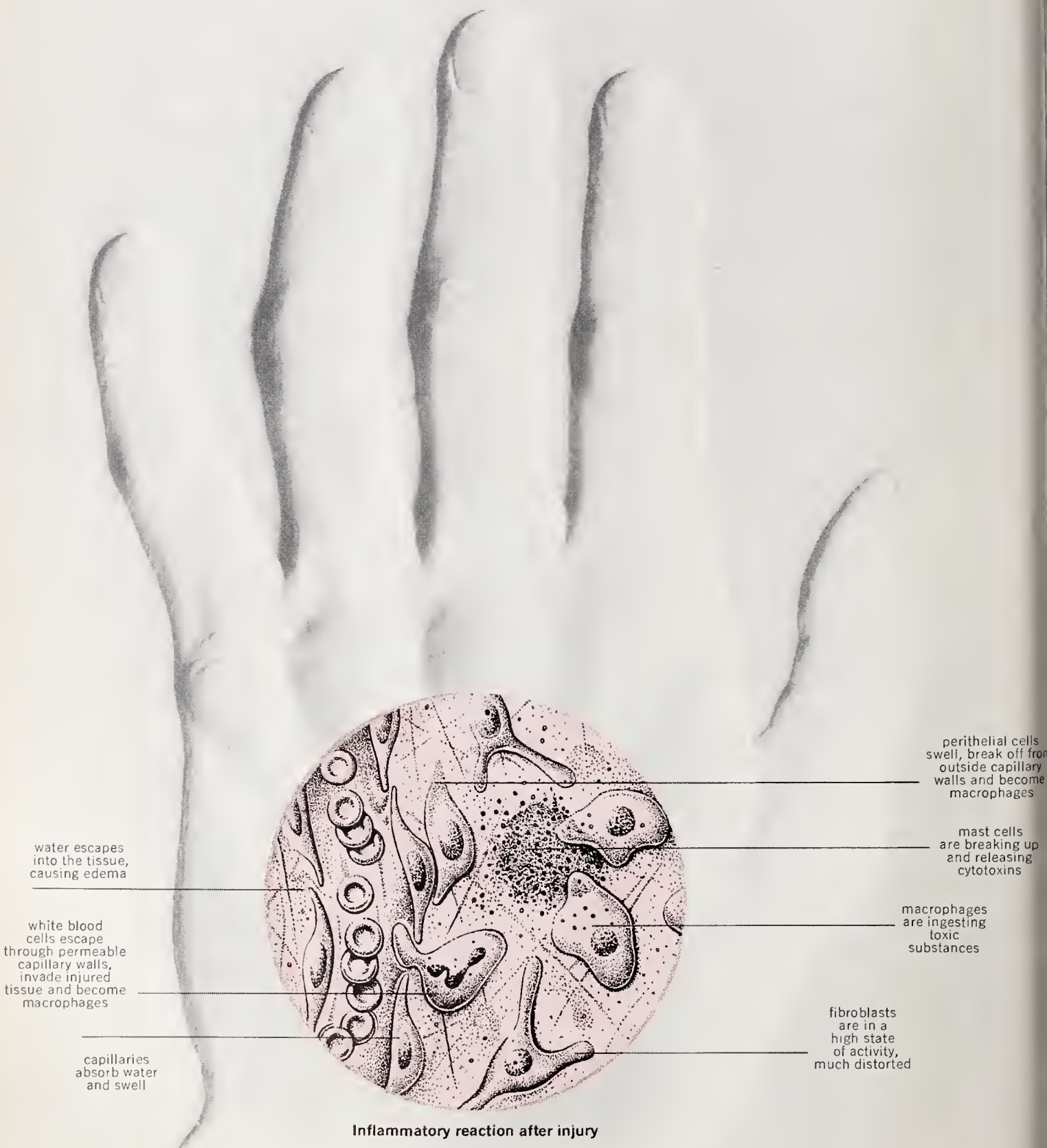
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References: 1. Kanee, B.: Canad Med Ass J 88:999 (May 18) 1963. 2. Scholtz, J. R.: Calif Med 95:224 (Oct.) 1961. 3. Jansen, G. T., Dillaha, C. J., and Honeycutt, W. M.: Arch Derm 92:283 (Sept.) 1965.

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R. H. Chappell, M.D.*

Obstetrics and Gynecology
John Walter Jones, M.D.*
James W. Harrison, M.D.*
Thomas P. Thompson, Jr., M.D.*

ADMINISTRATION
Mrs. Nancy A. Kemp

*Diplomate of The American board of his specialty

**Required training for Certification by the American Board of his specialty has been completed.

San Angelo Medical & Surgical Clinic

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W. V. Smith

American Medical Association Volunteer Physicians for Vietnam program for a 60-day period of civilian service in South Vietnam. He will leave for Vietnam on Sept. 2.

Dr. Joseph G. Murphy of Galveston has been unanimously elected to the Supreme Council of the Knights of Columbus. The announcement of his election was made in August.

Dr. Helen L. Tinnin has been appointed associate professor of health education and assistant to the dean for program development at the University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education. Dr. Tinnin will assume her new duties in September. She was previously at the University of Alabama Medical Center in Birmingham. She received her Ph.D. in Health Education from Ohio State University.

Dr. Truman G. Blocker, Jr., executive director and dean of the University of Texas Medical Branch in Galveston, was recently appointed by President Johnson to the Health Resources Advisory Committee of the Office of Emergency Planning.

Dr. Otto Lippmann of Austin was an official American Medical Association representative to the International Congress of Ophthalmology held Aug. 14-19 in Munich, Germany.

Two women physicians serving residencies in Dallas hospitals have received grants from the Minnie L. Maffett Fellowship Fund of the Texas Federation of Business and Professional Women's Clubs. Recipients are **Dr. Dolores Carruth** and **Dr. Aretta J. Rathmell.** Dr. Carruth will study neonatology at Southwestern Medical School beginning in 1967. Her $5,500 grant is the first full fellowship ever to be given by the Texas Business and Professional Women. Dr. Rathmell will be a fellow in psychiatry at Timberlawn Sanitarium in Dallas. Her grant is for $1,000.

Dr. Vernon Knight of Houston was cited in the Aug. 12 issue of *Medical World News* for his clinical investigations into viral respiratory diseases. Dr. Knight is chairman of the Microbiology Department and head of the Infectious Diseases Division of the Department of Medicine at Baylor University College of Medicine.

Dr. Joseph R. Shaeffer, San Antonio surgeon, died of cancer on Aug. 11. He was a member of the TMA Committee on Disaster Medical Care. He wrote an article about his struggle to keep on with useful work despite his illness; it was published in *Cancer News*, condensed in the Aug. 12 issue of *Medical World News*, and is the subject of a film now being shown by the American Cancer Society "as an inspiration to volunteer workers, physicians, and patients." He was medical director of the Santa Rosa Medical Center.

University Regents Consider Civil Defense Uses of Tower

The Board of Regents of the University of Texas is considering entering into an agreement with the Austin-Travis County Department of Civil Defense for the installation in the Main Building Tower of a Combined Voice Warning, Voice Direction and Siren Tones Remote Speaker Station. This installation would permit the University to use the voice warning system under emergency conditions for University purposes. The Tower would be an ideal central location for the overall Department of Civil Defense warning system in Austin.

W. W. Heath, chairman of the Board, said the Travis County Medical Society also requested the installation of a transmitter antenna atop the Tower to enable it to establish a non-commercial, non-competing radio paging service. This installation would provide a means of relaying messages to all physicians in case of a disaster and a means of directing and controlling medical care between the physicians, the ambulance services, and the hospitals.

Air Force Seeking Doctors To Fill Reserve Positions

The United States Air Force reports that doctors are needed to fill assignments in the Air Force Medical Reserve at Air Force bases throughout Texas. Physicians need not have had prior military service.

Physicians would be required to spend one weekend per month and a two-week summer tour on duty, with pay and allowances.

Interested physicians should write or call the Surgeon's Office, 4AFRR, Randolph AFB, San Antonio 78148. They may also contact the hospital executive officer at the following Air Force bases: Bergstrom, Austin; Carswell, Fort Worth; Ellington, Houston; Dyess, Abilene; Goodfellow, San Angelo; Laredo, Laredo; Laughlin, Del Rio; Randolph, San Antonio; Reese, Lubbock; Sheppard, Wichita Falls; Webb, Big Spring; and Kelly, San Antonio.

Board Schedules Examination

The Texas State Board of Examiners in the Basic Sciences will conduct its next examination on Monday and Tuesday, Oct. 10 and 11.

Details of time and place may be obtained by writing to the Executive Secretary, Texas State Board of Examiners in the Basic Sciences, 1012 Sam Houston State Office Building, 201 E. 14th St., Austin 78701.

New Books In TMA Library

Abramson, H.: *Resuscitation of the Newborn Infant and Related Emergency Procedures: Principles and Practice*, ed., 2, St. Louis, The C. V. Mosby Co., 1966.

Alexander, F., Eisenstein, S. and Grotjahn, M.: *Psychoanalytic Pioneers*, New York, Basic Books, Inc., 1966.

Burdette, W. J.: *Carcinoma of the Alimentary Tract*, Salt Lake City, Utah, University of Utah Press, 1965.

Burdette, W. J.: *Primary Hepatoma*, Salt Lake City, Utah, University of Utah Press, 1965.

Burdette, W. J.: *Viruses Inducing Cancer Implications for Therapy*, Salt Lake City, Utah, University of Utah Press, 1966.

Curry, J. L., and Howland, W. J.: *Arteriography Principles and*

Techniques, Philadelphia, W. B. Saunders Co., 1966.

Collins, V. J.: *Principles of Anesthesiology*, Philadelphia, Lea & Febiger, 1966.

Everson, T. C., and Cole, W. H.: *Spontaneous Regression of Cancer*, Philadelphia, W. B. Saunders Co., 1966.

Flynn, J. E.: *Hand Surgery*, Baltimore, The Williams and Wilkins Co., 1966.

Friedberg, C. K.: *Diseases of the Heart*, ed. 3, Philadelphia, W. B. Saunders Co., 1966.

Gordon, B. L.: *Current Procedural Terminology, Chicago*, American Medical Association, 1966.

Licht, S.: *Orthotics Etcetera*, New Haven, Conn., Elizabeth Licht, Publisher, 1966. (Vol. 1 of Physical Medicine Library.)

Liebman, S. D., and Gellis, S. S.: *The Pediatrician's Ophthalmology*, St. Louis, The C. V. Mosby Co., 1966.

New Orleans Academy of Ophthalmology: *Symposium on Surgery of the Ocular Adnexa Transactions*, St. Louis, The C. V. Mosby Co., 1966.

Sirak, H. D.: *Operable Heart Disease; Pathophysiology, Diagnosis, and Treatment*, St. Louis, The C. V. Mosby Co., 1966.

UCLA Forum in Medical Sciences No. 3: *Brain and Behavior*, vol. 3, Gorski, R. A., and Whalen, R. E., (eds.), Berkeley, Calif., University of California Press, 1966.

Westberg, G. E., and Draper, E.: *Community Psychiatry and the Clergyman*, Springfield, Ill., Charles C Thomas, Publisher, 1966.

Book Notes

Surgery of the Parotid Gland, Robin Anderson and Louis T. Byars, 177 pp. illus., St. Louis, The C. V. Mosby Co., 1965, $12.75.

THE ART and science of surgery is challenged when a surgeon deals with tumors of the parotid gland.

The complications of this surgery pertaining to paralysis of the facial nerve can never be reconciled by the patient nor the surgeon. The surgeon thus needs to be equipped with all the knowledge available to meet this challenge; with basic sources of reference dealing with this particular subject, aside from the knowledge of surgical anatomy and training. The monograph on this subject by Anderson and Byars is an answer to this quest. The authors draw on vast experience on this subject from the surgical anatomy to the management of complications.

This reviewer has assisted in the initial management of a patient with a destructive lesion of the face with marked comminution of the mandible and maxilla and total paralysis of the facial nerve. This injury was produced in a motorboat propeller accident. The patient's lesions were later reconstructed by Anderson and Byars. The end result is a graphic demonstration of their experience.

This source of reference is to be highly recommended to any surgeon who manages tumors of the parotid gland. It is a most thorough study on this subject.

J. J. MATA, M.D., Laredo

"Martha! Have you heard the latest about Mary Brown, Sue Smith, Ann Jones, Joyce Johnson, Sally . . ."

Diagnostic Pathology in Gynecology and Obstetrics, Peter M. Marcuse, 528 pp., illus., New York, Hoeber Medical Div. Harper & Row, 1966, $28.50.

DOCTOR MARCUSE has written a useful book for clinicians practicing obstetrics and gynecology. It is a rather rare work in that anatomical pathology and clinical pathology as they apply to obstetrics and gynecology are under the same hard cover. The two basic divisions of pathology are separated in most texts.

Written in two parts, "Designated Morbid Anatomy" and "Laboratory Methods," the book is well illustrated and has superb photomicrographs and gross pictures of the more commonly encountered obstetrical and gynecological pathologic alterations.

Many well done tables support the written text, and all are in easily readable block form.

Part one on morbid anatomy includes sections on methods and definitions, diseases of the female genital tract organ by organ, and specific disorders of pregnancy including a fairly comprehensive chapter on the placenta which is well illustrated and succinctly written.

Part two includes laboratory procedures related to infectious disease, hematology and endocrinology; the latter section is particularly well written. The scope of section two is not totally related to obstetrical and gynecologic disorders alone, hence, it is a limited primer of clinical pathology.

The book should be well received by the clinician practicing obstetrics and gynecology and would be particularly valuable to obstetrics and gynecology residents during their rotation on pathology since it represents an honest attempt on the part of the author to help the clinician use and understand the services offered by a modern pathology department in the field of obstetrics and gynecology.

R. E. HENDERSON, JR., M.D., Waco

summer 1966

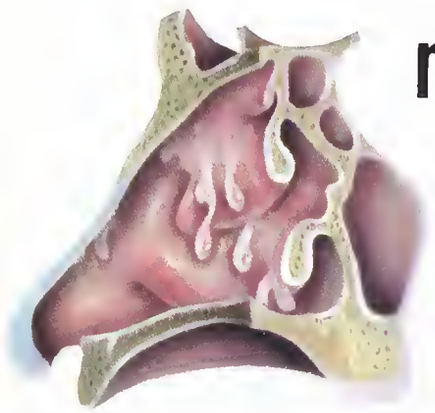
DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M.D., Director of Medicine.

this issue: emergency anesthesia and the common cold

When emergency anesthesia is complicated by the common cold

Barry Belonsky, M.D., F.A.C.A.
Staff Anesthesiologist, Hospital of The Albert Einstein College of Medicine, New York City



Medical facilities are often presented with unfamiliar patients who have unknown health histories. This is particularly true in emergency situations that arise due to accidents or acute illnesses. These cases may need prompt care requiring anesthesia, and if they involve colds, nasal allergies or other upper respiratory infections, can account for many complications which make up a major hazard during emergency anesthesia.

Administration of general anesthesia to a patient with a cold or upper respiratory infection is a hazardous undertaking. It should be avoided if at all possible. Indeed, the presence of U.R.I. is good reason for postponement of elective surgery.[1] In emergency surgery, regional or local block should be considered, but if general anesthesia is mandatory, it should be approached with utmost caution.

Since the attitude of "emergency surgery — hurry" has been replaced by "emergency surgery — watch out",[2] a knowledge of the complications is a great help in preventing them. Here is a brief outline of the problems involved and their treatment. Prevention of the complications is discussed later.

Complications during the induction of anesthesia

Most of the complications are a direct result of secretions and some a result of accompanying secondary infection. For example, *airway obstruction* due to excessive secretions occurs very commonly and is the direct effect of the cold. Respiratory exchange may be obstructed at any time during anesthesia because of excessive secretions, but is most likely to occur during induction. Suction apparatus must be available to overcome this.[3]

Excess secretions which stimulate and irritate the epiglottis and vocal chords can cause *laryngeal stridor and obstruction*. This can lead to complete laryngeal closure with resultant anoxia and death.

Bronchospasm and laryngospasm can result from secretions penetrating the bronchi and bronchioles. In laryngospasm, there are both inspiratory and expiratory stridor and difficulty in inflating the chest; in bronchospasm there is an expiratory wheeze, but not as much difficulty in inflation, although some resistance may be felt. Stridor is due to partial or complete closure of the vocal cords in spasm and the "crowing" sound is almost pathognomonic.

Secretions obstruct the nasal airways. This produces *difficulty in ventilation* through the mouth until the patient is deep enough to place an oral airway. An intravenous agent can be given to facilitate the induction of anesthesia.

Difficulties can arise if intubation is performed to ventilate the patient. For example, teeth can be broken by too vigorous attempts at intubation, or the intubation itself may be technically difficult due to secretions obstructing the view of the glottis. The postoperative sequelae of intubation ranges from mild laryngitis to pneumonia with atelectasis, and are seen far more commonly in patients suffering from colds than in normal patients.

Successive stages of laryngospasm which produce the characteristic stridor or "crowing" sound.

Progression of bronchioles into bronchospasm.

Complications during the maintenance of anesthesia *Bronchospasm* can occur in an unintubated patient due to secretions entering the bronchial tree from above, and acting as an irritant to the bronchi and bronchioles. Secretions accumulate quickly and the patient has to be suctioned continually. The whole cycle of coughing, bucking, laryngospasm and bronchospasm may ensue. The difficult decision here is whether it is better to suction the patient continually or to use an endotracheal tube which protects the cords and bronchi but introduces the risk of attendant complications.

Postoperative complications Postoperatively, complications can be more serious than even the intra-anesthesia complications, and occur much more frequently in a patient who has been intubated.[4]

Sore throat and pharyngitis can result both from the preoperative upper respiratory infection and from the drying of the mucous membranes which occurs during anesthesia.

Tracheitis and bronchitis often result from secretions trickling down the tracheobronchial tree.

Laryngitis is frequently seen in patients with upper respiratory infections who have been intubated. There is a significant increase in the incidence of laryngitis compared to that in patients without upper respiratory infections.

Subglottic edema is a condition which occurs mainly in children who have been intubated. This pathology results from an exudate developing in the areolar tissue just below the cords. Because of the small size of the child's trachea, even a 1 mm increase in size of the mucous membrane can severely impair the air passage. Children exhibit this by severe expiratory stridor and may even become cyanotic. This may so severely embarrass the child's breathing that it must be treated vigorously. Most authorities agree on the treatment[5,6,7,8] consisting of a high oxygen concentration in the inspired air (60%), plus high humidity (close to 100%). Adequate parenteral fluid intake and slight cooling of the body temperature (by a cooled oxygen tent) also help in mild cases. In severe cases, there may be hypoxia which increases the restlessness and the oxygen demand rises. Sedation is often necessary, although concomitant depression of the respiratory center is undesirable. An antihistaminic accomplishes this purpose well, and adds sedation. Since there is always a possibility that an allergic response plays a role in edema, some relief of the respiratory distress may occur. Steroids should be used to control inflammatory and allergic phenomena and swelling. If all this fails, and the patient is still restless and hypoxic, a tracheostomy should be performed immediately.

(concluded on following page)

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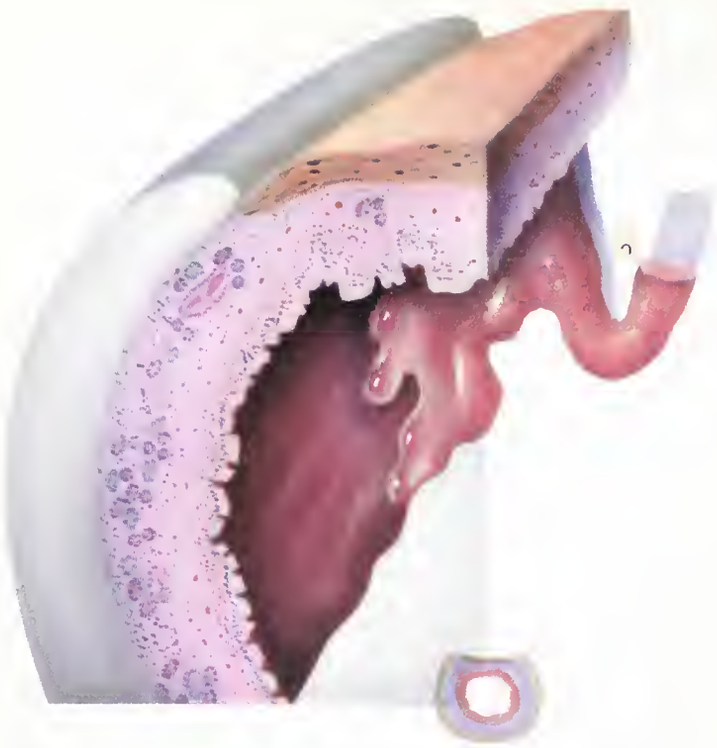

Cross section of trachea showing subglottic edema and lumen reduction due to mucous membrane congestion.

Pneumonia may also follow anesthesia administered to a patient with a cold. This can be caused by accumulated secretions becoming secondarily infected and causing consolidation of the lung. *Atelectasis* of the lung can result if one of the bronchioles becomes plugged by secretions, preventing aeration of the distal part of that lung. This is seen more frequently following upper respiratory infection because dry anesthetic gases aggravate the infection, causing secretions to change from watery to thick and viscid, and consequently difficult to suction.

Prevention of complications The first rule to prevent complications, of course, is to use a regional or local anesthesia whenever possible. But when emergency surgery is a must, in spite of the presence of a cold, allergy, or upper respiratory infection, here are some ways to prevent complications.

Give nose drops preoperatively. This can help shrink the congested nasal mucous membranes and reduce secretions for better air passage. (Results of this method are sometimes unsatisfactory because of the short duration of effect or rebound congestion.) For longer effect, oral antihistamines with nasal decongestants are often given to provide and maintain a drying effect on secretions.

To clear the tracheobronchial tree, instruct the patient to cough preoperatively. Cold steam or water nebulizers effectively humidify the nasal, pharyngeal and bronchial passages and often make the patient more comfortable. Tenacious secretions become more watery under humidification, clear more thoroughly preoperatively and are more easily suctioned from the airway during anesthesia.

Give intravenous fluids to those patients who appear dehydrated due to a cold. In a well hydrated patient the respiratory tract secretions are less viscid and more watery. This is particularly true in asthmatics.

Summary: Administration of emergency anesthesia to a patient with a cold or upper respiratory infection can lead to a chain of events that may result in increased postoperative morbidity and even death. This is because of the excess secretions formed in these conditions. Preoperative measures to prevent or reduce these secretions should be undertaken and will result in smoother and safer anesthesia.

References 1. Ellis, G.: Anesthesia and the Common Cold, Anesthesia, 1955, Vol. 10, p. 78. 2. Smith, R. M.: Anesthesia for Infants and Children, 1963, p. 353. 3. Gillespie, Noel A.: Endotracheal Anesthesia, 1963, p. 155 et seq. 4. Smith, R. M.: Anesthesia for Infants and Children, 1963, p. 353. 5. Written, F. W.: Postoperative Laryngeal Stridor, British Journal of Anesthesia, 1966, Vol. 38, p. 73. 6. Snyder & Gants: Respiratory Obstruction at the Glottic Level, Anesthesiology, 1953, Vol. 14, p. 195. 7. Doming, M. V. & Oech, S. R.: Steroid and Antihistaminic Therapy for Postintubation Subglottic Edema in Infants and Children, Anesthesiology, 1961, Vol. 22, p. 933. 8. Gillespie, Noel A.: Endotracheal Anesthesia, 1963, p. 118.

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Dosage: Adults—2 teaspoonfuls; Children 6 to 12 years—1 tsp.; Children 1 to 6 years—½ tsp. Administer every four hours. Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness, or gastrointestinal upsets. Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

(Advertisement)

Twins: Twice the Trouble, Twice the Fun. Betsy Holland Gehman, 224 pp., Philadelphia, J. B. Lippincott Co., 1965, $4.95.

THIS DELIGHTFUL and breezy book is just what the author claims—"personal" and "practical." The subject is introduced with a brief review of twins in mythology, mysticism, and history. This is followed by factual and accurate medical information concerning twins. Both of these subjects are of real interest to all laymen, twin and non-twin alike.

The meat of the book, however, is Mrs. Gehman's recounting of the constant day-by-day problems which twins present to a mother and her detailed and very helpful suggestions for their solutions. These problems and their solutions include necessary and unnecessary equipment, feeding, time savers, energy-savers, accident warnings, and even precise instructions on the selection of a baby-sitter.

The author concludes with a series of interviews with twins and with families of twins. In these interviews is the same theme which is woven throughout the book: twins are a very special group, but each twin is an individual.

DOROTHY WYVELL, M.D., Midland

Physiologic Foundations for Marriage Counseling. Joseph B. Trainer, M.D., 287 pp., St. Louis, C. V. Mosby Co. 1965. $8.00.

THE AIM of this book is to provide solid information on the biology and medicine of sex, marriage, and reproduction. The topics discussed attempt to include all the major problems people may have to deal with in marriage. Therefore the book includes not only the physiology of sex but the essential qualities that the human brings to the association. It deals with the related problems of contraception, infertility, and abortion, and it introduces the potential problems of development and genetics.

Recent Deaths

DR. T. C. STRICKLAND

Dr. Thomas C. Strickland, 73, who practiced in Greenville for 38 years, died in Dallas, on May 11, 1966, following a brief illness. He specialized in treatment of eye, ear, nose, and throat disorders.

Dr. Strickland was born on Oct. 3, 1893, in White Rock, north of Greenville. His parents were Dr. L. E. and Lena Jones Strickland.

Dr. Strickland was graduated from Wesley College in Greenville and from North Texas State Normal College (now North Texas State University) in Denton. He received his medical degree from Baylor University College of Medicine, then located in Dallas, in 1923.

DR. STRICKLAND

He had practiced in Marshall, Fort Worth, and Greenville. He had been a member of both the Hunt and Tarrant Counties Medical Societies. He served the Hunt County Medical Society as secretary in 1933 and was president in 1940. He was a member of Texas Medical Association and the American Medical Association.

Dr. Strickland was a member of Central Christian Church in Greenville, of the Rotary Club, and of the Masonic Lodge. He was medical advisor to the Missouri, Kansas, and Texas Railroad in Greenville.

In 1923, Dr. Strickland married Sybil Love in Dallas. Mrs. Strickland died in 1963 in Greenville.

Survivors include Mrs. James Owens of Greenville; and two sisters, Mrs. J. W. Ward of Greenville and Mrs. Lina Fitzgerald of Oyster Bay, New York.

DR. E. P. HUTCHINGS

Dr. Edgar Powell Hutchings, president of the Board of Trustees of the Torbett, Hutchings, Smith Memorial Hospital in Marlin for 16 years, died on July 1, 1966, at the age of 73. Cause of death was coronary infarction.

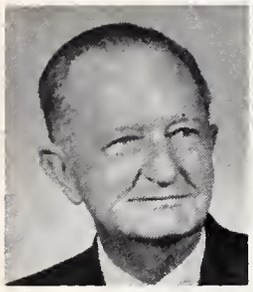
Dr. Hutchings came to the Torbett Sanatorium and Clinic in 1920. He was president of the board of the Torbett Clinic and the Torbett Hospital (now the Torbett, Hutchings, Smith Memorial Hospital and Torbett, Hutchings, Smith Clinic) beginning in 1949. He retired from that office on Sept. 20, 1965.

Dr. Hutchings was a member and former president of the Fall County Medical Society. He was a member of Texas Medical Association, the American Medical Association, the American College of Surgeons, and the Texas Society of Ophthalmology and Otolaryngology.

He was a director of the Marlin National Bank and a member of the First Baptist Church in Marlin.

Dr. Hutchings was born on July 27, 1892, in Marlin. His parents were Edgar P. Hutchings, Sr., and Lucy L. Lamkin Hutchings. He received preliminary education in the Marlin public schools and academic education from Vanderbilt University in Nashville, Tenn.

He entered Vanderbilt University School of Medicine in 1912 and



DR. HUTCHINGS

received his M.D. degree from that school in 1917. He interned at St. Louis City Hospital in 1917 and 1918 and served his residency at the Manhattan Eye, Ear, Nose, and Throat Hospital in New York City in 1922.

From 1918 to 1919, Dr. Hutchings served as a first lieutenant in the US Army Medical Corps in France and Germany.

Dr. Hutchings took postgraduate work at New Orleans Eye, Ear, Nose and Throat Hospital in 1924; Barnes Hospital in St. Louis in 1936; and Manhattan Eye, Ear, Nose, and Throat Hospital in 1935.

In December of 1937, Dr. Hutchings married Glenn C. Mitchell in Waco. Mrs. Hutchings died on Nov. 24, 1955.

Dr. Hutchings is survived by a nephew, Grady Hutchings, Jr., a member of the US Marine Corps in Camp Pendleton, Calif.

DR. T. H. COBBLE

A physician in Rusk for 60 years, Dr. Thomas Henderson Cobble, died July 2, 1966, in Rusk of a heart attack. He was 83.

Dr. Cobble, a general practitioner, had patients "from river to river," the Neches River on the west and the Angelina River on the east. He made his first calls on horseback and later in a horse-pulled buggy. Having delivered 5,500 babies during the years of his practice, he was called the "Dean of Doctors" by citizens in Cherokee County.

Dr. Cobble was born on Sept. 2, 1882, in Rusk. He was the son of Daniel F. and Mary Elizabeth Cobble. He attended Rusk public schools and East Texas Baptist Institute in Rusk. He completed his premedical educational work at Washington University in St. Louis. He received his M.D. degree from the University of Louisville School of Medicine, Louisville, Ky., in 1904.

After completion of medical school, Dr. Cobble practiced two years in Mabank, Tex. He then moved to Rusk in 1906 to begin his 60 years of practice there.

In December, 1911, Dr. Cobble married Addie Coupland in Rusk.

Dr. Cobble was a member of Cherokee County Medical Society and had been secretary of the society for 30 years. He was a member of the American Medical Association and of Texas Medical Association, being elected to honorary TMA membership in 1962. He was the county health officer for Cherokee County.

Active in community affairs, Dr. Cobble was a member and past president of the Rusk Kiwanis Club. He served for 42 years on the Selective Service Board of Cherokee County and received commendatory citations from Presidents Franklin Roosevelt, John Kennedy, and Lyndon Johnson. He was a member and past chairman of the Board of Stewards of the First Methodist Church of Rusk. He was a member of his Masonic lodge for 47 years.

Dr. Cobble was the first chief of staff of the Rusk Memorial Hospital in 1949. He had also been a staff physician at Nan Travis Memorial Hospital in Jacksonville, Tex.

Dr. Cobble is survived by his wife and three daughters, Mrs. Knox Ray and Mrs. Angelina Spivey, both of Rusk, and Mrs. George P. Francis of Austin.

DR. COBBLE

DR. R. G. LOVELESS

Dr. Roy Gordon Loveless, 69, an ophthalmologist in Lubbock, died there on July 16, 1966, after an illness of several years.

Dr. Loveless had practiced in Lubbock from 1945 to 1964 when he retired. He had previously practiced in Slaton, Lamesa, and Dallas. He was a member of Lubbock-Crosby-Garza Counties Medical Society. He had been president of the Lubbock-Crosby Counties Medical Society in 1950 and secretary in 1948. He had also been a member of Dallas County and Dawson-Lynn-Terry-Gaines Counties Medical Societies. Dr. Loveless had been a member of Texas Medical Association since 1931 and was elected to inactive membership at the 1966 Annual Session. He was also a member of the American Medical Association.

Active in community and civic affairs, Dr. Loveless was a member of the board of stewards of St. John's Methodist Church. He had been a member of the Slaton Rotary Club and served the organization as president in 1935. He was a member of the Lubbock Downtown Rotary Club after moving to Lubbock and was named honorary life member in May, 1966. He had been a York Rite Mason since 1933.

Dr. Loveless was born on June 4, 1897. His parents were John Quincy and Lula Alma Gordon Loveless. He graduated from Hillsboro High School and did premedical work at Southern Methodist University. He received his M.D. degree from Baylor University College of Medicine in 1930.

After serving a year's internship at St. Paul's Hospital in Dallas, Dr. Loveless began his practice in Lamesa. In 1932 he moved to Slaton where he practiced until 1944. He then practiced for one year in Dallas and began his practice in Lubbock in 1945.

Dr. Loveless served as a first lieutenant in the Reserve Medical Corps from 1930 to 1940. He did postgraduate work at Chicago Eye, Ear, Nose, and Throat Hospital in 1945. He served on the staff of St. Mary of the Plains Hospital and was associated with the Plains Clinic for 20 years.

In 1926, Dr. Loveless married Anna Katherine Bills in Dallas. Mrs. Loveless survives her husband as do two daughters, Mrs. Martha Cook of Fort Worth and Mrs. Ben Terry of Corpus Christi; a brother, George W. Loveless of Lubbock; and a sister, Mrs. Alma Brewer of Dallas.

DR. LOVELESS

DR. E. D. McDONALD

Dr. Earl Dayton McDonald, a physician who had practiced in Santa Anna, Fort Worth, Dallas, and Arlington, died in Arlington on July 20, 1966, of congestive heart failure and lymphocytic leukemia.

Dr. McDonald was born on June 18, 1900, in Woodville, Ohio. He was the son of W. W. and Myrtle Bell McDonald.

Dr. McDonald graduated from high school in Robinson, Ill. He attended Southern Methodist University in Dallas for his premedical work. He received his medical degree from Baylor University College of Medicine, then located in Dallas, in 1928. He served his internship at St. Louis City Hospital in 1928-29.

Dr. McDonald, a general practitioner, had been a member of Coleman County, Tom Green Eight, Dallas, and Tarrant Counties Medical Societies. He served the Coleman County Medical Society as president in 1932 and as secretary in 1934-1937. He was president of the Fourth District Medical Society in 1933 and secretary in 1932. He was also a member of Texas Medical Association and of the American Medical Association. He was a former and the first chief of staff at Arlington Memorial Hospital.

Frequently active in community affairs, Dr. McDonald had served as Mayor of Santa Anna from 1934 to 1938. He was a past master of the Mountain Masonic Lodge 661 at Santa Anna. He was one of the founders and the first president of the Arlington Kiwanis Club, and was president of the Arlington Medical and Dental Society at the time of his death.

Survivors of Dr. McDonald include his wife, Mrs. La Vinia Jessie McCarter, whom he married in 1952 in Arlington; a son, Earl D. McDonald, Jr., of Dallas; two step sons, Jerry H. McCarter of Richardson and James M. McCarter stationed in Da Nang, South Vietnam; his mother, Mrs. W. W. McDonald of Dallas; a sister, Mrs. Martha Bergen of Dallas; and two brothers, Lee McDonald of Kilgore and K. G. McDonald of Longview.

DR. McDONALD

DR. JOHN THOMPSON

On July 25, 1966, Dr. John Thompson died of an acute myocardial infarction at his home in McGregor.

Dr. Thompson, a general practitioner from a family of several doctors, had been a physician in McGregor for 35 years, from 1931 to 1966. He had practiced in Houston from 1928 to 1930. He had been active in McGregor affairs, serving on the McGregor School Board from 1937 to 1948. He was a member of the Rotary Club and was a past president. He was also a member of Ridgewood Country Club.

He was a member of the McLennan County Medical Society, Texas Medical Association, the American Medical Association, and the American Academy of General Practice.

Dr. Thompson was a graduate of what is now the University of Texas Medical Branch in Galveston in 1926. He had attended St. Andrews College in Toronto, Ontario, Canada; Sheffield Scientific School at Yale University in New Haven, Conn.; and the University of Texas at Austin. He interned at the Postgraduate Hospital in New York City in 1927-28.

Dr. Thompson was born in Galveston on Oct. 13, 1899. His parents were Dr. James E. and Elenor Roeck Thompson. Dr. James Thomson was a British surgeon who came to the United States in order to teach at the University of Texas Medical Branch. In 1931 Dr. John Thompson married Hester Baudoux in Ardmore, Okla.

Survivors include Mrs. Thompson; a stepson, Charles Baudoux of Fort Worth; three brothers, Drs. Edward Thompson of Galveston, Frederick Thompson and James E. Thompson, both of New York City; and four sisters, Mrs. Stanley Phillips of Charlottesville, Va., Mrs. J. L. Mosle of Houston, Mrs. Donald Heigeluth of New Haven, Conn., and Mrs. Henry Cave of New York City.

DR. THOMPSON

DR. R. L. VINEYARD

Dr. Roy Leonard Vineyard, 80, a practicing physician in Amarillo for 43 years, died there on July 28, 1966, following a long illness.

Dr. Vineyard had been president of Potter County Medical Society and a member of Texas Medical Association. He was a member of First Baptist Church in Amarillo and was a 32nd degree Mason.

The youngest of nine children, Dr. Vineyard was born on July 17, 1886, in Dacula, Ga. His parents were George Stephen and Mildred Caroline Drumond Vineyard. The family moved to a ranch in the Texas Panhandle when Dr. Vineyard was three years old.

Dr. Vineyard received his M.D. degree from the University of Texas Medical Branch, then the School of Medicine, in Galveston in 1913. He had premedical training at the University of Texas in Austin from 1905 to 1908. He served his internship and residence at Scott and White Memorial Hospital in Temple. He then joined his brothers, Dr. G. T. Vineyard and Dr. S. P. Vineyard in partnership in Amarillo in 1914. He did postgraduate study at Guy's Hospital in London after World War I.

During World War I, Dr. Vineyard served with the British Army as a medical officer. He attained the rank of major.

On May 1, 1915, Dr. Vineyard married Vesta Francis Cooper in Temple. Mrs. Vineyard survives; as do a son and daughter, Roy L. Vineyard, Jr., of Amarillo and Mrs. Doris Hodde of Corpus Christi; a brother, John Vineyard of Midland; and four grandchildren.

DR. VINEYARD

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Breast-feeding and the "modern mother"

Despite a mild resurgence of interest in the importance of breast-feeding a few years ago, many women today do not choose to nurse their young. This is for a variety of reasons—social, economic, cultural and sometimes medical. In such cases the physician's task is to find the most suitable means of preventing lactation and easing the pain of breast engorgement.

The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

However, factors other than effectiveness must also be considered. The agent selected should not interfere in any way with parturition, subsequent uterine involution and the restoration of normal ovarian cyclic function. Furthermore, it should not cause rebound breast engorgement or other manifestations of hormonal imbalance.

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Providing single-dose therapy for the prevention of lactation and breast engorgement, Deladumone OB is a potent androgen-estrogen combination with a prolonged action. The optimal balance of androgenic and estrogenic hormones achieved in this preparation minimizes the disadvantages inherent in single hormone therapy, such as rebound breast engorgement. Involution of the uterus and resumption of menstrual cycles are not affected.

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Dosage:

As a single injection of 2 cc. before the onset of the second stage of labor.

Contraindications:

Established or suspected mammary cancer or genital malignancy.

Precautions and Side Effects:

Certain patients may be unusually responsive to either estrogenic or androgenic therapy. In such individuals virilization, uterine bleeding or mastodynia may occur.

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Deladumone OB, providing 180 mg. testosterone enanthate and 8 mg. estradiol valerate per cc., is available in 2 cc. Unimatic® disposable syringes and in 2 cc. vials. Both preparations are dissolved in sesame oil, with 2% benzyl alcohol as a preservative. *Before use, consult product literature for full prescribing information.*

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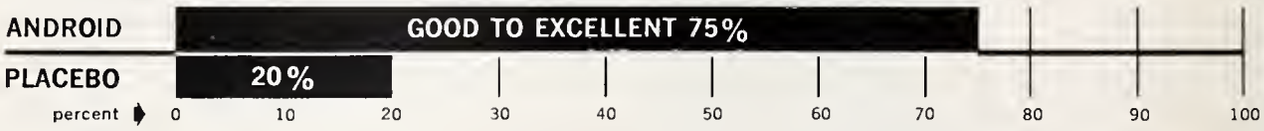

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1. Forty cases reported.
2. Excellent to good results, 75% with Android, 20% with Placebo.
3. Cites synergism between androgen and thyroid.
4. No side effects in patients treated.
5. Alleviation of fatigue noted.
6. Case histories on 4 patients.
7. Although psychotherapy still needed, role of chemotherapy cannot be disputed.

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Chlorobutanol 0.15%
In Isotonic Solution

COMING MEETINGS

American Medical Association, Clinical Meeting, Las Vegas, Nov. 27-Dec. 1, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Austin, Public Relations Conference, Oct. 1, 1966; Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

SEPTEMBER

American Society of Clinical Pathologists, Washington, D.C., Sept. 16-24, 1966. Dr. Lester H. Hoyt, Methodist Hospital, Indianapolis, Ind. 46207, Sec.

Texas Pediatric Society, Fort Worth, Sept. 29-Oct. 1, 1966. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

OCTOBER

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966. Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. Charles R. Ebersol, 219 E. 42nd St., New York 10017, Sec.

American College of Gastroenterology, Philadelphia, Oct. 23-26, 1966. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, Houston, Oct. 24-26, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Surgeons, Annual meeting, San Francisco, Oct. 9-14, 1966. Dr. Samuel P. Harbison, 55 E. Erie St., Chicago, Ill.

American Heart Association, New York City, Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Philadelphia, Oct. 1-5, 1966. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio, Sec.

Texas Surgical Society, Houston, Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Association of Obstetricians and Gynecologists. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill., Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Nutrition, Dr. Robert A. Peterman, 3 Craig Court, Totowa Borough, N. J., Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, February, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American Congress of Physical Medicine and Rehabilitation, Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., Sec.

American Gynecological Society. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Hospital Association, Chicago, Aug. 21-24, 1967. Mr. James E. Hague, 840 N. Lake Shore Drive, Chicago 11, Sec.

American Medical Women's Association, Washington, D.C., Nov. 2-5, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Surgical Association. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis, Ind., Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Gastrointestinal Research Forum, Chicago, Nov. 4, 1966. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104 Sec.

Gastrointestinal Research Group, Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Private Clinics and Hospitals, Austin, December, 1966, A. A. Hubbard, Alvarado Community Hospital, Alvarado, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La., Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southern Surgical Association, Boca Raton, Fla., Dec., 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore, Md.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston, Pres.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of General Practice, Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Division, American Cancer Society, Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Heart Association, Houston. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

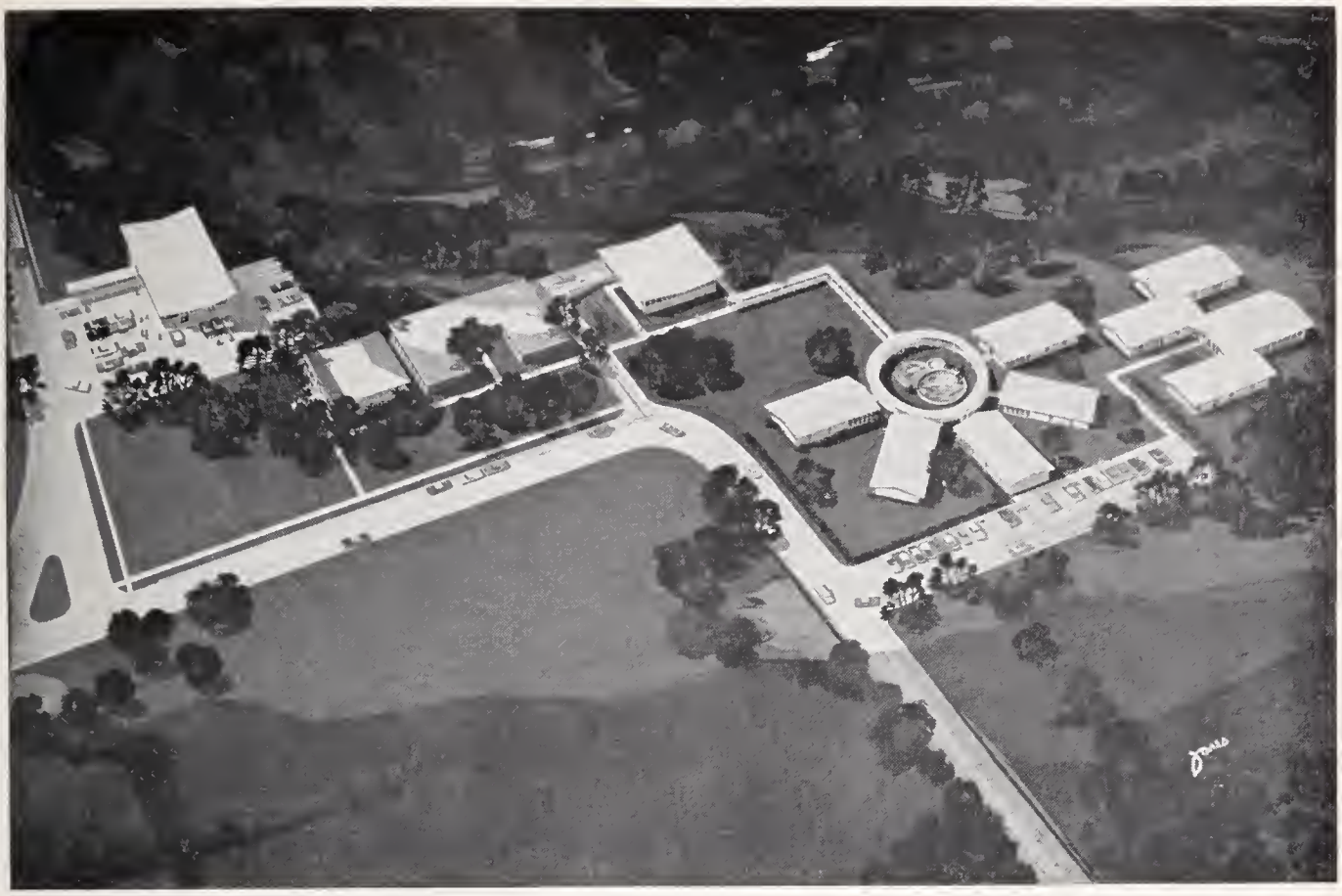
Blackford Memorial Lectures, Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 17-19, 1967. Dr. W. Tom Arnold, 6448 Fannin, Houston 77025, Sec.

Southwest Regional Cancer Conference, Arlington, Oct. 1, 1966. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.



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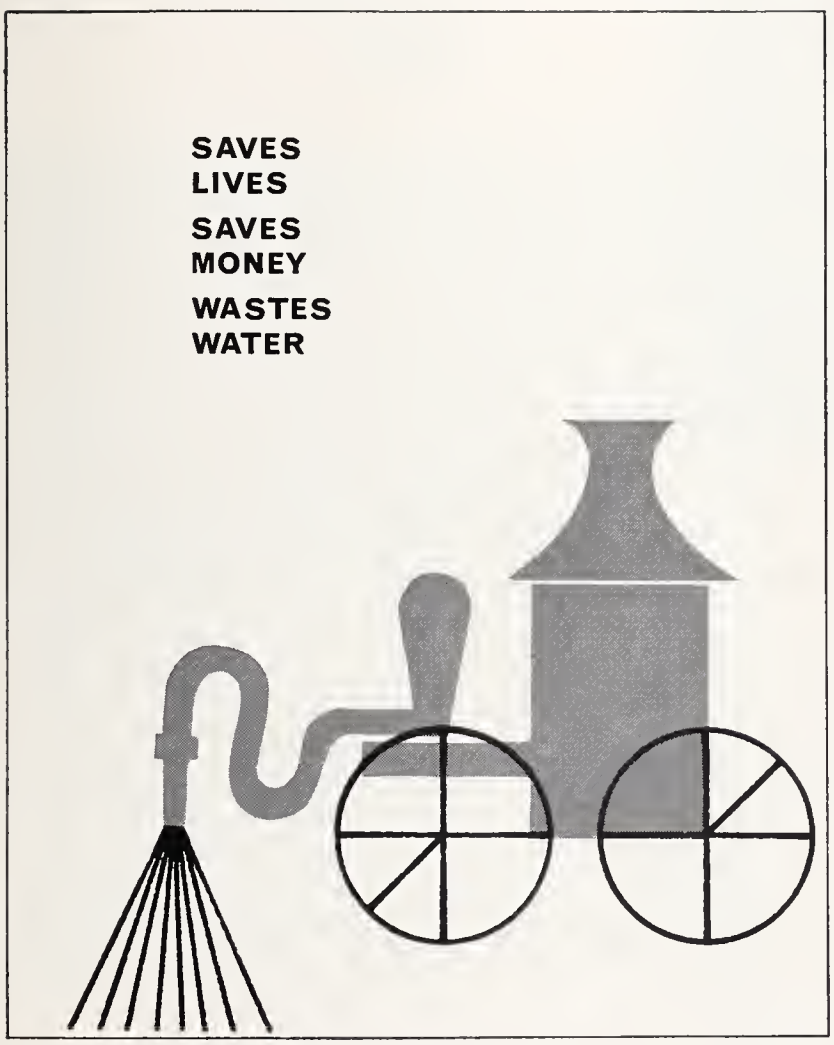
METAHYDRIN (trichlormethiazide) is prescribed by physicians because it not only approximates the diuretic efficacy of parenteral meralluride injection . . . but, *it is the least expensive of all "brand-name" thiazides.* Therefore, when you prescribe METAHYDRIN (trichlormethiazide) your patients receive the thiazide diuretic that removes a little more salt and water than earlier thiazides, with relatively less loss of potassium . . . and, it's therapy they can more easily afford . . . *only pennies a day.*



METAHYDRIN®
(trichlormethiazide)
oral diuretic

Dosage: One 2 or 4 mg. tablet once or twice daily.

Precautions: As with all effective diuretics, vigorous therapy may produce electrolyte depletion. Patients with severely reduced renal function should be observed carefully since thiazides may be contraindicated. Care should be taken with patients predisposed to diabetes or gout. Patients with a tendency to potassium deficiency, as in hepatic cirrhosis or diarrheal syndromes, or those under therapy with digitalis, ACTH, or certain adrenal steroids, also should be watched carefully.

Side Effects: Nausea, flushing, constipation, skin rash, muscle cramps and gastric discomfort have occasionally been noted; rarely thrombocytopenia and bone marrow depression, photosensitivity, cholestatic jaundice, pancreatitis, perimacular edema, gout and diabetes have been caused by the administration of thiazides.

Contraindications: Complete renal shutdown; rising azotemia or development of hyperkalemia or acidosis in severe renal disease; demonstrated hypersensitivity.

How Supplied: Bottles of 100 and 1000 tablets.

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LAKESIDE | PRODUCTS FOR PATIENTS YOU SEE EVERY DAY

When depressed patients say:

"I can't sleep at night"

"I'm tired all day long"

NORPRAMIN®

(desipramine hydrochloride)

non-sedating • rapid-acting
ANTIDEPRESSANT

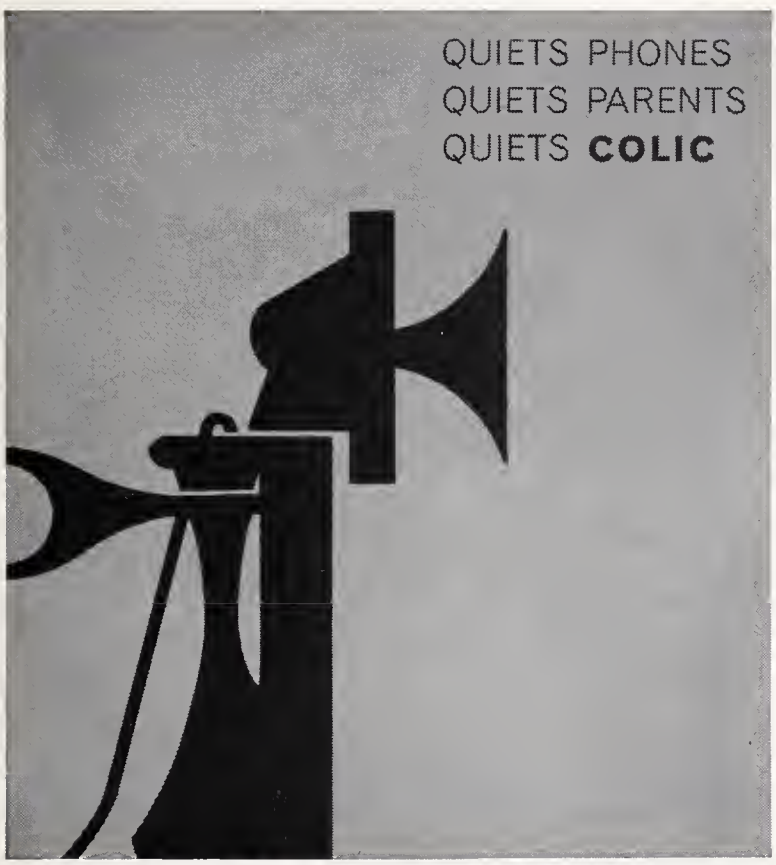
helps restore normal patterns of sleep and activity

Norpramin (desipramine hydrochloride) often reverses the signs and symptoms of depression including sleep disturbances feeling of sadness, guilt, anxiety, worthlessness and bodily complaints without physical basis. In 2-5 days most patients become more hopeful, active and less weighed down by their problems.

Norpramin (desipramine hydrochloride) has only slight sedative qualities, nevertheless sleep disturbances and restlessness are relieved as depression is lifted. If anxiety or tension develop or persist a tranquilizer may be added or dosage reduced. Side effects are usually mild, occurring in about 1 of 4 patients.

Indications: In moderate to severe depression—neurotic or psychotic. **Dosage:** Optimal results are obtained at a dosage of two 25 mg. tablets t.i.d. (150 mg./day). **Contraindications and Precautions:** Glaucoma, urethral or ureteral spasm, recent myocardial infarction, severe coronary heart disease and epilepsy. Should not be given within two weeks of an MAO inhibitor. Safety in human pregnancy has not been established. **Adverse Effects:** Usually mild, may include: dry mouth, constipation, dizziness, palpitation, delayed urination, "bad taste", sensory illusion, tinnitus, agitation and stimulation, sweating, drowsiness, headache, orthostatic hypotension, flushing, nausea, cramps, weakness, blurred vision and mydriasis, rash, allergy, transient eosinophilia, granulopenia, altered liver function, ataxia and extrapyramidal signs. **Supplied:** Norpramin (desipramine hydrochloride) tablets of 25 mg., in bottles of 50, 500 and 1000.

PRODUCTS FOR PATIENTS YOU SEE EVERY DAY LAKESIDE LABORATORIES, INC., Milwaukee, Wisconsin 53201



In colicky infants Pediatric Piptal with Phenobarbital slows down spasm, diminishes pain and crying and improves feeding patterns. It permits sleep and rest for patient and family. The less than hypnotic amount of phenobarbital in the recommended dose affords a mild, calming action and enhances the antispasmodic action of Piptal (pipenzolate bromide). The latter drug, as reported in the medical literature, has a favorable ratio of effectiveness to side-effects which is unusual in anticholinergics and thus is particularly appropriate to pediatric use.

PEDIATRIC PIPTAL® WITH PHENOBARBITAL

each cc. contains 6 mg. phenobarbital (warning: may be habit forming); 4 mg. Piptal® (pipenzolate bromide), and 20% alcohol.

Pleasant-tasting Pediatric Piptal with Phenobarbital is miscible in milk, formulas and fruit juices, and may also be given by dropper directly on the infant's tongue. Dosage is 0.5 cc. 15 minutes before feeding; in severe cases, 1.0 cc. four times daily. High doses may occasionally cause constipation with tenesmus and, rarely, flushing without fever. It is contraindicated in bowel obstruction or sensitivity to phenobarbital or anticholinergics. Available in 30 cc. dropper bottles, droppers calibrated to deliver 0.5 cc.

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Sustained circulatory, respirator
and cerebral stimulation for th

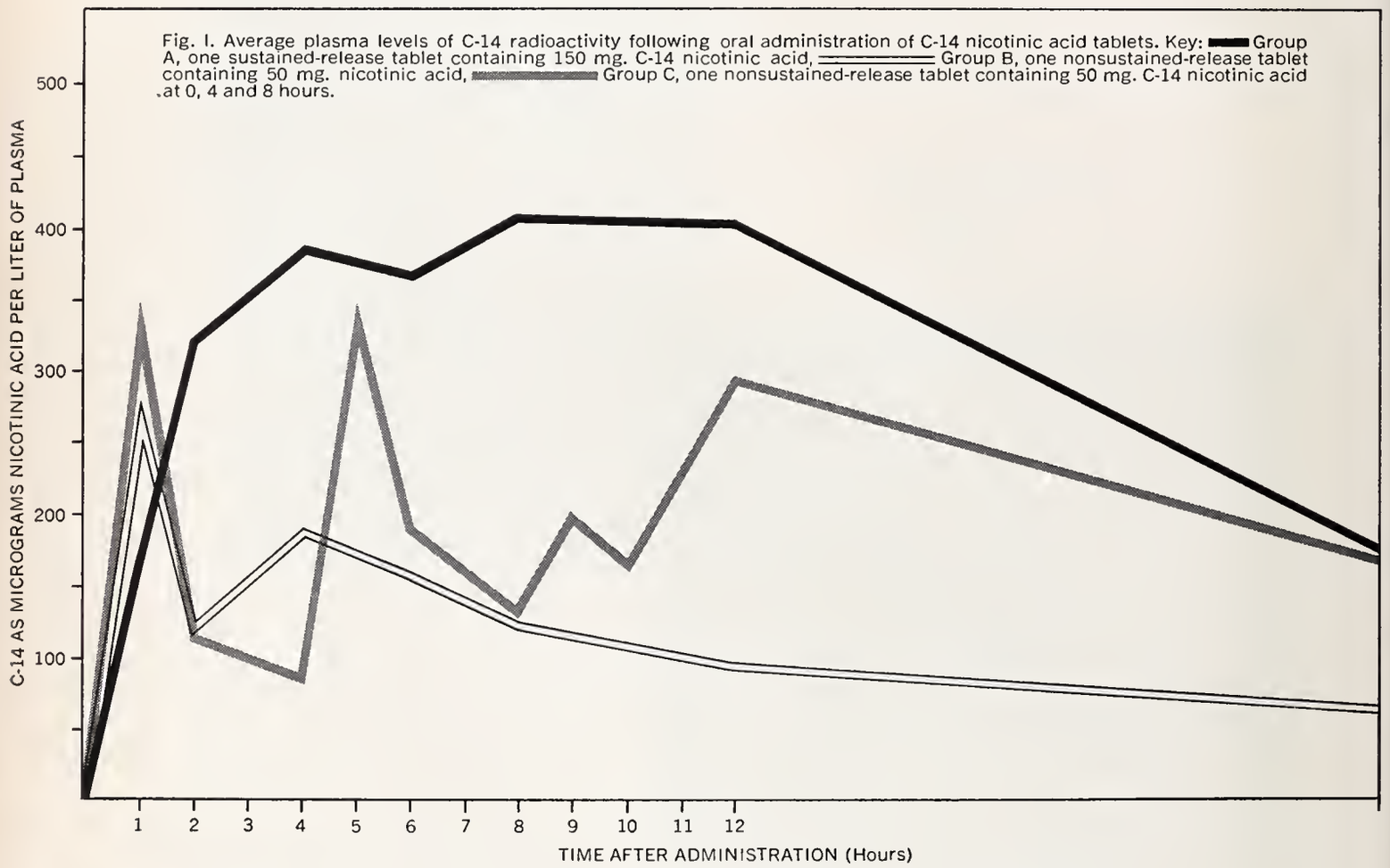
Fig. I. Average plasma levels of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MICROGRAMS NICOTINIC ACID PER LITER OF PLASMA
500
400
300
200
100
1
2
3
4
5
6
7
8
9
10
11
12
TIME AFTER ADMINISTRATION (Hours)

(fewer absent doses by absent-minded patients)

Human volunteer subjects were administered Geroniazol TT tablets with the nicotinic acid component made radioactive with C-14. Plasma and urine samples were analyzed. (See Figures I and II) The radioactive tracer study substantiated the previous clinical evidence that the release of nicotinic acid from the Geroniazol TT tablet produced a gradual rise in plasma levels to a plateau for a total of 12 hours and more.

Such proven sustained activity makes the management of geriatric patients much easier by minimizing the possibility of neglected doses through absent-mindedness or senile confusion. Therapy *can* be continuous on a daily dose of only one Geroniazol TT tablet every 12 hours.

The gradual release of nicotinic acid in Geroniazol TT will provide the well-known peripheral vasodilatation needed in patients with deficient circulation and with a minimum amount (if any) of "flushing." Also, cerebrovascular circulation is complemented by pentylenetetrazol, long-established as a cerebral and respiratory stimulant.

Geroniazol TT improves the typical, unfortunate, signs of senile confusion. Patients become more alert,

ged and debilitated

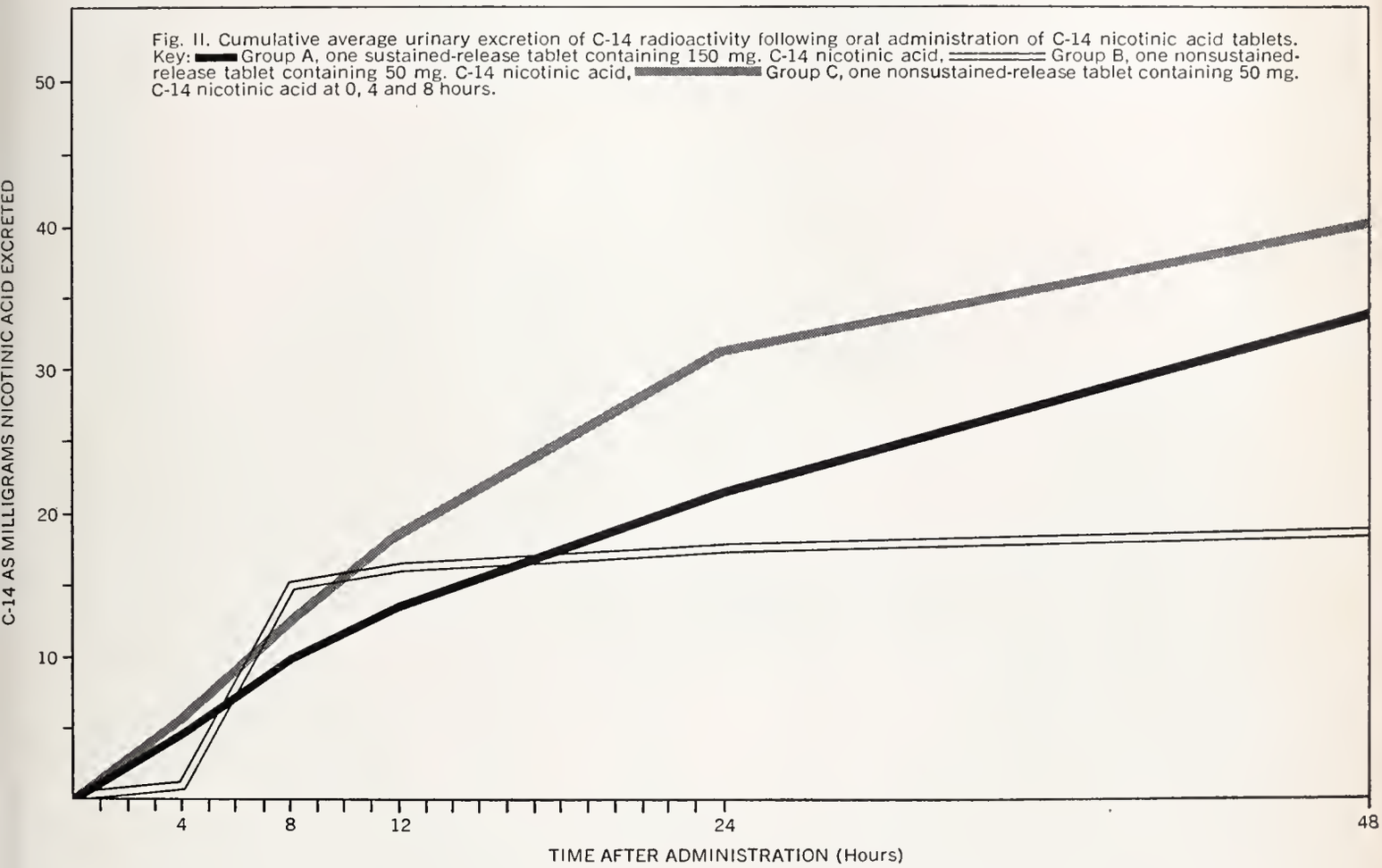
Fig. II. Cumulative average urinary excretion of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MILLIGRAMS NICOTINIC ACID EXCRETED
50
40
30
20
10
4
8
12
24
48
TIME AFTER ADMINISTRATION (Hours)

less confused and moody. Personal care, memory, emotional stability, social attention improve. Fatigue, apathy and irritability are reduced.

A prescription for 100 tablets of Geroniazol TT will permit your patients to enjoy the benefits of time-prolonged nicotinic acid/pentylenetetrazol therapy, at an economical price. Dosage is only one tablet every 12 hours.

Contraindications: There are no known contraindications.

Precautions: Exercise caution when treating patients with a low convulsive threshold.

Side Effects: Side effects are rarely encountered, however due to the vasodilatation effect of nicotinic acid, transitory mild nausea, flushing, tingling and pruritus are possible.

Dosage: One tablet every 12 hours.

Supplied: Prescribe bottles of 100 tablets, to take advantage of recent price reduction.

References: 1. Report by Nuclear Science & Engineering Corp., Pittsburgh, Pa., in files of Philips Roxane Laboratories. 2. Connolly, R.: W. Virginia Med. J. *56*:263 (Aug.) 1960. 3. Curran, T. R., and Phelps, D. K.: Am. Pract. & Digest Treat. *11*:617 (July) 1960.

Geroniazol® TT

nicotinic acid 150 mg., pentylenetetrazol 300 mg.
Tempotrol® Time Controlled Tablet

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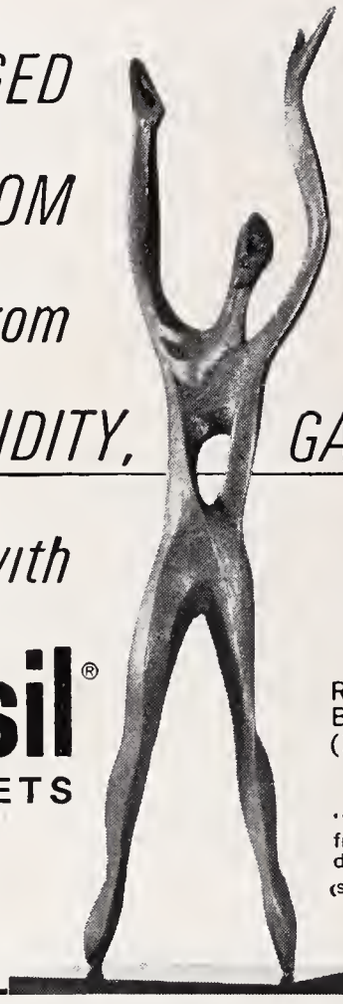

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Box of 144 Tablets
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- exceptional economy—High neutralizing capacity, cost of only 1¢ or less per tablet.
- convenient—medication anywhere, anytime from handy 12 tablet roll pleases patients.

Active Ingredients: Calcium Carbonate Precipitated 0.489 Gm., Magnesium Carbonate 0.011 Gm., Magnesium Trisilicate 0.006 Gm.

1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
*U.S. Pat. No. 3,062,714

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In sinusitis, colds, or U.R.I., Dimetapp lets congested patients breathe easy again. Each Extentab brings welcome relief all day or all night, usually without drowsiness or overstimulation. Its key to success? The Dimetapp formula—Dimetane (brompheniramine maleate), a potent antihistamine reported in one study to have elicited side effects as few as the placebo,* teamed with decongestants phenylephrine and phenylpropanolamine—in a dependable 10- to 12-hour form.

*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

Contraindications: Patients hypersensitive to antihistamines. Not recommended for use during pregnancy.

Precautions: Until the patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. Administer with care to patients with cardiac or peripheral vascular diseases or hypertension.

Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability, or excitement may be encountered.

Dosage: 1 Extentab morning and evening, or as needed.

Supplied: Bottles of 100 and 500.

Also available: Dimetapp® Elixir for conventional *t.i.d.* or *q.i.d.* dosage. See package insert for further details.

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Phenacetin (3 gr.) .....194.0 mg.
Hyoscyamine sulfate .....0.031 mg.
Codeine phosphate.....¼ gr. (No. 2),
½ gr. (No. 3), 1 gr. (No. 4)
(Warning: may be habit forming)

Contraindications: Hypersensitivity to any ingredient.

Precautions: As with all phenacetin-containing products, avoid excessive or prolonged use.

Side Effects: Side effects are uncommon—nausea, constipation, and drowsiness have been reported.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumcthiazide a rather dramatic reduction in dose range is accomplishcd. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effectivc antihypertensive agent.

Contraindications: Severe renal impairment; previous hypersensitivity.

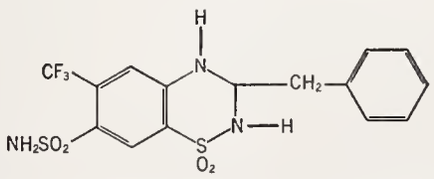
Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: **1.** Southworth, H.: Proc. Soc. Exper. Biol. & Med. *36*:58, 1937. **2.** Mann, T. and Keilin, D.: Nature *146*:164, 1940. **3.** Pitts, R. F., and Alexander, R. S.: Am. J. Physiol. *144*:239, 1945. **4.** Schwartz, W. B.: New England J. Med. *240*:173, 1949. **5.** Friedberg, C. K., in Moyer, J. H., and Fuchs, M.: Edema Mechanisms and Management, Philadelphia, W. B. Saunders Co., 1960, p. 259. **6.** Cumming, J. R.; Tabachnick, E., and Seelig, M., in Moyer, J. H., and Fuchs, M.: op. cit., p. 254. **7.** Werko, L., in Moyer, J. H., and Fuchs, M.: op. cit., p. 188. **8.** Beyer, K. H., Jr., in Moyer, J. H., and Fuchs, M.: op. cit., p. 274. **9.** Maren, T. H., and Wiley, C. E.: J. Pharmacol. & Exper. Therap. *143*:230, 1964. **10.** Earley, L. E., and Orloff, J.: Ann. Rev. Med. *15*:149, 1964. **11.** Fuchs, M., and Mallin, S. R., in Moyer, J. H., and Fuchs, M.: op. cit., p. 276. **12.** Ford, R. V., in Moyer, J. H., and Fuchs, M.: op. cit., p. 290. **13.** cited in Fuchs, M., and Mallin, S. R. (ref. 11): op. cit., p. 283.



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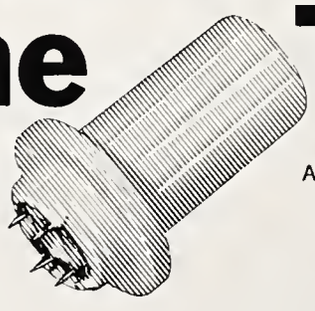

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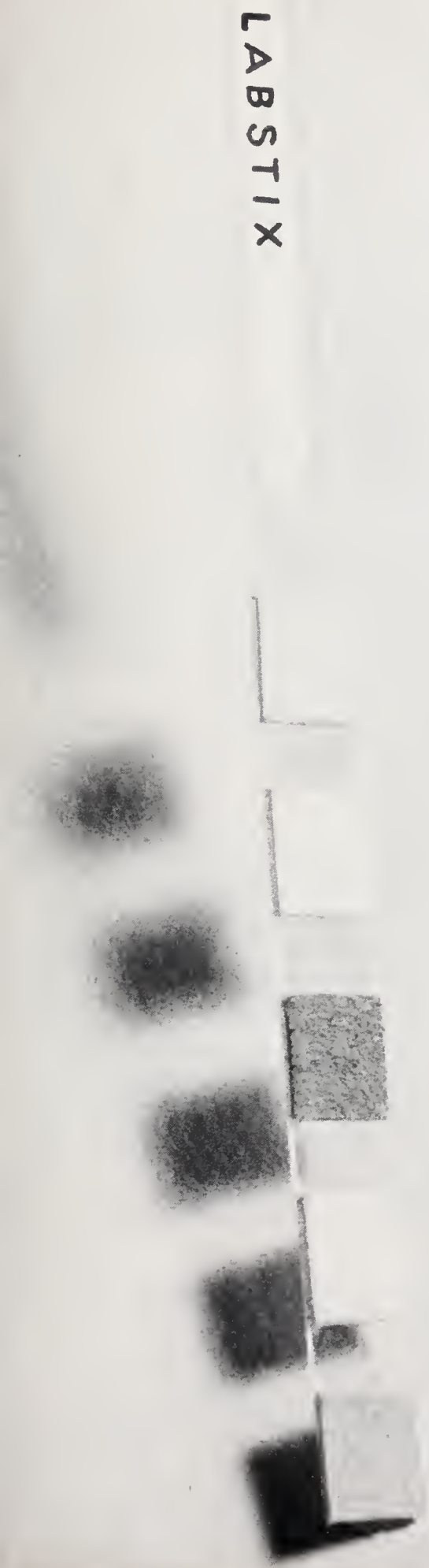

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WANTED: General Practitioner to assume well-established practice in Moody, Texas. Available facilities include fully equipped office and three examining rooms. Located in Central Texas, Moody has a trade area of 3,500. Interested physicians please contact Garland Baker, Frank Denny, Ray Donaldson or Leroy Wall, Moody, Texas.

WANTED: GENERAL PRACTITIONER to associate with established two-man group in south central Texas. Clinic, hospital and nursing home combination. No investment or other expenditures required. Clinic and hospital fully equipped. Should earn $25,000 to $30,000 year net. Guarantee or salary could be negotiated. Please reply to Box 881, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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NEEDED: ORTHOPEDIST to join 14-man specialty group. New million-dollar clinic, city 35,000 West Texas, clinic adjoins accredited hospital. No other orthopod in city, drawing area 100-mile radius. Requires no investment. Generous salary and many fringe benefits. Call collect if interested. Will reimburse applicant for trip for interview. Malone and Hogan Clinic, Big Spring, Texas.

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WANTED: INTERNIST, GENERAL SURGEON, GENERAL PRACTITIONER. Must be board eligible or certified. To be associated with well established expanding group. Salary open, no investment, partnership after second year. University Medical-Surgical Clinic, 511 College Avenue, Lubbock, Texas 79401.

WANTED: ONE INTERNIST, ONE GENERAL PRACTITIONER AND ONE PEDIATRICIAN to join established group in Dallas, either Board qualified, or with at least two years residence, not over 35 years of age. Must have Texas license or be able to get Texas license by reciprocity. Salary open, depending on training. Partnership in two years. Group has own fully accredited hospital. Anyone interested, apply to Box 939, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701. Full details concerning group and the opportunities it offers will be furnished on inquiring.

WANTED BOARD CERTIFIED or eligible anesthesiologist to join Anesthesia Associates in San Angelo, Texas. This is private practice with fee for service. Direct all correspondence to Dr. O. G. Zacharias, 224 East Harris Avenue, Suite A, San Angelo, Texas 76901.

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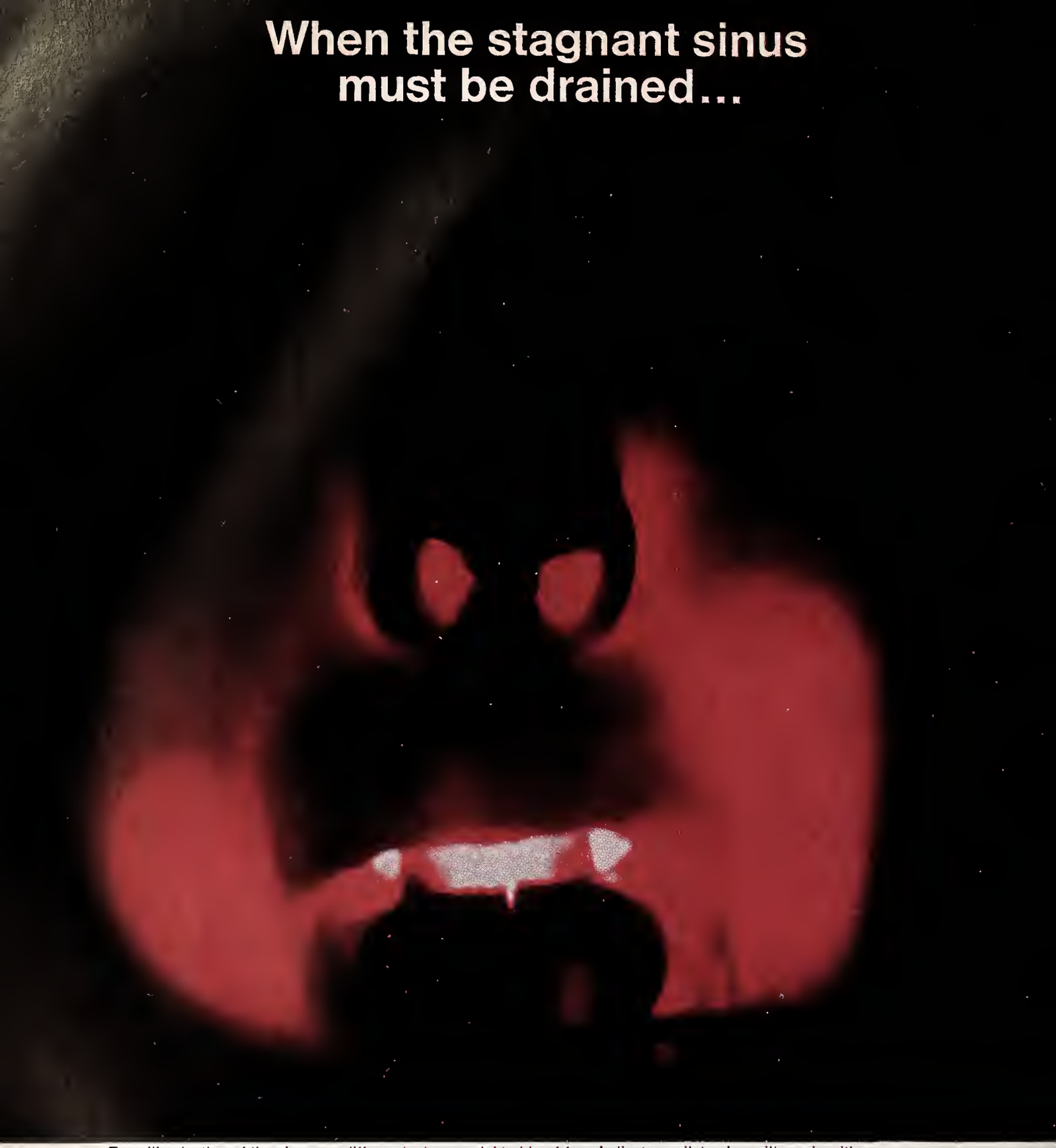

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Breast-feeding and the "modern mother"

Despite a mild resurgence of interest in the importance of breast-feeding a few years ago, many women today do not choose to nurse their young. This is for a variety of reasons—social, economic, cultural and sometimes medical. In such cases the physician's task is to find the most suitable means of preventing lactation and easing the pain of breast engorgement.

The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

However, factors other than effectiveness must also be considered. The agent selected should not interfere in any way with parturition, subsequent uterine involution and the restoration of normal ovarian cyclic function. Furthermore, it should not cause rebound breast engorgement or other manifestations of hormonal imbalance.

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†The need for these substances in human nutrition has not been established.

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
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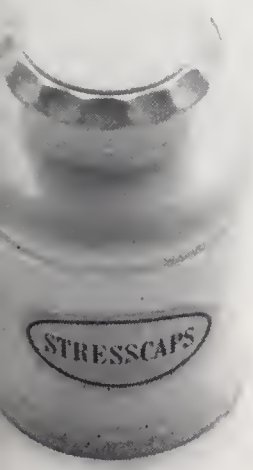

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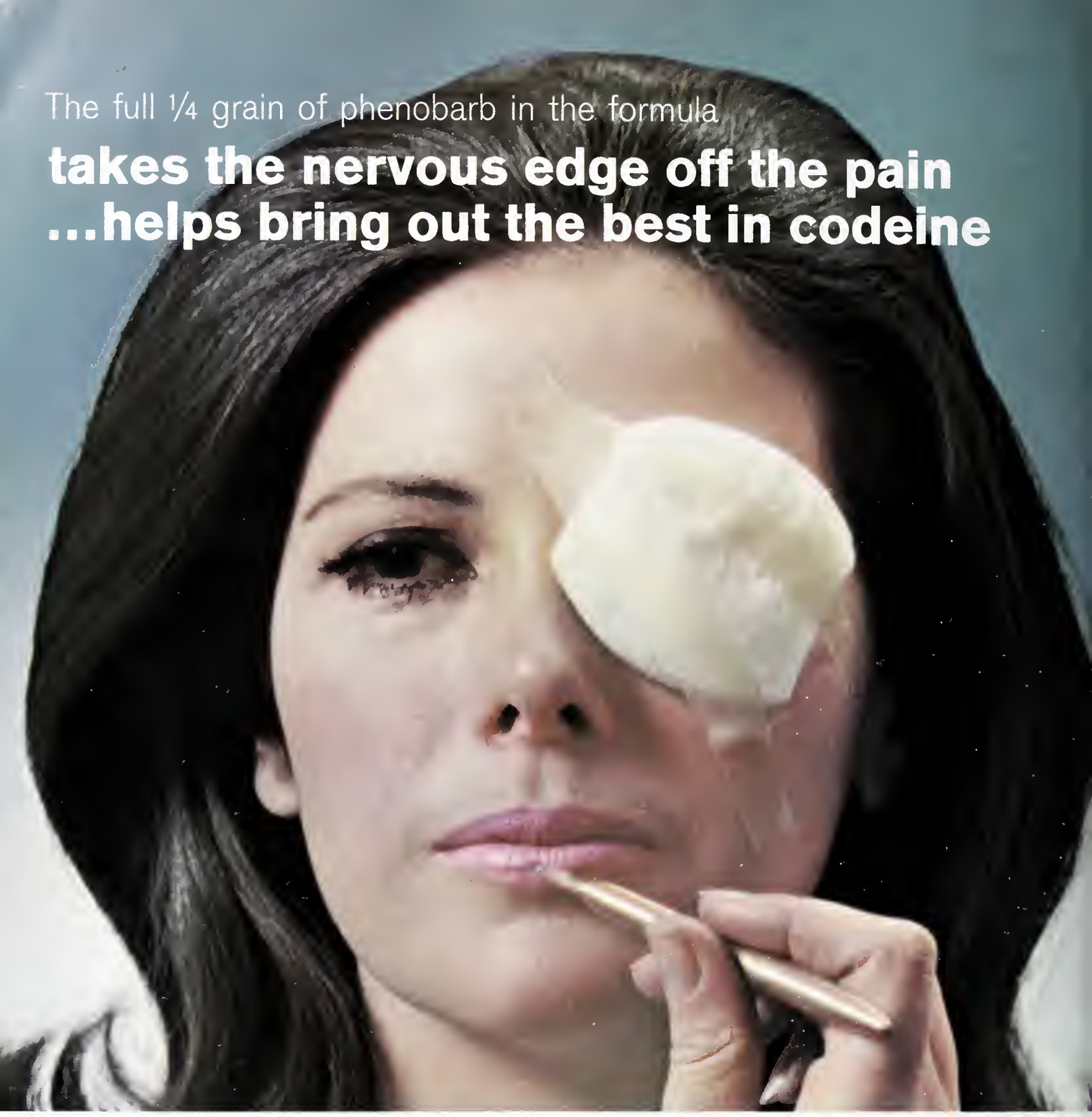

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1. Forty cases reported.
2. Excellent to good results, 75% with Android, 20% with Placebo.
3. Cites synergism between androgen and thyroid.
4. No side effects in patients treated.
5. Alleviation of fatigue noted.
6. Case histories on 4 patients.
7. Although psychotherapy still needed, role of chemotherapy cannot be disputed.

CONTRAINDICATIONS — Methyl testosterone is not to be used in malignancy of reproductive organs in male, coronary heart disease, hyperthyroidism. Thyroid is not to be used in heart disease, hypertension unless the metabolic rate is low.

CAUTION: Federal law prohibits dispensing without prescription.

*"Sexual impotence treatment with methyl testosterone - thyroid (ANDROID) a double blind study" - Montesano, Evangelista: Clinical Medicine, April 1966.

REFER TO PDR

ANDROID
Each yellow tablet contains:
Methyl Testosterone ..... 2.5 mg.
Thyroid Ext. (1/6 gr.) ..... 10 mg.
Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 tablet 3 times daily.
Available:
Bottles of 100, 500, 1,000.

ANDROID-HP
Each red tablet contains:
Methyl Testosterone ..... 5.0 mg.
Thyroid Ext. (1/2 gr.) ..... 30 mg.
Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 tablet 3 times daily.
Available:
Bottles of 100, 500, 1,000.

ANDROID-X NEW
Each orange tablet contains:
Methyl Testosterone ..... 12.5 mg.
Thyroid Ext. (1 gr.) ..... 64 mg.
Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 or 2 tablets daily.
Available:
Bottles of 60, 500.

ANDROID-PLUS
Each white tablet contains:
Methyl Testosterone ..... 2.5 mg.
Thyroid Ext. (¼ gr.) ..... 15 mg.
Thiamine HCL ..... 25 mg.
Ascorbic Acid (Vit. C) ..... 250 mg.
Glutamic Acid ..... 100 mg.
Pyridoxine HCL ..... 5 mg.
Niacinamide ..... 75 mg.
Calcium Pantothenate ..... 10 mg.
Vitamin B-12 ..... 2.5 mcg.
Riboflavin ..... 5 mg.
Dose: 1 tablet twice daily.
Available:
Bottles of 60, 500.

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Vasomist

(ANTIHISTAMINIC)

VASOCONSTRICTOR, NASAL DECONGESTANT AND LOCAL ANTIHISTAMINIC

Active Ingredients:
Phenylephrine Hcl 0.25%
Phenylpropanolamine Hcl 0.50%
Chlorobutanol 0.15%
Tripelennamine Hcl 0.50%
In Isotonic Solution

USE: Temporary relief of nasal congestion due to colds and hay fever.

DIRECTIONS: Adults and Children: Instill two or three drops into each nostril, using no more than needed to provide relief. Repeat every three to four hours as needed but not oftener.

AVAILABILITY

In plastic squeeze bottles containing ¾ fluid oz. and in dropper bottles containing 1 fluid oz.

HUMCO LABORATORY
TEXARKANA, ARK.-TEX.

Vasomist

(PLAIN)

VASOCONSTRICTOR AND NASAL DECONGESTANT

Active Ingredients:
Phenylephrine Hcl 0.25%
Phenylpropanolamine Hcl 0.50%
Chlorobutanol 0.15%
In Isotonic Solution

P® DISPOSABLE UNIT
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A RELIABLE, PROVEN PRODUCT . . .

The BSP test, introduced in 1925, remains one of the most sensitive laboratory procedures available for determining hepatic function.

A STERILE, CONVENIENT PUT-UP . . .

Each BSP Disposable Unit contains: a sterile, glass, disposable syringe with the 5 mg./kg. BSP dosage schedule imprinted on the barrel, a sterile needle, alcohol swab and a 7.5 ml. or 10 ml. size ampule of Bromsulphalein.

The BSP unit is designed for maximum patient protection. The precalibrated syringe makes weight calculations unnecessary and provides for proper dosage regardless of patient-weight.

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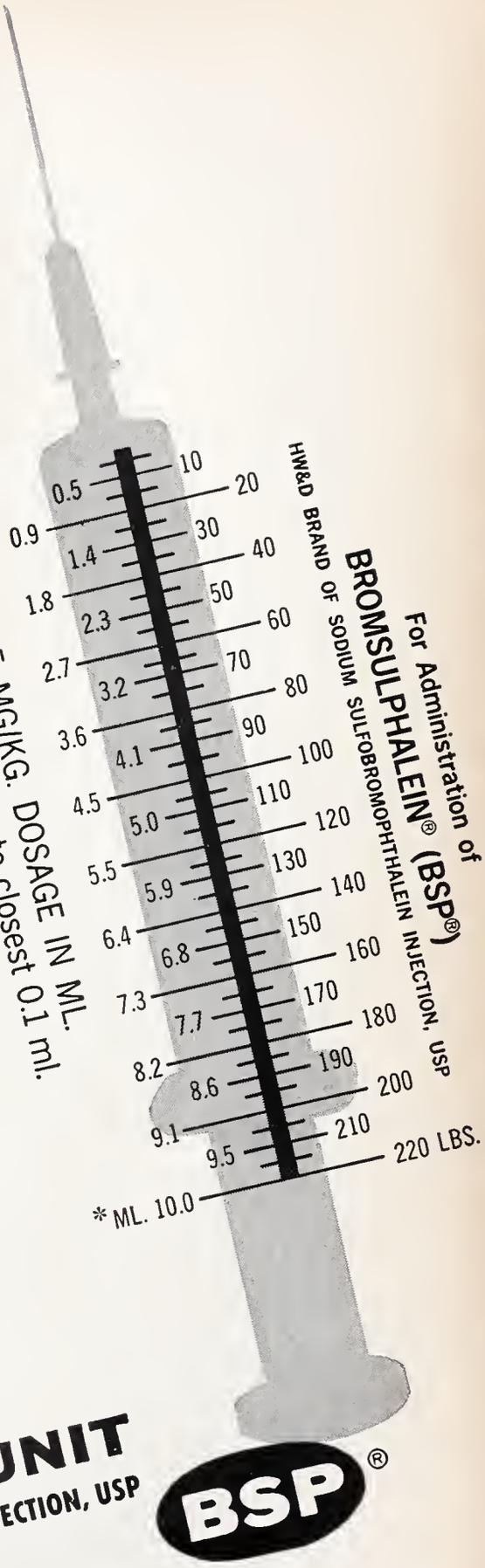
Prepared for economic unit dispensing, this completely disposable put-up affords a savings in time and labor—the most costly commodities.


For Administration of
BROMSULPHALEIN® (BSP®)
HW&D BRAND OF SODIUM SULFOBROMOPHTHALEIN INJECTION, USP
5 MG/KG. DOSAGE IN ML.
* dosages given to closest 0.1 ml.
0.5 0.9 1.4 1.8 2.3 2.7 3.2 3.6 4.1 4.5 5.0 5.5 5.9 6.4 6.8 7.3 7.7 8.2 8.6 9.1 9.5 * ML. 10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210 220 LBS.

BSP® DISPOSABLE UNIT

HW&D BRAND OF SODIUM SULFOBROMOPHTHALEIN INJECTION, USP

(50 mg. per ml.)

HYNSON, WESTCOTT & DUNNING, INC.

BALTIMORE, MARYLAND 21201

(BSP02)

"A DRAMATIC INCREASE IN SALMONELLOSIS HAS BEEN OBSERV

ALMONELLA
CARRIERS
PRESENT A
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FIFTH
COLUMN."[2]

Furoxone exerts "outstanding activity against Salmonella sp."[3] and "has enhanced the physicians' armamentarium in that it now affords a relatively effective, safe bactericidal agent, which may be used to treat the active disease as well as the carrier state".[2]

SALMONELLA SPECIFIC

FUROXONE®

FURAZOLIDONE LIQUID/TABLETS

Often succeeds where others fail in a wide variety of bacterial diarrheas. Specifically within the broad bactericidal range of Furoxone:

Salmonella paratyphi	Salmonella montevideo
Salmonella schottmülleri	Salmonella newport
Salmonella typhimurium	Salmonella anatum
Salmonella choleraesuis	Salmonella derby
Salmonella enteritidis	

Proper antibacterial therapy for individual cases should help in reducing the total number of cases in an epidemic situation.

Side effects are infrequent. A few hypersensitivity reactions to Furoxone have been reported including a fall in blood pressure, urticaria, fever, arthralgia and a vesicular or morbilliform rash. These reactions subsided promptly following withdrawal of the drug. Primaquine-sensitive patients may develop a mild reversible hemolytic anemia. Nausea, emesis, headache or malaise may occur occasionally. To obviate alcohol-disulfiram type reactions, advise against use of alcohol-containing drugs during therapy and four days thereafter.

Do not give to infants under one month of age.

Composition: Furoxone Liquid contains, per 15 cc. tablespoonful, furazolidone 50 mg., pectin 225 mg., and kaolin 3.0 Gm. Furoxone Tablets each contain 100 mg. of furazolidone.

References: 1. Editorial: J.A.M.A. *189*:691 (Aug. 31) 1964. 2. Foertsch, J. H.: J. Oklahoma Med. Assn. *57*:449 (Oct.) 1964. 3. Paul, H. E., and Paul, M. F.: The Nitrofurans—Chemotherapeutic Properties, in Schnitzer, R. J., and Hawking, F. (Eds.) Experimental Chemotherapy, Vol. 2, New York, Academic Press, 1964.

Originators and Developers of The Nitrofurans
EATON LABORATORIES
Division of The Norwich Pharmacal Company
NORWICH, NEW YORK

Frankly, most antihypertensives are pretty good if you give an adequate dose. I'm looking for one with a simple regimen so that mix-ups in doses and therefore the chance of side effects are minimized.

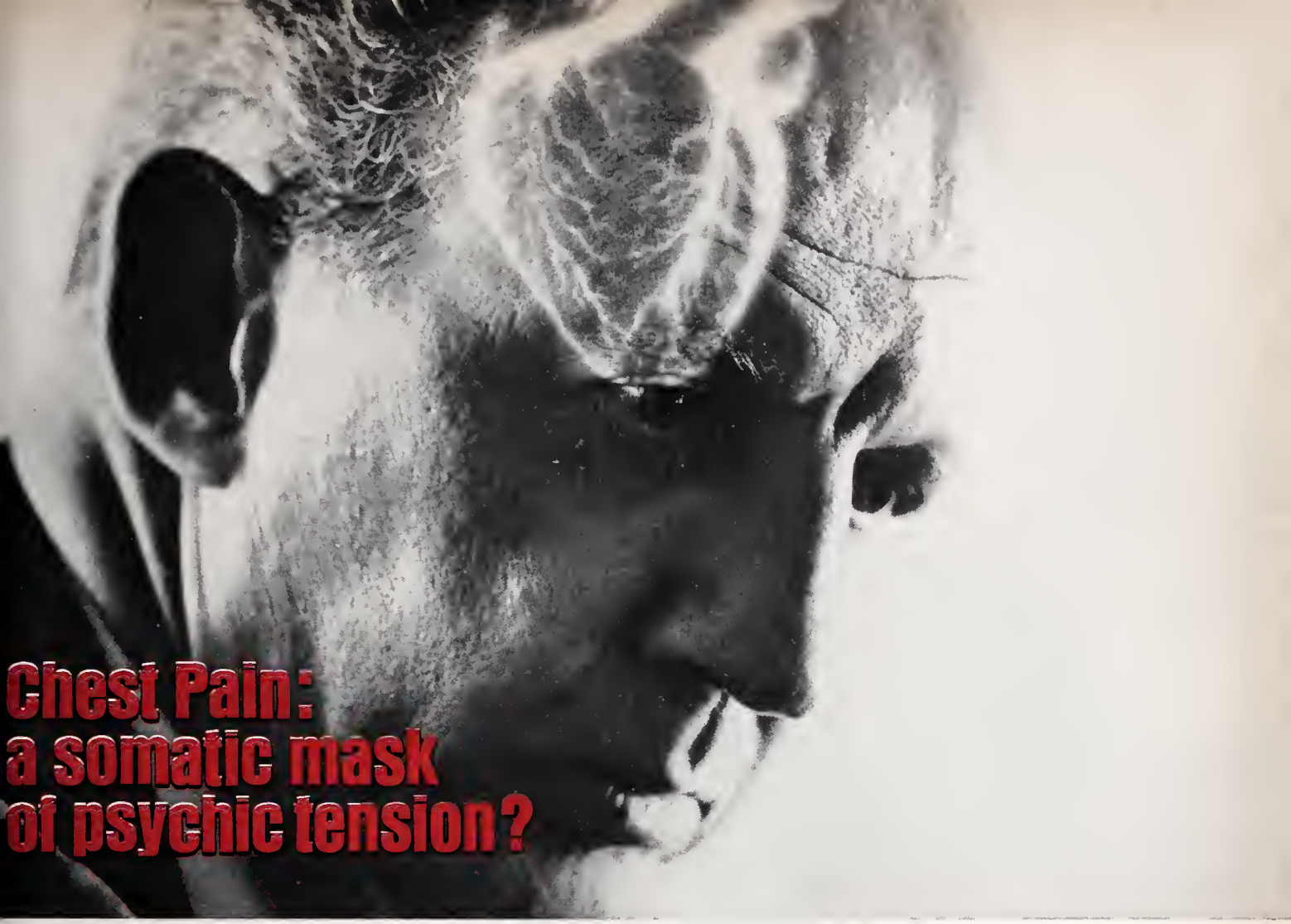
Regroton®

chlorthalidone 50 mg. reserpine 0.25 mg.

1 tablet daily brings pressure down

Advantage: Both components of Regroton are long-acting.
Average dosage: One tablet daily with breakfast.
Contraindications: History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases.
Warning: Discontinue 2 weeks before general anesthesia, 1 week before electroshock therapy, and if depression or peptic ulcer occurs. With administration of enteric-coated potassium supplements, the possibility of small bowel lesions should be kept in mind.
Precautions: Reduce dosage of concomitant antihypertensive agents by one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take particular care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use with caution in patients with ulcerative colitis, gallstones, or bronchial asthma.
Side effects: Nausea, vomiting, diarrhea, muscle cramps, headaches and dizziness. Potential side effects include angina pectoris, anxiety, depression, drowsiness, hyperglycemia, hyperuricemia, lassitude, leukopenia, nasal stuffiness, nightmare, purpura, urticaria, and weakness.
For full details, see the complete prescribing information.
Availability: Bottles of 100 and 1000 tablets.

Geigy



"Heart symptoms"—chest pain, tachycardia, arrhythmia—invariably alarm and preoccupy the patient, though they may be completely without organic basis. Such symptoms often are somatic masks of psychic tension, arising from constant encounters with stressful situations.

When the problem is diagnosed as emotionally produced, consider Valium (diazepam) as adjunctive therapy. Valium (diazepam) acts rapidly to calm the patient, to reduce his psychic tension and relieve associated cardiovascular complaints.

NEUROTIC FATIGUE—the chronic tiredness resulting from emotional strain which so often accompanies psychogenic "heart" symptoms—also can be alleviated by this highly useful agent. Valium (diazepam) often achieves results where other psychotherapeutic agents have failed.

Valium (diazepam) is generally well tolerated, and usually does not impair mental acuity or ability to function. If side effects such as ataxia and drowsiness occur, they usually disappear with dosage adjustment.

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

Supplied: Tablets, 2 mg, 5 mg and 10 mg; bottles of 50 for convenience and economy in prescribing.

Roche Laboratories Division of Hoffmann-La Roche Inc.
Nutley, N.J. 07110

for somatic symptoms of psychic tension

2-mg, 5-mg, 10-mg tablets

Valium®
(diazepam)

ROCHE

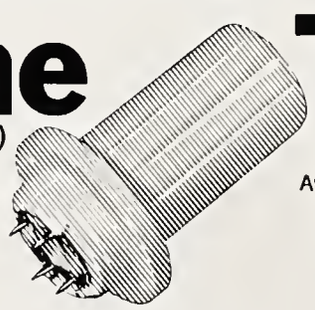
what time is it?

For the past
two years
there's been
one new case
of active tuberculosis
reported for every
four thousand
of U.S. population.

it's time to tine.

Tuberculin, Tine Test

(Rosenthal)



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Available in 5's and 25's.
Order now
from your pharmacist
or your Lederle
representative.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

414-6—4046R

Now, now, Mrs. Forsythe, we've never lost a cold patient yet.

When she's experiencing acute discomfort from cold symptoms, it's small wonder the patient becomes distressed about her condition.

She will breathe easier when you prescribe Novahistine LP. Novahistine LP is a long-acting decongestant that helps restore normal mucus secretion and ciliary activity—physiologic mechanisms which prevent infection of the respiratory tract. A dose of two tablets taken in the morning and repeated in the evening will usually keep air passages clear for 24 hours.

Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution patients who operate machinery or motor vehicles that drowsiness may result.

Each Novahistine LP tablet contains: phenylephrine hydrochloride, 25 mg., and chlorpheniramine maleate, 4 mg.

PITMAN-MOORE Division of The Dow Chemical Company, Indianapolis DOW

NOVAHISTINE® LP

For relief of nasal congestion.

arrest diarrhea

LOMOTIL®

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride . . . . . 2.5 mg.
(Warning: May be habit forming)
atropine sulfate . . . . . 0.025 mg.

Effectiveness: Lomotil possesses a unique degree of effectiveness in both acute and chronic diarrhea.

Convenience: Lomotil is supplied as small, easily carried, easily swallowed tablets and as a pleasant, fruit-flavored liquid.

Versatility: The therapeutic efficiency, safety and convenience of Lomotil may be used to advantage alone or as adjunctive therapy in diarrhea associated with:

- **Ulcerative colitis**
- **Acute infections**
- **Irritable bowel**
- **Regional enteritis**
- **Drug therapy**
- **Food Poisoning**
- **Functional hypermotility**
- **Malabsorption syndrome**
- **Ileostomy**
- **Gastroenteritis and colitis**

Dosage: For correct therapeutic effect—Rx correct therapeutic dosage. The recommended initial daily dosages, given in divided doses, until diarrhea is controlled, are:

Children: Age	Total Daily Lomotil Dosage	Lomotil Liquid Dosage (Each teaspoonful [4 cc.] contains 2 mg. of diphenoxylate HCl)
3-6 months	3 mg.	½ tsp. 3 times daily
6-12 months	4 mg.	½ tsp. 4 times daily
1-2 years	5 mg.	½ tsp. 5 times daily
2-5 years	6 mg.	1 tsp. 3 times daily
5-8 years	8 mg.	1 tsp. 4 times daily
8-12 years	10 mg.	1 tsp. 5 times daily

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) Based on 4 cc. per teaspoonful. Maintenance dosage may be as low as one-fourth the initial daily dose.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be kept out of reach of children since accidental overdosage may cause severe respiratory depression. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

Everyone says she's a barrel of fun

But what does she think?

Many overweight patients can benefit from the appetite control provided by the sustained anorexigenic-tranquilizing action of BAMADEX SEQUELS: anorexigenic action of amphetamine; tranquilizing action of meprobamate; prolonged action through sustained release of active ingredients.

Bamadex® Sequels®

DEXTRO-AMPHETAMINE SULFATE (15 mg.) WITH MEPROBAMATE (300 mg.) SUSTAINED RELEASE CAPSULES

to help establish a new dietary pattern

Contraindications: Dextro-amphetamine sulfate: in hyperexcitability and in agitated prepsychotic states. Previous allergic or idiosyncrotic reactions to meprobamate.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Designed for the comfort, convenience and privacy of the individual, these facilities offer an atmosphere of a private home combined with considerate professional care.

Every effort is made to help the individual live as active a life as he wants. Occupational and physical therapy are available, as are games, crafts, gardening, music and reading. Religious services are held regularly.

Residents must be admitted under the continuous care and direction of a physician. Care by registered nurses is supervised on a 24-hour basis.

For further information, including statement of policies and rates, write

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C. B. FRANCIS 305 East 7th Street Austin, Texas

Blood-glucose screening for all your patients?

...because "Abnormalities of glucose metabolism are among the [most common] encountered in clinical practice...."* Simple, quick, economical blood-glucose screening with DEXTROSTIX® Reagent Strips is practicable in every regular physical examination, emergency situation, and whenever hypo- or hyperglycemia may be of clinical significance—for "The precision and accuracy of DEXTROSTIX ...meet the need for an always available simple screening method...."* All that is required for screening with DEXTROSTIX is 60 seconds and a globular drop of capillary or venous blood. Abnormal readings will be a valuable aid to diagnosis; normals will help you establish an important baseline for future reference.

*Marks, V., and Dawson, A.: Brit. M. J. *1*:293, 1965.

DEXTROSTIX—
provides a clinically useful determination when performed according to directions†

†DEXTROSTIX is not intended to replace the more precise analytical laboratory methods.

Yes—all your patients

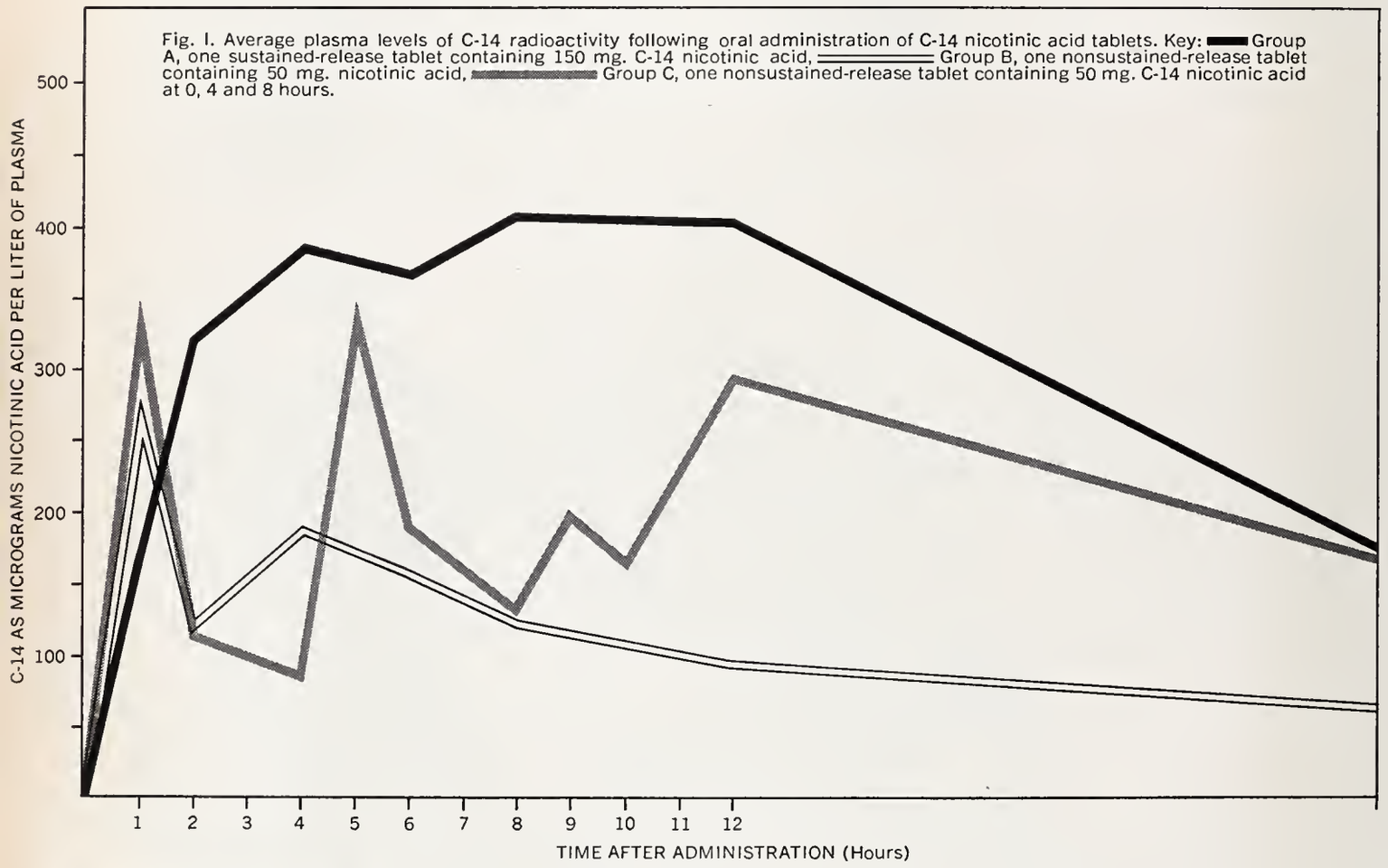
AMES COMPANY, INC.
Elkhart, Indiana

AMES

09165

Sustained circulatory, respiratory and cerebral stimulation for the

Fig. I. Average plasma levels of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: ▬ Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, ═══ Group B, one nonsustained-release tablet containing 50 mg. nicotinic acid, ▬ Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.



(fewer absent doses by absent-minded patients)

Human volunteer subjects were administered Geroniazol TT tablets with the nicotinic acid component made radioactive with C-14. Plasma and urine samples were analyzed. (See Figures I and II) The radioactive tracer study substantiated the previous clinical evidence that the release of nicotinic acid from the Geroniazol TT tablet produced a gradual rise in plasma levels to a plateau for a total of 12 hours and more.

Such proven sustained activity makes the management of geriatric patients much easier by minimizing the possibility of neglected doses through absent-mindedness or senile confusion. Therapy *can* be continuous on a daily dose of only one Geroniazol TT tablet every 12 hours.

The gradual release of nicotinic acid in Geroniazol TT will provide the well-known peripheral vasodilatation needed in patients with deficient circulation and with a minimum amount (if any) of "flushing." Also, cerebrovascular circulation is complemented by pentylenetetrazol, long-established as a cerebral and respiratory stimulant.

Geroniazol TT improves the typical, unfortunate, signs of senile confusion. Patients become more alert,

aged and debilitated

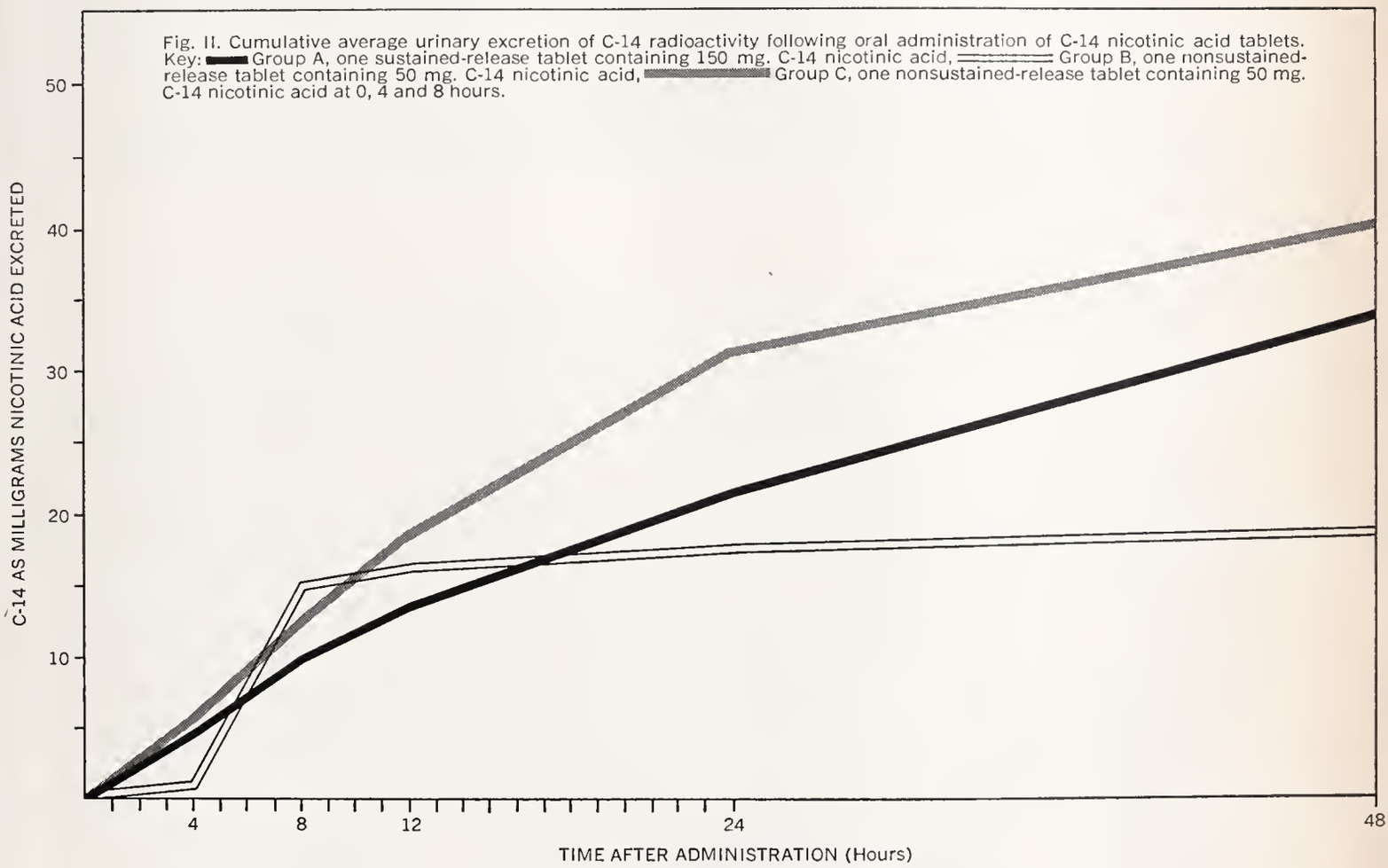


Fig. II. Cumulative average urinary excretion of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MILLIGRAMS NICOTINIC ACID EXCRETED
50
40
30
20
10
4
8
12
24
48
TIME AFTER ADMINISTRATION (Hours)

less confused and moody. Personal care, memory, emotional stability, social attention improve. Fatigue, apathy and irritability are reduced.

A prescription for 100 tablets of Geroniazol TT will permit your patients to enjoy the benefits of time-prolonged nicotinic acid/pentylenetetrazol therapy, at an economical price. Dosage is only one tablet every 12 hours.

Contraindications: There are no known contraindications.

Precautions: Exercise caution when treating patients with a low convulsive threshold.

Side Effects: Side effects are rarely encountered, however due to the vasodilatation effect of nicotinic acid, transitory mild nausea, flushing, tingling and pruritus are possible.

Dosage: One tablet every 12 hours.

Supplied: Prescribe bottles of 100 tablets, to take advantage of recent price reduction.

References: 1. Report by Nuclear Science & Engineering Corp., Pittsburgh, Pa., in files of Philips Roxane Laboratories. 2. Connolly, R.: W. Virginia Med. J. *56*:263 (Aug.) 1960. 3. Curran, T. R., and Phelps, D. K.: Am. Pract. & Digest Treat. *11*:617 (July) 1960.

"First with the Retro-Steroids"
PHILIPS ROXANE LABORATORIES
Division of Philips Roxane, Inc., Columbus, Ohio
A Subsidiary of Philips Electronics and Pharmaceutical Industries Corp.

Geroniazol® TT

nicotinic acid 150 mg., pentylenetetrazol 300 mg.
Tempotrol® Time Controlled Tablet

Norinyl® tablets

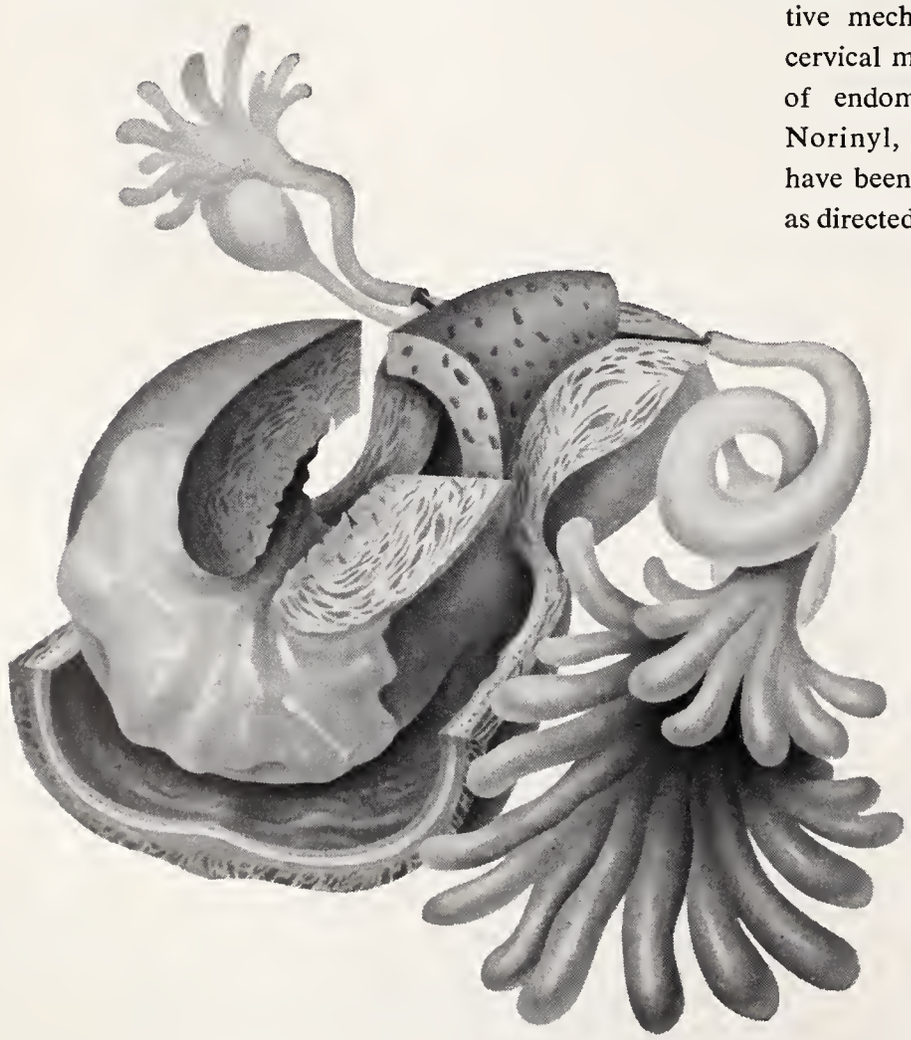
(norethindrone 2 mg. c mestranol 0.1 mg.)

for multiple contraceptive action that has produced a record of unexcelled effectiveness

inhibition of ovulation by means of 2 time-proved hormonal agents

production of a cervical mucus hostile to sperm motility and vitality

creation of an endometrium unreceptive to egg implantation



no unplanned pregnancies

Norinyl provides multiple action for maximum assurance of success. It does not depend on ovulation inhibition alone for contraceptive effectiveness. The mechanism of action of combined hormonal therapy results in ovulation inhibition reinforced by other protective mechanisms, including a hostile cervical mucus[1-13] and an acceleration of endometrial changes.[1-3,7-16] With Norinyl, no unplanned pregnancies have been reported to date when used as directed.

plus important supportive benefits that help her through those critical early months of oral contraception

low incidence of side effects

Low incidence of BTB and spotting, nausea and amenorrhea tends to minimize side effect problems and increases patient cooperation.

no confusion about dosage

An unbreakable "confusionproof" package makes it easy to adhere to prescribed dosage schedule: individually sealed tablets numbered from 1 through 20 *plus* monthly calendar record enables patient to double-check dosage intake by day and corresponding tablet number.

Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

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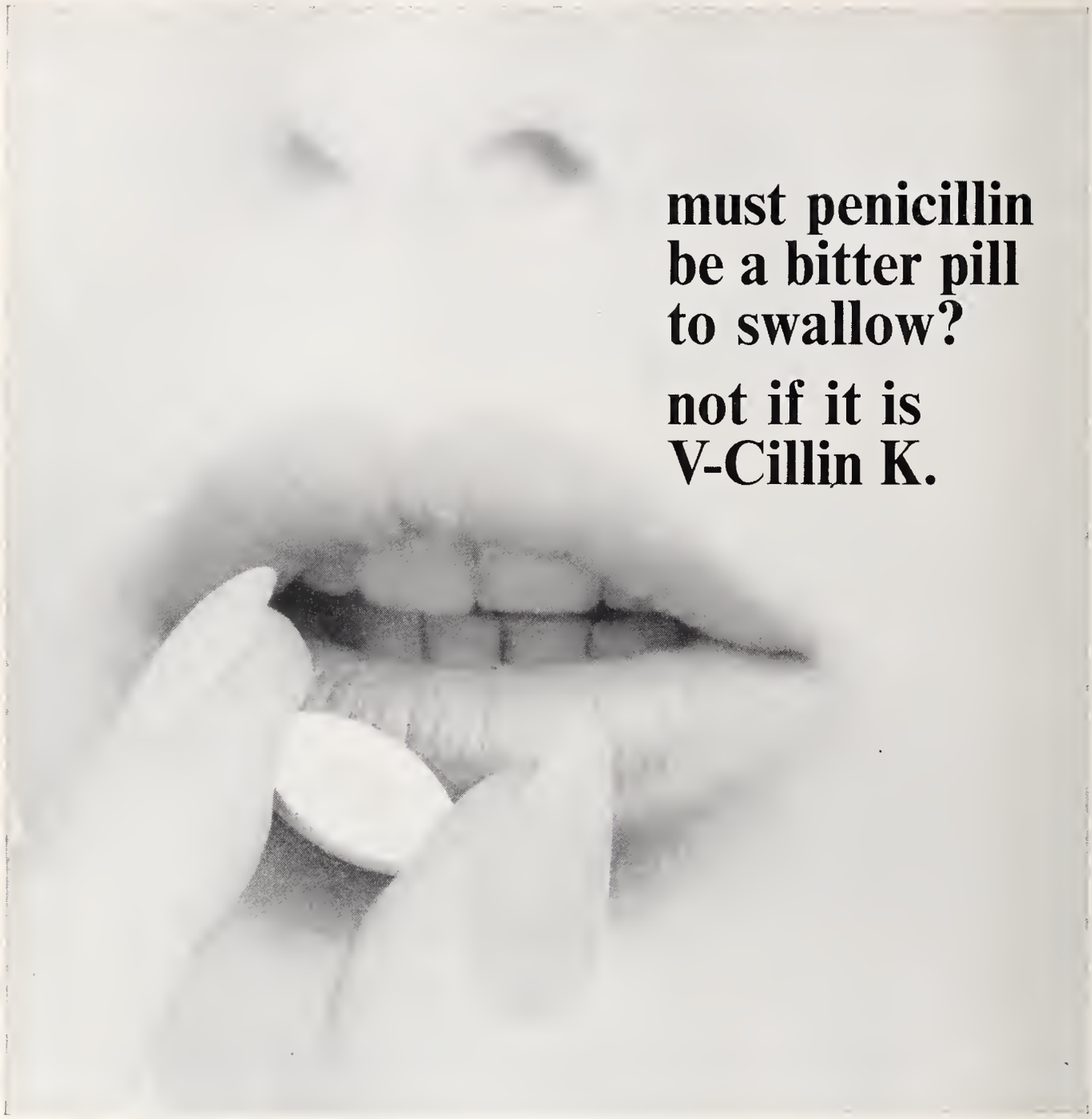

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Carotid Endarterectomy Is Valuable In Prevention, Treatment of Stroke

CHANGING CONCEPTS in the management of strokes during the past few years make it incumbent upon the practicing physician to be familiar not only with the symptoms and signs of paralytic strokes but more especially with the features of attacks of cerebrovascular insufficiency which may herald future strokes. It is now well established that in at least 30 to 40 percent of patients who present with cerebrovascular insufficiency syndromes, the arteriosclerotic occlusive lesions are in the extracranial vasculature and are thus amenable to surgical removal. In most instances the intracranial vessels are surprisingly free of demonstrable disease. Cerebral arteriography, both carotid and vertebral, has become of increasing importance in studying patients with known or suspected cerebral ischemia in order to localize the causative lesions. Carotid endarterectomy has thus assumed a major role in the definitive management of these patients. With proper selection of cases and careful attention to technical details, this operation can be performed with low morbidity and with overall operative mortality approaching 1 percent. General anesthesia is commonly used. Various safety measures are available to eliminate any undue risk to the patient. A safe and simple one is the use of a plastic catheter as a temporary inlying bypass shunt. Partially occlusive carotid lesions are most suitable for operative removal. In total occlusion of the carotid, flow can be restored successfully in only about 40 percent of the cases.

The ideal patient for surgical treatment presents with transient cerebral ischemia, either focal or general. These attacks include dysfunction of the face, arm, leg, eye or speech, and dizziness, blackout spells or headaches. Three-fourths of these patients have repeated attacks, and about half of them go on to have a paralytic stroke if they are not treated. Eliminating the cause of such attacks, therefore, is a useful measure in stroke prevention. Recent reports indicate that in about 90 percent of patients the attacks of ischemia are completely eliminated

following carotid endarterectomy. In others, subsequent attacks are less frequent. Many of these patients have bilateral lesions and should have both carotids operated upon, but this should be done in separate stages at least one week apart. A carotid bruit is helpful in diagnosing a transient ischemic attack when the patient is seen in the asymptomatic phase. The judicious use of carotid arteriography to study such patients will bring to light an increasing number of persons in whom strokes can be prevented.

The role of endarterectomy in the management of patients with frank strokes is more controversial. It has become increasingly evident that arteriography and surgery should not be done as emergency measures in patients with profound paralysis and coma since the mortality is prohibitive and little is to be gained in the way of functional improvement. Such patients should be treated conservatively by the usual supportive measures, and suitable studies should be considered or carried out only after a stable situation has been achieved. Operation may be indicated in these patients to prevent further strokes or to hasten recovery. Frank stroke patients with less severe deficits who improve within a few days may be studied and operated upon with the expectation of further improvement and the prevention of future strokes. At least 60 percent of frank stroke patients give a history of previous transient ischemic attacks. This points up again the importance of recognizing such attacks and following through with an aggressive diagnostic and therapeutic approach.

Routine auscultation of the neck in patients without neurologic symptoms has revealed a surprisingly large number of patients with cervical carotid bruits. When studied by arteriography a majority show significant occlusive lesions. Without treatment many will go on to have a stroke. Operation in these cases may be termed prophylactic, but should be seriously considered when a lesion is demonstrated, especially if there is bilateral involvement. A carotid bruit is an important finding since a stenosis of 50 percent or greater is usually necessary for the bruit to become audible. Stenosis of this degree in the internal carotid artery warrants removal. Not only is blood flow reduced, but emboli from the plaque may pass distalward into the brain to produce symptoms. A cervical bruit may arise from the external carotid and require no treatment, but 90 percent of bruits originate in the internal carotid and are thus significant. Arteriography is necessary to distinguish these lesions. The absence of a bruit does not rule out pathologic conditions since the artery may be totally occluded or so tightly stenosed that no noise is audible.

In summary, it is important for the physician to be aware of the significant features of transient cerebral ischemic attacks since treatment is most rewarding from the standpoint of prevention of future strokes. Auscultation of the neck for carotid bruits is an important screening test for the presence of extracranial occlusive lesions. Increased application of cerebral arteriography in the investigation of patients with cerebrovascular insufficiency will reveal an increasing number of lesions responsible for stroke syndromes. The judicious use of carotid endarterectomy will result in the prevention of strokes in many patients and the improvement of neurologic status in others.

—JESSE E. THOMPSON, M.D., Dallas.

Radiation Hazards In Diagnostic Radiology

A SUBSTANTIAL body of evidence, some of which is circumstantial, indicates that low level radiation produces deleterious somatic and genetic effects in man. The somatic effects appear mainly in the bone marrow and result in leukemia[1,2] and perhaps other serious blood dyscrasias. The incidence of such bone marrow changes is certainly rare (about one case of leukemia per million per rad of exposure per year) and can only be detected where relatively large numbers of persons are exposed. The genetic effects in man due to low level radiation are for the most part subtle and difficult to detect.[3] Certainly, the detection and evaluation of such changes would depend on observation of a large number of people for many years. Such information is as yet unavailable, and most of our information comes from extrapolation to man from experimental results obtained in insects, rats and mice. Consequently, we have a twofold problem in evaluating radiation hazards due to diagnostic radiology: (1) to determine the amount of radiation administered to the general population in diagnostic radiologic procedures, and

(2) to equate this to the risks of producing serious somatic and genetic damage.

The concern of the general public and the medical profession was stimulated by the 1957 report of the National Academy of Sciences[4] in which it was stated that the largest single source of radiation exposure to the population in the United States originated from diagnostic radiologic procedures. The report went on to estimate the yearly gonadal dose from this source to be roughly equal to that of the background radiation which is present in our natural environment and cannot be avoided. This is about 100 to 125 milliroentgen equivalents man per year at sea level. However, a precise appraisal of the amount of radiation administered for diagnostic purposes in a country as large and diverse as the United States is a large task. For instance, a registration of radiologic installations in California shows that nearly one-half of the diagnostic facilities are under the control of non-radiologists, and a significant number of operators of x-ray machines are not physicians. Fortunately, many states now have radiation health codes which specify the physical factors under which x-ray machines must be operated. It is only recently, however, that there has been anything like adequate supervision available to enforce these specifications and thereby lead to standardizing exposure techniques.

Another variable is the frequency of use in various sections of the country of diagnostic radiologic procedures. Thus, it is probable that diagnostic x-rays are more frequently used in urban areas and in the vicinity of medical centers than in smaller communities and rural areas. Considerable helpful information was provided during the 1960 general census when questions were asked at random as to the number and type of x-ray examinations obtained during the preceding year.[5] This publication also supplied the age of the patients who received diagnostic procedures. This is another important factor in determining the genetic effects of radiation since these effects fall off quite sharply after 40 years of age and are completely negligible after the age of 50. By using the data contained in this publication, more precise estimates of weighted gonadal doses have been made.[6,7] These studies indicate that the weighted gonadal dose in the United States is of the order of one-fourth to one-fifth of the background level and is in the range of 16 to 23 milliroentgen equivalents man per year. Certainly, this is a level that might be reasonably expected to be obtained if the examinations are carried out under good technical conditions. Also, these studies are in close agreement with the extensive and well regulated survey (Adrian Report) in Great Britain.[8] Estimates of the absorbed dose to the bone marrow are as yet incomplete, but it again appears that this will be only a fraction of background radiation. Consequently, although a complete and final answer as to the extent of the hazards of diagnostic radiation has not been reached, it is now clear that the amount of radiation administered should be of the order of only one-fourth to one-fifth of that originally suggested in the report of the National Academy of Sciences.[4]

This crucial question arises: how much should the physician modify his practice regarding the use of diagnostic radiologic procedures? The present evidence suggests that the risk of somatic and genetic damage in patients over age 40 is minimal. On the other hand, the physician should be more critical of radiologic procedures which expose the lower abdomen of young people, particularly those procedures which involve fluoroscopy of the lower abdomen. This particularly applies to the examination of children, and of women during pregnancy. At present, these individuals constitute a minority of patients who come into most diagnostic radiologic departments. For the great majority of radiologic procedures, only the dictates of sound medical and economic practice should prevail. We should be sure that the radiation is administered under the supervision of physicians who are aware of the problems encountered in the economical use of radiation. Furthermore, referring physicians must exercise their best judgment in evaluating the risks of proceeding without adequate diagnostic information, which in most cases far exceeds the relatively minor risk of exposure to diagnostic x-rays.

—Robert N. Cooley, M.D., Galveston.

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Early Referral Aids Children Who Have Acute Leukemia

EARLY REFERRAL of children with acute leukemia to medical institutions specializing in leukemia research is recommended. Therapeutic results achieved in these institutions suggest that these patients can be helped; moreover, since acute leukemia is an uncommon disease, research will be facilitated by adequate numbers of patients. Because there is ample basis for the opinion among clinical investigators and basic scientists that the control of acute leukemia before many years pass is a real possibility, clinical and laboratory research is focused on leukemia patients at a number of medical institutions in the United States. This research is proceeding in three major categories:

1. *Viral.*—During the past ten years, it has been established unequivocably that viruses are a major cause of leukemia in rodents. The application of virological and other research "tools" to clinical material has produced suggestive—*though not conclusive*—evidence that viruses may play a role in the cause of human leukemia.

2. *Chemotherapeutic.*—The survival of children with leukemia has been increased fourfold since the first of five successful chemotherapeutic agents was introduced in 1947. At this date there are children who have survived eight years after diagnosis and have remained free of disease. Intensive use of combinations of these chemotherapeutic agents has reduced the number of leukemic cells in some patients to low levels, and the possibility of eradicating the leukemic cell populations with this approach appears promising.

3. *Research in Supportive Therapy.*—The major cause of morbidity and death and the major limitation to chemotherapy in patients with leukemia is hemorrhage and infection due to thrombopenia and leukopenia. Platelet transfusions have proven successful in the treatment and prevention of such hemorrhage and, in experimental studies, granulocyte transfusions have been effective in the treatment of life-threatening infections. Perhaps most important is the fact that such replacement therapy makes it possible to employ more intensive and, perhaps ultimately, curative chemotherapy.

These programs require extensive clinical and laboratory resources which are available only in these medical institutions. It is anticipated that once the control of leukemia is achieved, medical science will be a step forward in the control, in general or in part, of the other neoplastic diseases. To this extent, patients with acute leukemia are in the vanguard and are participating in programs which may not only help them and future patients with leukemia, but ultimately the cancer problem generally.

—GRANT TAYLOR, M.D., Houston.

A Small Hospital's Approach To Management of Strokes

WITHIN RECENT years rapid and radical changes in surgical and physiological concepts have greatly helped to remove the stroke syndrome from the area of hopelessness and passiveness to one of challenge, responsibility, and action. New developments in the fields of pathology, surgery, biochemistry, internal medicine, radiology, and physical medicine have led to a more aggressive and hopeful attitude toward the patient with stroke syndrome.

In spite of these changes, an informal survey conducted in 1965 by the Texas Medical Association's Committee on Cardiovascular Diseases disclosed the existence of the following standing emergency room orders in metropolitan hospitals for disposition of patients presenting with stroke syndrome:

1. If the patient can swallow, send him home.

2. If the patient cannot swallow, intubate him, observe him in the emergency room, showing the family how to care for the tube, and then send him home.

These hospitals all had intern and residency training programs.

Such an attitude of despair toward these patients is no longer justified.

While much of the work on the stroke syndrome has been done in medical centers, the mechanism and principles are now well enough understood and the techniques have been simplified so that every physician who deals with geriatric patients should have in mind a positive program to be followed in the management of the stroke syndrome.

This is the program used in a small-town general hospital in Central Texas.

1. All patients with a preliminary diagnosis of stroke syndrome are immediately moved to the hospital.

2. A complete history and physical examination is done by the admitting physician.

3. The entire x-ray and diagnostic laboratory concentrates on completing as rapidly as possible the diagnostic work-up.

A special effort is made to determine whether there have been previous evidences of cerebrovascular insufficiency. Such symptoms as vertigo, aphasia, visual disturbance, weakness in the face and the extremities, are carefully evaluated. The history also is carefully searched to help determine the exact onset of the present episode of symptoms and to determine the rate of progression of the symptoms. This is a most important element in the evaluation of the patient's prognosis. Finally, a careful drug history is obtained, especially related to hypotensive, diuretic, anticoagulant, and tranquilizer drugs.

Physical examination is equally important. The evaluation of the sensorium and careful evaluation of neurological deficit should be made on admission and, should surgery be undertaken, the neurological deficit should be reevaluated just prior to operation.

After completion of the history and physical examination, skull roentgenograms are obtained primarily in an effort to visualize the pineal body. Eye ground examination is done to seek evidence of increased intracranial pressure. If there is no evidence in the eye grounds of intracranial pressure elevation and the pineal body, when visualized, is not deviated from the midline, then a spinal fluid examination is done with particular interest in the presence or absence of blood in the spinal fluid and the manometric evaluation of the spinal fluid pressure. While the history and physical examination are being completed, complete blood count, urine, blood urea nitrogen, blood sugar, pH, arterial carbon dioxide pressure, and oxygen pressure tests, and electrocardiogram and chest roentgenograms are done as indicated.

It must be kept in mind that these patients are in the age group in which uremia, acidosis associated with respiratory failure or diabetes, hypoglycemic reactions, auricular fibrillation or myocardial infarction with attendant embolus, or Adams-Stokes syndrome, brain tumor, subarachnoid hemorrhage, and metastatic bronchogenic carcinoma can mimic cerebrovascular insufficiency.

In recent years the iatrogenic stroke syndrome has become more common because of some more powerful and effective drugs causing excessive dehydration, hypotension, and tranquilization. Each can be a source of stroke syndrome.

At this point in the hospital routine, a tentative categorization of diagnoses is made:

1. An expanding intracranial lesion, which calls for an early referral to a neurosurgeon.

2. Cerebrospinal infection, in which case antibotic therapy is immediately begun.

3. Medically related diseases with stroke syndrome as part of the primary pathology and those with stroke syndrome as secondary manifestation.

4. Cerebrovascular disease.

Patients falling into group four along with those in group three who have correctable physiological abnormalities, are considered for carotid angiography.

In general, indications for carotid angiography in patients who are otherwise physiologically normal are relatively unlimited. Our tendency in recent years has been to progress more toward active treatment of so-called impending stroke. However, this does not eliminate attempting to make an exact anatomical diagnosis on many patients who present within the first 12 hours after a so-called completed stroke episode.

In spite of the insistence of physicians in the medical centers that complete four-vessel studies must be done, we believe on a practical basis that primary carotid angiography done percutaneously enables us to anatomically define 90 to 95 percent of our cases which are amenable to vascular surgery.

It immediately becomes obvious that many patients presenting with the stroke syndrome, once an etiologic diagnosis is undertaken and, in many instances, successfully made, will not have indications for carotid angiography.

A recent evaluation of a random sample of 100 patients managed under the described regimen revealed that these patients had 166 admissions with a major diagnosis of stroke

syndrome; 49 were subjected to angiography and 23 of these were operated upon.

The overall mortality of the series based on total admission was 14 percent. The mean age of these patients was 76 years.

More important, however, than the mortality figures in this group of patients is the residual self-help ability shown by the survivors. Only 23 of the patients were subjected to surgery. It is therefore essential that a positive program for the remaining 77 cases is kept in mind. This program must encompass not only careful medical management of the numerous problems of this group, but also a positive program of physical retraining, social orientation, and family training.

Attitudes of despair and oversolicitude must be replaced by self assurance and hope for the patient and the expectation by the family of active function on the part of the patient.

Such a program is only possible when based upon accurate diagnosis so that definitive therapy can be started within the first few hours after admission. This time element is highly critical and even a few days' delay in beginning physiotherapy and retraining procedures greatly impairs their ultimate effectiveness and greatly prolongs the time required for retraining.

The program as outlined has evolved since 1959. The rewards in personal satisfaction as well as improved patient care have been great. Mortality rates have been reduced by two-thirds, hospital attitudes of hope infect whole families, and the dismissal of a decerebrated vegetable almost never occurs.

—Silas W. Grant, M.D., and
James M. Buie, M.D.,
Hillsboro.

PERSPECTIVE

Grief Begins Not with Death, But with Knowing It Is Near

ALTHOUGH it is not frequently thought of in these terms, the process of grief frequently begins before death, and can include the dying patient, his friends and relatives, *and* the professionals who seek to deal with the process of dying. Much consideration and study has been given to postmortem grief, but we need to know more about the interpersonal and intrapersonal process which is premortem grief.

We begin with a broad definition for the grief process which has helped me understand what is going on in the grief stricken person: Grief is the necessary total process, emotional, spiritual and physical, in which the individual terminates his relationship with an important person or thing in order to reinvest his life in other persons or things. It is a "total process" because it often includes emotional and physical suffering as well as painful religious reassessments. It refers to the termination of a relationship with a "person *or* thing" because grief may result from the loss of one's job or prized possession as readily as from the loss of one's mother-in-law; in fact, perhaps more readily! And grief is a "necessary" process for, without it, the old relationship cannot be adequately ended and other relationships will always suffer from emotional deprivation.

From the moment that a person learns that he is facing death, he begins to grieve. All of the feelings that are vital elements in the grief process may come into play: shock, disbelief, denial, anger. The dying person involuntarily begins the process of letting go of the world and those whom he has loved and who have loved him. If the grief process is successful, the dying person arrives at the moment of death with a peace born of an emotional detachment from the concerns of life. He is, in this sense, ready to die. At times, this process is so complete that researchers have been led to explore the possibility that the time of death is primarily determined by the completion of the process rather than physical deterioration.[1]

On the other hand, if the grief process is thwarted, the act of dying may be painful and tormented indeed. Our fear of the act of dying is considerably higher than our fear of death itself;[2] and perhaps not without good reason. Denial, isolation and depersonalization are the types of unhelpful but frequently expressed attitudes that contribute to the breakdown of the grief process. The patient whose minister tells him that he shouldn't worry but only put his trust in God can sense the fact that his minister does not want him to share his deepest fears and forebodings. The weather may become the accepted and comfortable topic of the day,

but the dying person has been denied the right to grieve and the potential service of sensitive pastoral care. The family that pretends that the dying person is going to get well, and spends endless hours reassuring everyone concerned by making plans for the future, contributes to the emotional isolation that is increasingly characteristic of the dying person's world. Robbed of human warmth and meaningful contact with important others, the patient quickly learns that he must not express the very feelings which he must express *if* grief is to be constructive. The physician who, accustomed to seeing people die, shuts off his emotional responses in the name of "professionalism" or "objectivity," sabotages the grief process by removing himself as a resource and by depersonalizing the dying person. When *all* persons seeking medical help become "cases," the dying person can no longer trust the physician with his deepest concerns.

Openness, honesty and freedom of contact and emotional expression are necessary characteristics of the kind of communication that the dying person needs in order to grieve. But, at the same time, these are not his needs alone. His relatives and friends need them too. In the name of some dubious god of mercy, many people have had their grief processes crippled in infancy by the game of "let's pretend." Immediate discomfort is avoided, for facing emotionally loaded moments of life can be uncomfortable, but they have also learned that it is unacceptable to express their deepest emotions. They defend themselves against unacceptable behaviour by covering over their real emotions and offering bogus representations of emotion in their stead. If the relatives of the dying person can be helped to begin their grief upon discovery of the imminence of death, share in the process with the dying person himself, and complete the process after death, their needs and the patient's will be served.

Those persons whose job it is to minister to the dying, whether the ministry be medical or religious, also need the freedom to grieve. If we avoid our feelings about death —the nurse by being busy, the physician by the rituals of medical procedure, the orderly by being flippant, and the clergyman by the desperate intonation of prayers—we become so involved with our own needs that the humanity of the patient may be lost and his needs relegated to a secondary position. It simply is *not* true that we have no feelings about death or about the people in the death beds we attend. What a breath of fresh air it is when in a group of clergymen, someone admits that he has felt anxious and unsure when called upon to minister to a dying person, and no one questions his faith or his pastoral competence! Other professional groups also have difficulty communicating about their real feelings in this area.

Physicians and clergymen often find that discussing the total care of the terminally ill provides a good initial bridge for communication between the two professional groups. Each is more aware of its need for the services and support of the other at this point than at almost any other juncture in their respective professional practices. It would be my guess that at least part of the reason for their mutual accessibility in this particular area is their involvement in the process of premortem grief. Even if they are unaware of their emotional commitment, its demands are best met when there is cooperation and sensitive understanding of each other's strengths and needs.

I hope that this helps to underline the need for far greater exploration of the emotional processes that precede death, for grief should not be thought of as a totally postmortem experience. It often begins before death and continues until some resolution has been achieved. If the grief process is encouraged and begun prior to the fact of death, the chances for an emotionally and religiously constructive resolution are enhanced. Ultimately, there are no exceptions to our Lord's observation: "Blessed are those who mourn, for they shall be comforted."[3]

—Rev. Robert E. Buxbaum,
Chief of Chaplaincy Services,
San Antonio State Hospital.

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Mediconfusion

Despite profound efforts by the Social Security Administration and Blue Cross to explain Medicare to the public and to the physician, confusion is prevalent.

Many physicians declare the cost of caring for these citizens is greater because extra time is necessary to explain the program to them and because additional help is needed to process claims. This situation is compounded, indeed, when the recipients are also receiving Old Age Assistance and the office patient-load is increased.

Another complaint by physicians is slow payment of fees. Texas Blue Shield reports that some 15,000 claims are received daily, and that 96 percent of these have satisfied the criteria of reasonable fees. The remaining 4 percent will be reviewed by a special joint committee of the Texas Medical Association and Blue Shield. If fees are higher than usual, the physician should enclose a short note of explanation.

Physicians have reported more difficulty in obtaining hospital beds; yet utilization committees seem to be functioning smoothly and only occasional difficulty has been reported in having physicians sign certification forms.

There now seems to be a ray of hope in solving the problems of the hospital-based specialist on the direct-billing procedure.

To help you with lessening your Mediconfusion, I offer the following helpful hints:

1. Get the Medicare number on each patient over age 65.
2. Get the OAA number on all those receiving Old Age Assistance benefits.
3. Have an ample supply of IRP (Individual Responsibility) or Blue Cross forms in your office.
4. Give a short explanation of higher-than-usual fees.
5. Keep accurate ledger records on all patients.

Sincerely,

James D. Murphy M.D.

President
Texas Medical Association

fall 1966

DORSEY Season

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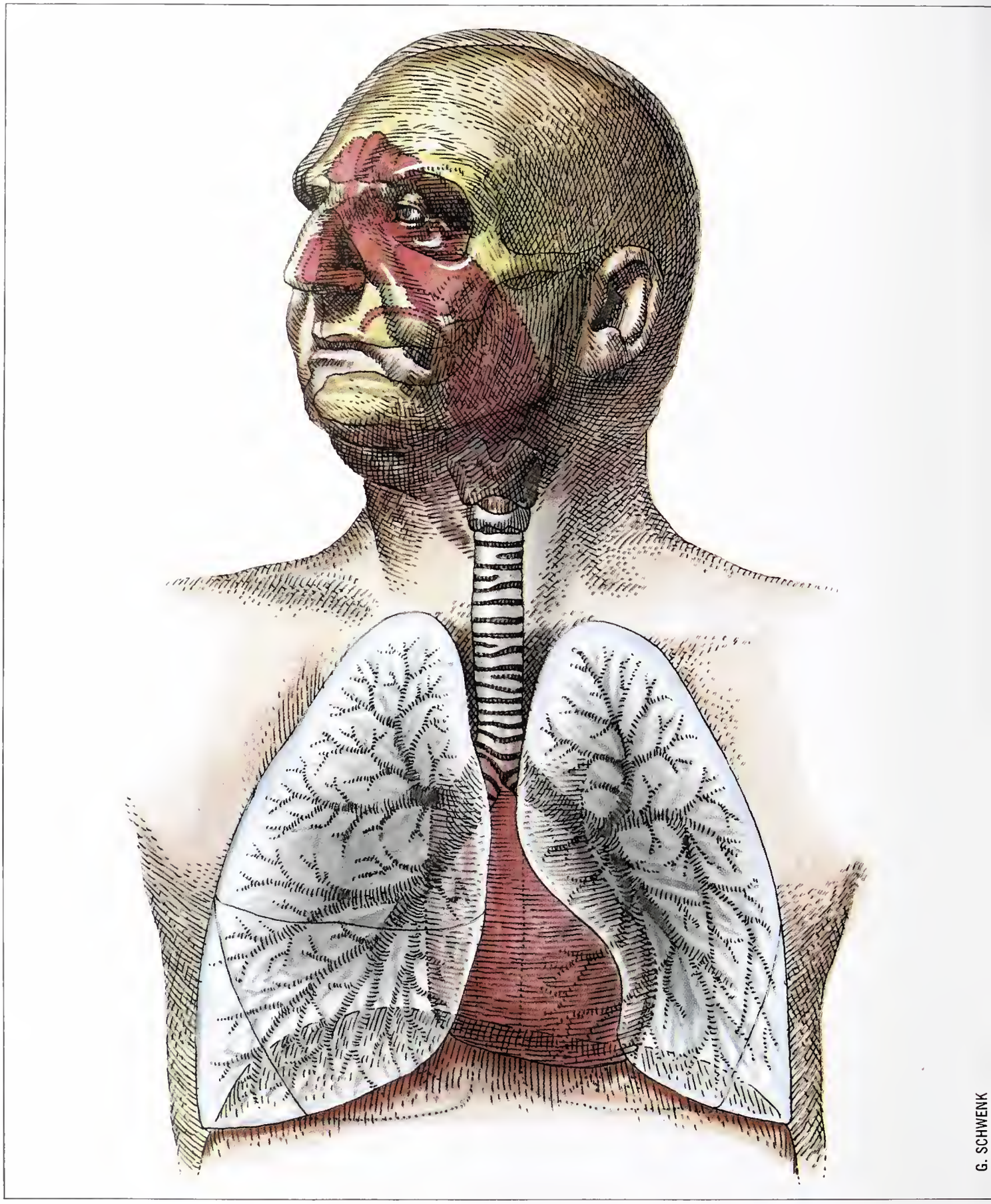
this issue: the common cold and the aging patient

the common cold and the aging patient

Louis J. Vorhaus, II, M.D., F.A.C.P.



G. SCHWENK

Effects of aging on the anatomic and physiologic aspects of the respiratory apparatus.

The chest becomes more fixed, less mobile and less elastic as the bronchial walls and thoracic ligaments lose elasticit The diaphragm and intercostal muscles atrophy and weaken. The lungs become smaller, flabbier and weigh less decreasing vital and total lung capacity, increasing residual volume and the alveolar dead space.

Sir William Osler described pneumonia as the welcome friend of the aged patient, because the patient with pneumonia usually died quietly. But today, the well-informed physician is an even better friend of the aging patient, since it is better to live than to die, no matter how quietly.

One of the first avenues of approach in the control of the hazards of respiratory disease in the aging patient is prompt and proper attention to the common cold or upper respiratory infection. The common cold may be the first step in the relatively short path to lower respiratory infection, broncho-pneumonia and death. This train of events occurs frequently among older persons. Indeed, pneumonia is one of the most common causes of their admission to hospitals and ranks high on the list of geriatric killers. Colds are more debilitating in elderly people and the aged are more likely candidates for secondary infections such as sinusitis and bronchitis. These infections, in turn, are more prone to lead to broncho-pneumonia, because of lowered resistance and anatomic and physiologic changes in the lungs of the elderly.

What is different about the respiratory tree of an aged person and that of an otherwise healthy younger adult? Aging certainly takes its toll on all parts of the body, affecting both anatomic and physiologic aspects of the respiratory apparatus. These changes are in part due to the wear and tear that occurs over the years; the repeated bouts of respiratory infection, long exposure to atmospheric pollutants, to occupational inhalants, smoking, malnutrition, obesity, inactivity and the development of other diseases which may affect the lungs.

With the passage of years, the lungs change. They become scarred and emphysematous and lose their compliance. The whole chest becomes more fixed, less mobile and less elastic.

The anatomic changes that occur in aging render the lungs less efficient. Tests of pulmonary function in senescence show a deterioration characterized by a decrease in vital capacity and total lung capacity, an increase in residual volume and alveolar dead space. Maximum breathing capacity is reduced and uniformity of ventilation deteriorates. These problems are often aggravated by the obstructed breathing, fever and secondary infection associated with the common cold, placing an additional stress on the entire cardiopulmonary reserve.

In addition, the efficiency of a cough is below par in older persons even though they are in good health. This is partly due to the decreased respiratory excursions and distensibility of the chest wall, and partly from loss of elasticity of bronchial walls which tend to make them collapse in a cough.

The elderly patient's resistance to infection is often reduced. Nutritional deficiencies are more common in aged people. There is some evidence to indicate that their capacity to respond to stress is less efficient. Finally, there is often relatively meager symptomatic response to acute disease. The absence of obvious or dramatic clinical signs and symptoms of severe illness is particularly dangerous because, coming as it

(concluded on following page)

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does in a person whose defenses are weakened both locally and systemically, pulmonary disease may progress rapidly to irreversible stages before medical attention is sought. Respiratory infection is especially hazardous because the aged patient responds badly to hypoxia. Not only is his response to oxygen lack impaired, but the work of breathing, due to decreased compliance of the lung and increased stiffness of the thorax, is markedly augmented.

Many patients late in life are in a precarious and delicate cardiopulmonary balance which is easily decompensated from relatively minor insults such as colds and upper respiratory infections.

For all of these reasons, geriatricians long have stressed the importance of preventing respiratory insults. Today we have better ways of treating respiratory infection, improved techniques for clearing the lungs and bronchial tubes of secretions and better understanding of ways of improving ventilation. We possess a broader spectrum of antimicrobial agents including newer ones to deal with previously resistant organisms. Even so, the death rate from pneumonia is high in older people, and it is preferable to avoid the disease than to treat it. To do so, attention must be paid to the general maintenance of good health and all that implies, as well as to the prevention, elimination and treatment of associated conditions that predispose to or cause pneumonia such as chronic upper or lower respiratory infection, respiratory allergy, chronic sinusitis and exposure to inspired irritants.

Colds and other minor respiratory infections, which favor the development of broncho-pneumonia, should be treated vigorously and promptly, particularly those patients whose aging process has been accompanied by the development of chronic pulmonary disease. Upper respiratory passages should be cleared with decongestants. Sinuses should be drained adequately. And, when indicated, appropriate antimicrobial therapy should be instituted before serious infection of the lower respiratory tree supervenes.

In decades past it was understandable that physicians welcomed pneumonia for the aged patients because it offered them a quiet and peaceful demise. Today we recognize that in many cases, peaceful as it may have been, such deaths were often avoidable. With the current knowledge and understanding of the problems that respiratory infections impose on aging people, vigilant medical attention can often restore them to a vigorous, rewarding and productive life so that the many opportunities that exist today for people to enjoy their golden years are realized and not stolen by untimely death.

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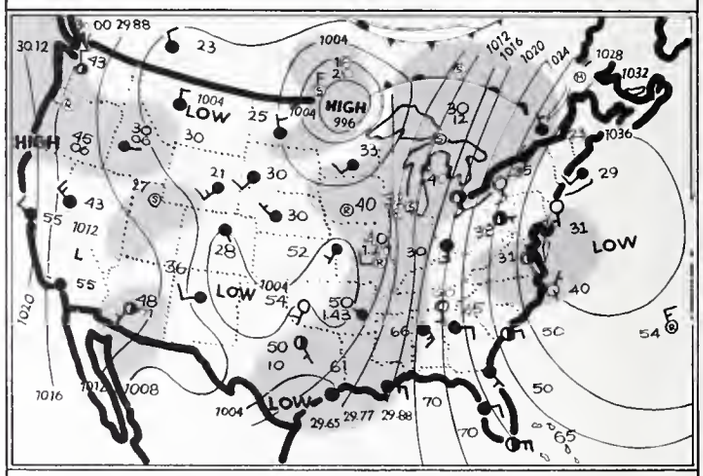
Weather Forecast Throughout the Nation



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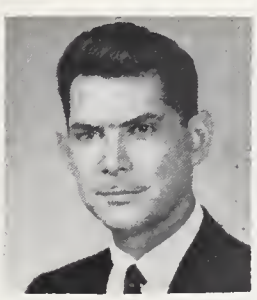
meet the authors

BOB R. ALFORD, J. R. DICKEY, and **F. EUGENE PRATT** are with the Department of Otolaryngology, Baylor University College of Medicine, Houston. **Management of Facial Paralysis, p. 56.**

NORMAN E. WRIGHT, Amarillo, practices otolaryngology and emphasizes head and neck surgery. **Neurilemmoma of Cervical Portion of Vagus Nerve, p. 61.**

LEROY J. KLEINSASSER is a Dallas surgeon, associated with Baylor University Medical Center there. He discusses **Surgical Treatment of Strokes, p. 65.**



LUIS LEON-SOTOMAYOR, clinical instructor in medicine at the University of Texas Medical Branch, Galveston, was serving in the US Army Reserve when he and his co-author, **DR. ROBERT DIETRICH,** reported a case of **Marfan's Syndrome with Coarctation and Aneurysms of Aorta, p. 69.**

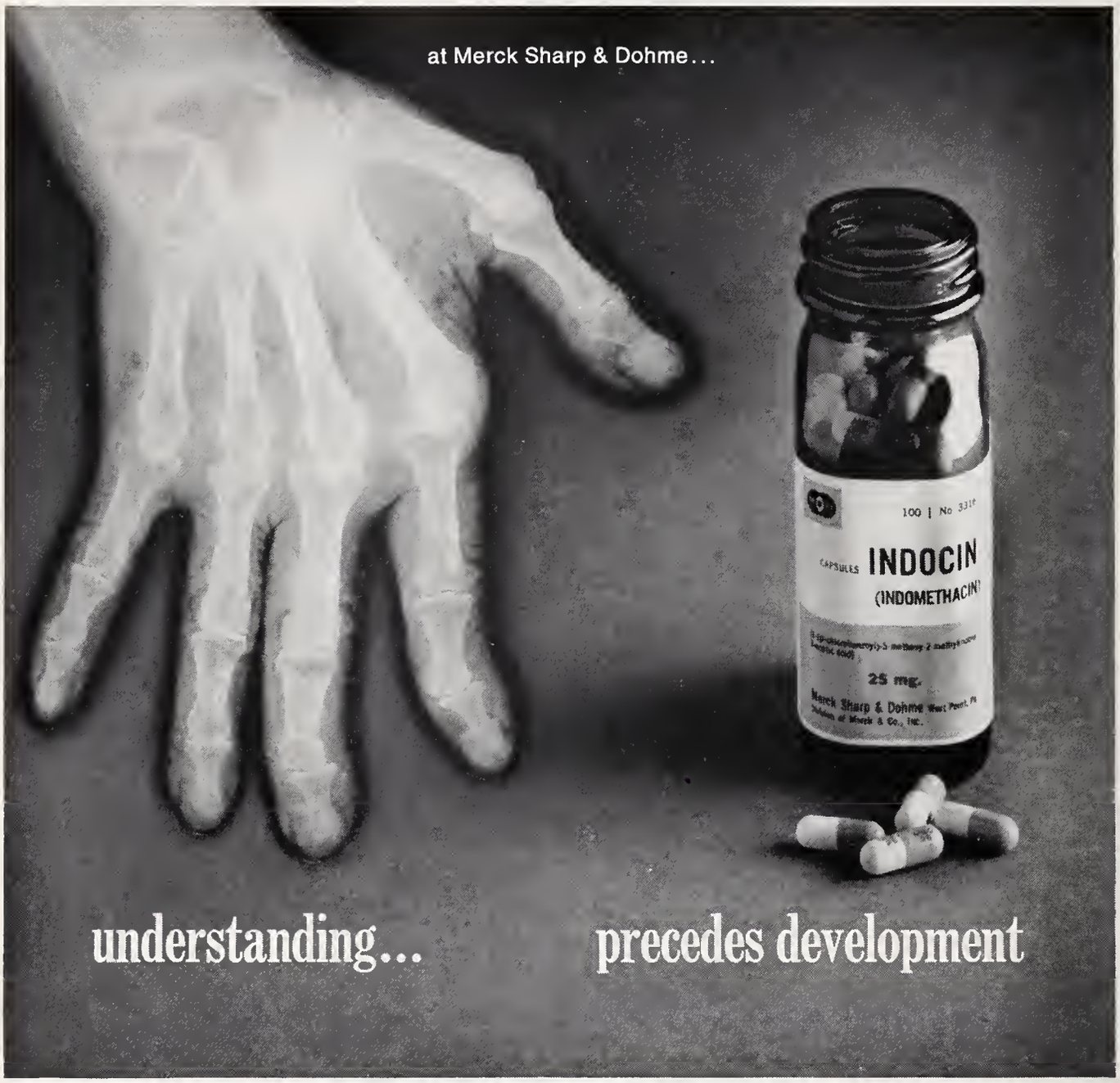
HOWARD LEVIN, now in San Diego, was a fellow in hematology at Baylor, where **CLARENCE P. ALFREY, JR.,** is associate professor of medicine. Both worked with the Radioisotope Service, VA Hospital, on **Thrombotic Thrombocytopenic Purpura; Temporary Remission Induced by Prednisone, p. 72.**

ROY D. WILSON, associate professor in the Department of Anesthesiology, at the University of Texas Medical Branch, outlines the need for more qualified anesthetists in **Trends in Anesthesia Coverage in Texas, p. 80.**

RALPH J. CAPAROSA is clinical assistant professor of otolaryngology at the University of Pittsburgh School of Medicine. In April, he was a guest of the Texas Medical Association and the Texas Otolaryngological Association. **Meniere's Disease; Diagnosis and Management, p. 84.**

DUDLEY JONES, 1965 graduate of University of Texas Southwestern Medical School, was an intern and is now a resident in pathology at Parkland Memorial Hospital, Dallas. **Clinicopathologic Conference, p. 92.**

ALEXANDER GOL, who was trained at Guy's Hospital Medical School, England, is assistant professor of neurosurgery, Baylor University College of Medicine, Houston. **Hippocampectomy for Relief of Intractable Pain, p. 76.**



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Management of Facial Paralysis

REVIEW ARTICLES

THE SUCCESSFUL management of facial paralysis requires an understanding of the disease process producing the paralysis and the precise effect of the disease upon the functional integrity of the facial nerve. The appropriate medical or surgical measures may then be enlisted to ensure a satisfactory end result in each patient. The purpose of this paper is to review the practical clinical considerations in facial paralysis and to describe the diagnostic tests that are helpful in determining the most effective therapy.

Etiology of Facial Paralysis

Facial paralysis has been noted to occur in many disease processes, but is most frequently due to trauma, otitis media, herpes zoster, tumors, or an idiopathic cause (Bell's palsy). Traumatic facial palsy may result from surgical procedures (neurosurgical or otological) or from external force (head injuries, temporal bone fractures, or lacerations). Both acute and chronic otitis media, at times, may cause facial paralysis. In chronic otitis media, a cholesteatoma is often responsible for the palsy. Herpes zoster of the geniculate ganglion area of the facial nerve may produce facial weakness, and presents as the well-known Hunt's syndrome. An idiopathic disease process, usually referred to as Bell's palsy, is responsible, however, for the large majority of peripheral facial paralysis. Vascular ischemia of the nerve is the most commonly accepted pathologic mechanism for the idiopathic cases. More recently, however, a virus has been suggested by Blatt[1] as the causative agent in Bell's palsy patients.

In Table 1, an etiologic classification is presented for 55 patients with facial paralysis (central and peripheral) that were treated in the Baylor University Affiliated Hospitals Program between 1962 and 1965.

Neuroanatomy

Anatomical considerations are important in the diagnosis and treatment of patients with facial paralysis. The unusual peripheral course of the facial nerve is worthy of review here because it bears directly upon diagnostic and therapeutic techniques. After it exits from the brain stem, the facial nerve enters the internal auditory meatus and courses through the temporal bone to the stylomastoid foramen, where it leaves the bone and turns forward to enter the parotid gland and divide into branches supplying the facial muscles (Fig. 1). Lesions at any level

TABLE 1.—*Etiology of Facial Paralysis, 1962-1965.* Baylor University Affiliated Hospitals

Etiology	*No. Cases*
Idiopathic (Bell's palsy)	22
Otitis media	7
Associated with viral lesions (herpes zoster)	6
Trauma	
Fractures	5
Postoperative: neurosurgery or otology	4
Birth injury	2
Associated with other conditions (diabetes, seizures, multiple sclerosis, cerebral tumor, leukemia, cardiovascular conditions, carcinoma of parotid)	9
Total	55

The neuroanatomical, clinical diagnostic, and electrodiagnostic features of facial paralysis are discussed; management is based upon a thorough understanding of these factors. The primary objective is complete recovery of facial function without the unfortunate consequences associated with degeneration of the nerve. It is believed that this approach provides a firm scientific basis for the management of facial paralysis.

BOB R. ALFORD, M.D.
J. R. DICKEY, M.D.
F. EUGENE PRATT, M.D.

of the nerve, from the brain stem to the parotid arborization, usually result in paralysis of all branches to the facial muscles. However, lesions more central than the facial nerve nucleus in the brain stem produce an incomplete facial paralysis characterized by preservation of forehead muscle function. This sparing of forehead function is the result of bilateral cortical innervation of these muscles.

The facial nerve is a mixed nerve containing efferent (motor) and afferent (sensory) fibers. There are two types of efferent fibers: one group provides motor supply to the facial muscles and has its cell bodies in the facial nucleus in the brain stem; the other group supplies secretory fibers to the lacrimal gland, nasal mucous glands, and the submaxillary and sublingual salivary glands. The cell bodies for the latter group of fibers are in the superior salivary nucleus. The efferent fibers to the lacrimal gland and nasal glands leave the facial nerve at the geniculate ganglion in the greater superficial petrosal nerve. Those fibers to the submaxillary and sublingual glands leave the nerve near the stylomastoid foramen in the chorda tympani branch.

Similarly, there are two types of afferent fibers in the facial nerve: one group carries taste sensation from the anterior two-thirds of the tongue via the chorda tympani nerve; the other group conveys sensation from part of the ear canal and ear drum. All afferent fibers have their cell bodies in the geniculate ganglion.

Knowledge of the site of the lesion is important in the diagnosis and treatment of patients with facial nerve palsy. The different motor and sensory functions mediated by the fibers of the facial nerve usually make it possible to determine this precisely. For ex-

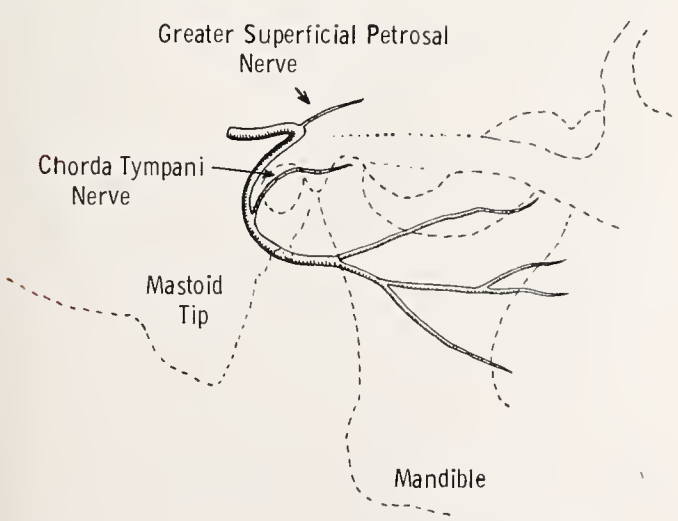

Fig. 1. Facial nerve with its peripheral branches.

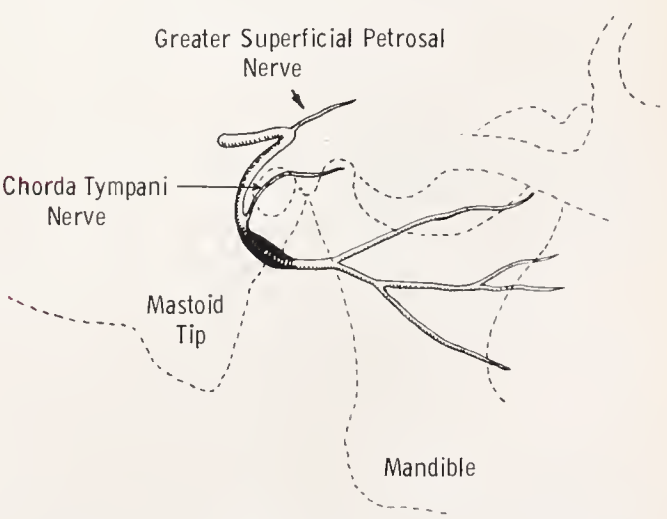

Fig. 2. Lesion peripheral to the chorda tympani branch.

ample, if taste is present and lacrimation is normal, the lesion is distal to the chorda tympani branch (Fig. 2). However, if taste is absent on the involved side, but tearing is present, the lesion is between the geniculate ganglion and the chorda tympani, that is, in the mastoid or middle ear segment of the nerve within the temporal bone (Fig. 3). If taste and tearing are both impaired, the lesion is at, or central to, the geniculate ganglion (Fig. 4).

Clinical Diagnostic Studies

A careful history of the onset and clinical course of the facial weakness is often revealing and provides the physician with information from which a positive therapy program may be planned. For example, in traumatic cases, a delayed onset of the paralysis is associated most often with good return of function; but cases that have immediate onset of facial weakness require serious consideration for exploration and repair of the nerve, as an unsatisfactory return of function may occur.

In idiopathic Bell's palsy patients, pain at the onset of the paralysis is a serious sign as these patients often have reactions of degeneration. In our patients, eight had pain at the onset and underwent degeneration. In contrast, only two of nine patients without pain had reactions of degeneration.

Careful clinical examination of the patient also provides information that is useful to the physician in determining what course of therapy to follow. It is important, therefore, that the clinician assess the function of the facial muscles at every visit with the patient during the course of facial paralysis. Records of the function will demonstrate, over a period of weeks, whether or not there is improvement in the patient's facial motion. When assessing movement of the face, it is best to test and record the movement of each major muscle group (frontal, orbicular, nasal, and labial) separately. This can be done by asking the patient to wrinkle the forehead, close the eye tightly, wrinkle the nose, whistle, and smile. A quantitative estimate of the strength of the muscle groups may also be made.

Other clinical diagnostic tests of taste and lacrimal function permit the physician to localize the lesion. The most practical test for taste function is the use of chemicals. In chemical testing with sweet (sugar) and bitter (salt or vinegar) solutions, the tongue is dried off carefully and the patient is asked to indicate sweet or bitter whenever the test solution is applied. It is important that the tongue remain outside the mouth during the study in order to prevent the distribution of the test solution over a larger surface area than the anterior two-thirds of the tongue. A comparison is made with the normal side of the tongue. In the future, electrical testing of taste function may prove to be a more sensitive method; however, it is not generally available at present. In electrical tests, as described by Krarup,[4] a low current is applied to an electrode placed over the anterior two-thirds of the tongue and the patient is asked to indicate whether or not he perceives a metallic taste sensation.

The measurement of submaxillary salivary gland flow[1] is another test for chorda tympani function. It is performed by cannulating both submaxillary ducts and collect-

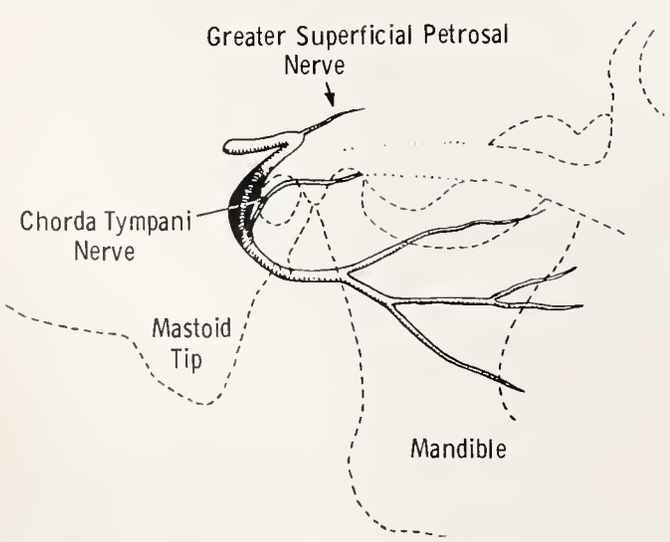

Fig. 3. Lesion between the greater superficial petrosal branch and the chorda tympani branch.

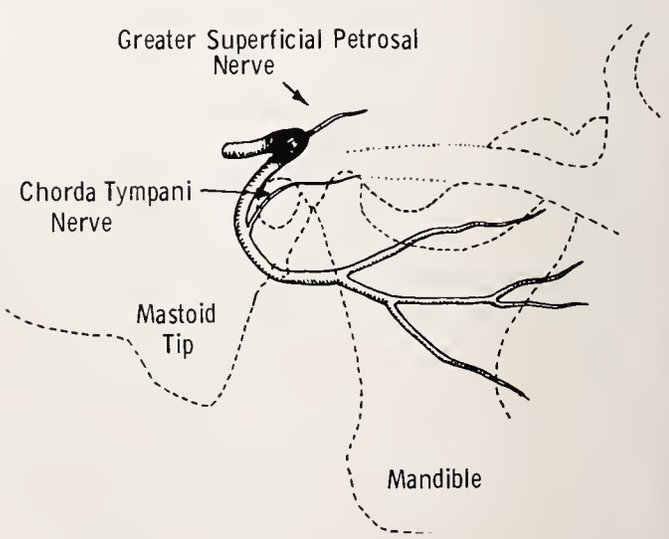

Fig. 4. Lesion at the geniculate ganglion.

ing the saliva in separate test tubes. A reduction in flow of saliva from the paralyzed side implies loss of chorda tympani function.

Lacrimation may be tested by placing a strip of filter paper in the lower conjunctival fornix of each eye. Provocation of tearing by stimulating the nose mechanically or with ammonia can produce a maximum flow. This test is referred to as the Schirmer test. Comparison of the tear flow from each eye is made. If tearing is decreased on the side of the paralysis, the disease process is at, or central to, the geniculate ganglion.

Electrodiagnostic Studies

In recent years refinements in electronic equipment have made it possible for clinicians to determine the neurophysiologic integrity of the facial nerve. At present, two different types of electrodiagnostic techniques are used to assess the neurophysiologic status of the facial nerve. They are nerve stimulation and electromyographic studies. Each of these techniques provides valuable information when performed correctly and at the proper time in the course of the paralysis.

The nerve stimulation technique that we have used primarily is the nerve excitability test. This study is carried out by stimulating the main trunk of the facial nerve at the angle of the mandible with an external electrode. A square wave pulse of short duration is used as the stimulus. The pulse intensity (current) is varied. The threshold of excitability is determined as the least amount of current that produces a just-visible twitch of the muscle groups in the major divisions of the facial nerve. Animal and clinical studies by Rosenberg and Alford,[5] Campbell,[2] and Collier,[3] have shown that nerve excitability tests are useful after 72 hours have elapsed from the onset of the paralysis. At that time, if degeneration of the nerve (denervation) is occurring, there will be an increase of 3 milliamperes (ma.) or more in the nerve excitability threshold on the paralyzed side as compared to the normal side. On the other hand, if the nerve is in a state of neurapraxia (conduction block), the nerve excitability threshold should not exceed that of the normal side by more than 2 ma. After 72 hours have elapsed, this study should be done daily in order to recognize as early as possible a change in threshold that would indicate degeneration. The testing is continued daily until there is clinical evidence of recovery of facial function.

The other electrodiagnostic technique, the electromyographic study (EMG), is helpful in determining the electrical activity of facial muscle fibers. A needle electrode is inserted into a facial muscle and observations are made at rest and upon attempted voluntary motion. The differences between a normal silent resting potential, voluntary motor unit potential, fibrillation potential, and polyphasic reinnervation potential, are easily observed. Although EMG studies are useful in determining degeneration (denervation), 12 to 14 days must have elapsed from the onset of the paralysis in order for this reaction to be recorded. This is much later than it is desirable to recognize such a serious event as degeneration. Therefore, early in the course of the paralysis, EMG studies are less valuable and informative than the nerve excitability studies. Electromyography may be helpful earlier in the course of a paralysis, however, if voluntary motor unit potentials can be demonstrated, as their presence suggests that functional continuity of the nerve is present. EMG studies are also useful in determining whether reinnervation is occurring. Polyphasic reinnervation potentials are present in advance of clinical improvement in facial function and provide valuable reassuring information when a paralysis has existed for some time.

From our own observations[5] and those of Collier,[2] the reliability of these electrodiagnostic studies in determining the neurophysiologic state of the facial nerve in patients with paralysis is good. In 29 patients in the Baylor Affiliated Hospitals Program who were studied with electrodiagnostic methods, all whose studies demonstrated neurapraxia recovered without incident. However, only 11 of 14 patients whose nerves degenerated recovered facial function; and, of these 11, 10 had synkinesis, incomplete return of function, or both. As would be expected, the average time for return of function was one month in patients whose electrodiagnostic studies indicated reactions of neurapraxia. However, in patients whose studies revealed degeneration, the recovery time averaged 2.7 months.

Because electrodiagnostic studies, particularly nerve excitability tests, reliably indicate degeneration early, it is possible to differentiate early between patients who will recover spontaneously and those who will have permanent impairment of facial function. By so doing, effective therapy programs can

be instituted that will be directed toward obtaining the best functional end result.

Management of Facial Paralysis

The major considerations in planning the most effective therapy program for patients with facial paralysis are the etiology of the palsy, the location of the lesion, and the functional state of the nerve.

Perhaps the most perplexing cases to manage are those of idiopathic etiology, i.e., Bell's palsy. Because the large majority of these recover spontaneously, the traditional treatment was one of expectant waiting. The 10 percent of patients who did not recover were consoled and occasionally operative decompression of the facial nerve was performed, but only after it was evident that return of function was not forthcoming. The 30 to 40 percent of patients who incompletely recovered movement, had synkinesis, or both, were considered to have a successful outcome in spite of their disconcerting function. It is this latter group of patients, as well as the 10 percent that fail to recover, that demand a reassessment of the diagnostic and therapeutic programs utilized in the past.

If the patient consults the physician soon (within 48 hours) after onset of idiopathic Bell's palsy or paralysis due to herpes zoster oticus, a medical program with steroids is instituted. This is primarily for the purpose of reducing edema and/or inflammatory reaction. Electrodiagnostic studies of neurophysiological function are done immediately and then daily thereafter until evidence of return of function is present. As demonstrated above, these studies provide information that permits the clinician to differentiate lesions of neurapraxia (conduction block) from lesions of denervation (Wallerian degeneration). Medical treatment may be continued in confidence if daily excitability tests persistently indicate neurapraxia. However, if these studies indicate that denervation is occurring, serious consideration must be given to surgical decompression of the nerve if one is to avoid the unfortunate consequences associated with regeneration of a degenerated nerve (i.e., incomplete return, synkinesis and contractures). In support of this, we found that in ten of our patients with Bell's palsy, five had electrodiagnostic studies that persistently indicated neurapraxia, and each had complete return of function. The other five patients had reactions of degeneration on testing. One of these did not recover and the other four recovered but had synkinesis movement. None of the patients whose nerves degenerated had decompression of the nerve at the first indication of degeneration (denervation). The authors believe that the problem of synkinesis movement would have been reduced if these patients had undergone nerve decompression when the reaction of degeneration was first evident.

The management of patients who develop a facial paralysis from chronic otitis media or acute otitis media is usually less complicated than the management of patients with Bell's palsy. It must be remembered, however, that those patients with chronic otitis media have a more serious prognosis than those with acute otitis media. In the patient with chronic ear disease, prompt surgical decompression of the nerve must be undertaken if one is to avoid the serious consequences of degeneration that often occur when this process remains untreated. In acute infections, electrodiagnostic studies should be performed at frequent intervals during the course of treatment and if an indication of degeneration appears, exploration of the middle ear and mastoid and decompression of the nerve should be done. However, in most instances of acute otitis media, the facial paralysis is transient and improves when the chemotherapeutic and local program for the infection becomes effective.

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Neurilemmoma of Cervical Portion Of Vagus Nerve

Norman E. Wright, M.D.

Neurilemmoma of the cervical vagus nerve is uncommon; its only constant presenting symptom is a lump or mass in the neck. Hazards of unilateral resection of the vagus nerve above and below the origin of the superior laryngeal are discussed. A case is presented in which dissection of the tumor from the nerve was possible, with complete recovery of function of the ipsilateral vocal cord within four months.

CASE REPORTS

OTOLARYNGOLOGISTS are often asked to diagnose a lump in the neck, and a number of causes readily come to mind: congenital cyst, lymphoma, metastatic tumor, aneurysm, carotid body tumor. A rather definite diagnosis is usually possible preoperatively. However, at times no definite factors in the history, physical examination, or laboratory findings suggest the nature of the disease, and exploratory surgery is required to furnish a definite diagnosis. Among the less common final diagnoses is neurilemmoma of the vagus nerve, and such a case is reported.

Pathology

The precise origin of the fibrous tumors of the nerves is not absolutely known. Genesis from the perineurium or the epineurium indicate a mesodermal origin, and such lesions should be termed neurofibromas. The neurilemma, however, is neuroectodermal in origin, and contains the cells of Schwann. Tumors from this source may contain melanocytes and nerve cells, with such lesions being called schwannomas or neurilemmomas. The publication of tissue culture studies by Masson, and Murray and Stout, is generally regarded as being fairly conclusive evidence of the origin of this tumor from neurilemma.

Neurilemmoma may arise from virtually any cranial, peripheral or sympathetic nerve, and grossly will have a globular or fusiform shape when small, but configuration of larger tumors may vary. If the nerve of origin is small, its fibers will be found in the capsule if they can be found at all. Due to tumor origin inside the epineurial sheath, the fibers of larger nerves separate into fasciculi spread over the surface of the tumor by the expansile nature of the growth.

Usually, these tumors grow slowly; interference with function is a late development, and pain is rare. The lesion is characteristically a solitary, benign, encapsulated structure which may vary in size up to several centimeters. Color varies from a shiny white to pink to pearl gray. The encapsulated state grossly suggests the benign nature of the lesion. Consistency will depend on the extent of cystic degeneration which has occurred. On section, cystic spaces of various sizes are seen; the larger the tumor the softer it is, and the greater the cystic areas.

Verocay described this tumor in 1908, and Verocay bodies have the microscopic appearance of organoid structures which resemble tactile corpuscles. The microscopic appearance of neurilemmoma is differentiated from neurofibroma chiefly by the attempts to form these specialized neural structures in the former. Histologically, a neurilemmoma has two types of cellular arrangement, designated as Antoni type A and Antoni type B.

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In the former, a more solid cellular pattern is observed, with palisading of nuclei or alignment almost in rows with the intervening spaces between the rows containing no nuclei. Cords or bands of such tissue are held together by a delicate connective tissue, and the Verocay bodies are composed of type A tissue. The type B tissue is loosely placed between the masses of type A, and is composed of Schwann cells set irregularly in a pattern of microcysts and delicate reticulin fibers. Consolidation of the microcystic lesions forms the cystic spaces, which explains the variable cross sectional appearance and consistency of the lesions. This degeneration is of a serous type, and no mucin, epithelium nor hyaluronic acid is present. Thick hyalin sheaths are often seen around the vessels. Stout[1] has stated that neurilemmoma rarely recurs after removal, even if the capsule is left behind. He also questions that malignant change ever occurs in these lesions.

Clinical Data

The exact incidence of this lesion is uncertain, since some reports list this lesion under classifications which include other nerves of origin.[2-4] To illustrate this point, of three 1962 articles dealing with neurilemmoma of the cervical vagus, 16, 23, and 12 cases were listed as the total number, including cases which the authors reported.[5-7] While the exact number recorded in all medical literature is not a matter of great importance, these figures indicate that the entity is not common.

No sex dominance is recorded. The age of patients with neurilemmoma in the head and neck ranges from 5 to 75 years with an average of 44.6 years in the series reported by Kragh, Soule and Masson.[4] Their study was based on 143 such patients seen at the Mayo Clinic between 1910 and 1957. From their group of 80 neurilemmomas arising in the lateral cervical region, the nerve of origin could be determined in only 22, of which 5 were from the vagus. Altany and Pickrell[6] stated that these tumors are most commonly found between the third and fifth decades. In a report from the New York Presbyterian Hospital in 1956, Gore, Rankow and Hanford[3] reported 389 cases of neurilemmoma. Of this number, 251 lesions, or 64.4 percent, were located in the central nervous system, and 138, or 35.4 percent, were outside that area. Of this group of 138 lesions, 52, or 38 percent, were found in the head and neck, with four involving the vagus nerve.

The only constant presenting complaint is a mass or localized swelling in the neck.[6] Ehrlich and Martin[2] comment that absence of a visible primary lesion, coupled with the usual long duration of the tumor, should lead to the diagnosis of a benign primary tumor, and that no one can proceed beyond this point on clinical aspects alone. Leichtling and associates[8] drew attention specifically to the production of cough by palpating the tumor in their two cases. Horwich and Hawe[9] had earlier mentioned this phenomenon in their two cases, but had placed no particular emphasis on this point. In the instance to be reported, this symptom was absent. The majority of recorded cases are in the upper part of the neck.[6,10]

Ehrlich and Martin[2] were able to make a preoperative diagnosis in five cases out of six neurilemmomas of the head and neck by means of aspiration biopsy. Conley[11] has also recommended this procedure as a diagnostic aid. Numerous authors mention the supposed side-to-side mobility of these lesions, but state that motion in a vertical plane is not expected. Differentiation from carotid body tumor is attempted by means of palpating the pulsation in the latter. Mobility of the lesion, however, will doubtless depend to a great extent on its size. Correct preoperative diagnosis without aspiration biopsy was not found in the literature.

History of radiation was recorded in two cases without destruction of either lesion.[2,4] The consensus is that these tumors are radioresistant, and should be treated by total surgical excision when feasible. If the fibers of the nerve cannot be identified sufficiently well to dissect them from the tumor, removal of a normal segment of nerve above and below is indicated.

Specific Hazards

The vagus nerve is intimately associated with many vital structures in the neck, but since this tumor enlarges by intracapsular expansion, its separation from those other structures can normally be accomplished. The question thus becomes primarily one of the results of unilateral vagectomy, and the anticipated results are observed clinically. When excision of the vagus is done below the nodosal ganglion where the superior laryngeal takes origin, the principal effect is ipsi-

lateral vocal cord paralysis. From that entity, the voice will have an abnormal, husky sound, but after a short time the opposite cord will normally swing beyond the midline to approximate to its paralyzed counterpart. Effective laryngeal closure during the act of deglutition can thus be expected, and many case reports bear this out.

Resection of the vagal trunk above the origin of the superior laryngeal nerve, however, produces a different problem physiologically, with induction of ipsilateral laryngopharyngeal anesthesia and paralysis. Martin and associates[12] state that tracheal aspiration and pneumonitis must be anticipated, with transitory dysphagia from interruption of motor branches to the pharyngeal constrictors. Conley[11] describes such a case in which tracheostomy was done at conclusion of the operation, and a nasogastric tube was required for six weeks. His patient subsequently learned to eat without difficulty.

Anastomosis of the nerve after resection is urged when possible, and placement of a nerve graft otherwise.[7,11] Bales and Raines[7] reported that the ends of the vagus nerve severed below the origin of the superior laryngeal were sutured, and after a year the vocal cord recovered its function. Report of successful graft of the superior laryngeal nerve was not found.

Report of Case

A 48-year-old white woman, seen initially on Dec. 26, 1961, had discovered a small lump in the lower right side of her neck some six months earlier. She sought advice from her family physician, though no other symptoms were associated. After various thyroid tests were done and found to be within normal limits, the patient was advised that a period of observation would be in order. She felt that the lesion had been growing slightly, and she had noted slight hoarseness occasionally after talking for prolonged periods, and an occasional sense of pain over the right mastoid, associated with stiffness in the right side of her neck of a few hours' duration. The inference drawn was that the patient probably was holding her neck rigidly while doing clerical work, and that muscle spasm was responsible for her symptoms. A negative history was obtained for cough, dysphagia, dyspnea, weight loss, regurgitation, esophageal obstruction, and the symptomatology of hypothyroidism and hyperthyroidism. The family history held no relevant factors. Review of past medical history disclosed a single pregnancy with normal delivery at term, and no disease except the contagious diseases of childhood. In addition, the patient stated that she had had a number of gynecologic operations, the last being removal of a cervical stump some three years previously.

The only positive finding on inspection and palpation of the neck was a mass measuring about 1.5 cm. in the lower right side of the neck, lateral to the trachea, and anterior to the medial border of the sternomastoid muscle. In vertical alignment the lesion was slightly above the inferior border of the cricoid cartilage. The mass was not tender to palpation, and cough was not elicited by this means. With the neck flexed, and the chin rotated slightly to the right, the mass seemed quite mobile in all directions, but seemed to be located fairly deeply in the neck. To both inspection and palpation the lesion did not move on deglutition. The thyroid gland was not enlarged to palpation. The rest of the general physical examination was within normal limits.

The patient was admitted to St. Anthony's Hospital on Jan. 1, 1962, and results of routine laboratory studies were within normal limits. On the following morning, under endotracheal anesthesia, exploration was carried out through a right transverse incision made through a skin crease at the level of the inferior border of the cricoid cartilage. The platysma was reflected with the skin, and palpation indicated the mass to be deep to the strap muscles. The medial border of the sternomastoid was detached from the strap muscles and retracted laterally with the superior belly of the omohyoid. The mass could be easily palpated, and its mobility confirmed. A retractor was placed to hold the other strap muscles medially, and the mass was grasped and lateral traction applied to it. Dissection down to the mass opened the carotid sheath with the common carotid artery being found medially, the internal jugular vein laterally, and the vagus nerve was visible above and below the mass. By a technique of capsular dissection the tumor was separated from the nerve, which in spite of being thinned somewhat by the presence of the tumor on its lateral border, could be preserved apparently intact. The cleavage plane seemed quite clean, and the tumor which grossly suggested neurilemmoma, was handed to the pathologist for sectioning in the operating room. Frozen

section examination confirmed this impression, and a few remnants of capsule were dissected from the nerve trunk. The wound was closed in layers after the precaution of inserting a small Penrose drain into the depths, to be removed the next day.

The next day permanent sections confirmed the frozen section diagnosis.

The patient was not aware of any dysfunction except for a minimal hoarseness in addition to what one usually expects following endotracheal anesthesia. She was discharged from the hospital on the third postoperative day. Sutures were removed from the neck wound on Jan. 8, and examination of the larynx with a mirror at that time indicated a slight weakness of the right vocal cord, but motion was definitely present. Improvement in cordal motion was noted on Jan. 25. On April 19, 1962, a colleague was asked to examine this patient, and he confirmed that this patient had entirely normal function of the larynx. She was last seen on Sept. 3, 1964, at which time the only demonstrable lesion was the scar on her neck.

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Council on Family Health Airs Home Safety Tips

Radio stations will participate this fall in a special educational program aimed at reducing home accidents and keeping medicines and other household products out of reach of children.

Safety advice to mothers is being sent to 1,000 radio stations, according to the Council on Family Health which prepared the series of ten interview programs. Some 50 tips on home safety are given by two prominent pediatricians, Dr. Samuel C. Southard of Atlantic City, N.J., and Dr. Allan B. Coleman of Washington, D.C., chairmen, respectively, of the Committee on Accident Prevention and the Subcommittee on Accidental Poisoning, of the American Academy of Pediatrics.

Surgical Treatment of Strokes

LeRoy J. Kleinsasser, M.D.

Early recognition of cerebrovascular insufficiency is imperative to permit appropriate surgical correction, the author declares. He discusses treatment of 96 patients treated surgically for extracranial interference of cerebral blood flow. Definite improvement occurred in 73 and further damage was prevented in 11 patients.

CLINICAL MEDICINE

ALTHOUGH strokes rank third as a cause of death in the United States, of far greater significance is the fact that two million people are now alive in this country who have suffered strokes. Eight out of ten stroke victims survive the acute initial phase of the disease, but many remain in a seriously disabled condition.[1] Since three out of four patients with occlusive stroke have symptoms forewarning of a disabling attack, and three out of four patients with symptoms of stroke have discernible narrowing of the blood supply to the brain, predominantly segmental and extracranial, it is imperative that cerebrovascular insufficiency be recognized early in order that appropriate surgical correction may be made.

TABLE 1.—*Arteries Operated Upon For Stroke (123 Vessels).*

Type of Operation	No. of Patients	Carotid	Vertebral	Innominate	Subclavian
Unilateral carotid	64	64			
Bilateral carotid	16	32			
Carotid-vertebral	9	9	9		
Carotid-innominate	2	2		2	
Subclavian	4				4
Unilateral vertebral	1		1		
	96	107	10	2	4

Clinical Material

Ninety-six patients have been operated upon for extracranial interference of cerebral blood flow (Table 1). One hundred twenty-three vessels have been operated upon; 107 carotid arteries, 10 vertebral arteries, 2 innominate and 4 subclavian arteries. Eight patients were asymptomatic, 61 had intermittent symptoms, 17 had progressing symptoms and ten had completed strokes (Table 2). A unilateral carotid operation was done in 64 patients, bilateral carotid operations in 16, carotid-vertebral operations in nine, carotid-innominate operations in two, subclavian operations in four and unilateral vertebral operations in one patient. There were 63 men and 33 women whose ages ranged from 42 to 88 years. Six were 40 to 49 years old; 31 were 50 to 59; 42 were 60 to 69; 16 were 70 to 79; one was 88 years old. The duration of symptoms varied from a few hours to 10 years.

Type of Stroke

If carotid endarterectomy is to be effective, one of its more desirable applications is to correct the high grade carotid artery ste-

TABLE 2.—*Classification of Strokes.*

Type of Operation	No. of Patients	Asymptomatic	Intermittent	Progressive	Completed
Unilateral carotid	64	3	39	15	7
Bilateral carotid	16	4	9	1	2
Carotid-vertebral	9	1	6	1	1
Carotid-innominate	2		2		
Subclavian	4		4		
Unilateral vertebral	1		1		
	96	8	61	17	10

nosis discovered in the asymptomatic patient on routine physical examination by the presence of a significant systolic murmur over the carotid artery confirmed by arteriography. Eight patients in whom no evidence of cerebrovascular insufficiency was apparent, were operated upon in the so-called prophylactic group for severe carotid artery constriction. Some complained of ear noises described as throbbing, ringing, whishing, and buzzing. On auscultation, a moderately loud or even high-pitched murmur was heard over the common carotid artery bifurcation. No complication or death occurred from this intervention. It is believed that wider application of surgery should be made in this group of patients.

The most satisfying group of patients are those whose problems are of the intermittent type with transient episodes of insufficiency involving the carotid or vertebral-basilar systems. Sixty-one patients with these manifestations improved after operation.

The progressive stroke continues to be the principal source of mortality and disability. In general, it is best to allow the stroke to continue to completion if it is progressing rapidly or has been progressing significantly over a period of hours. Seventeen patients with strokes progressing at varying rates of development were operated upon. Significantly, five of the nine deaths in this series were in patients with progressing strokes. Thus, more than one-half of the deaths occurred in the progressing stroke category, representing more than a 30 percent mortality.

In an earlier review of the problem[2] it was indicated that the patient with the advancing stroke, which occurs in one-sixth of the patients with strokes, represents the most hazardous group to treat operatively. Critical judgment is required in the employment of arteriography, since a progressing stroke may be hastened or precipitated by injudicious timing. If performed during a slowly progressing stroke or one of short duration, the patient should be allowed to recover from the anesthetic sufficiently to allow assessment of any further neurological deficit before surgery is done.

Once the stroke is completed, stabilization of the neurological situation is awaited before surgical correction of the offending lesion is undertaken. This is done hopefully to avoid any further damage rather than to correct the existing neurological deficit.

Arteriography

I prefer to do four-vessel visualization of the cerebral circulation by bilateral percutaneous carotid and retrograde brachial arterial injections of sodium diatrizoate (Hypaque) in 50 percent concentration. This is necessary to evaluate the variations of blood flow and to detect constrictions or occlusions of the carotid or vertebral-basilar systems. Any constriction of 50 percent is significant and will show a pressure gradient. This is done under general anesthesia which facilitates the procedure. No significant complications have occurred. Frequently, retrograde brachial arteriography will demonstrate aortic arch involvement of the innominate or left subclavian arteries. At times a forward angio-aortogram or a retrograde femoral aortogram may be required for visualization of the proximal portions of the innominate, carotid or left subclavian arteries. In any event, accurate mapping of the arterial tree is mandatory and several techniques of arterial visualization may be required in any given case.

The Operation

Temporary intraluminal shunts are utilized during carotid endarterectomy so that deliberate and careful surgery can be done to prevent cerebral ischemia. All carotid operations are staged and bilateral endarterectomy at one operation is avoided to prevent complications. General anesthesia is used exclusively. Carotid sinus activity during the operation producing hypotension or bradycardia is carefully avoided by use of 1 percent lidocaine injection into the region of the carotid bifurcation or appropriate use of atropine to block this effect. Neosynephrine reverses the hypotensive effect promptly. Heparin has been used exclusively at the operating table during arterial occlusion and shunting, and postoperatively where anticoagulants are indicated. It is believed that thrombosis, if it occurs, begins at the operating table due to some technical problem and cannot be prevented by anticoagulants in the postoperative course if such a condition prevails. Every effort is made to maintain patency of the external carotid artery because of the significant collateral flow value of this vessel to the cerebral circulation. Venous autografts, facial or saphenous veins, are used for patch-graft angioplasty in the few instances this is required. Plastic patches have proved troublesome in the presence of infection or they may

lead to aneurysmal formation. Long-term follow-up has disclosed aneurysms involving Dacron patches inserted into the carotid artery after endarterectomy; this requires further surgery under more trying circumstances. One patient developed an aneurysm after two years which was replaced with another Dacron patch, and the aneurysm recurred after another four years requiring saphenous vein graft replacement of the entire circumference of the distal common carotid and proximal internal carotid arteries for correction. Another patient developed a carotid aneurysm after Dacron-patch angioplasty four years later. He delayed his surgery until thrombosis occurred, and surgical correction was unsuccessfully attempted. A special problem has been the patient with occlusion of one internal carotid artery and significant stenosis of the remaining internal carotid requiring surgical correction. This has been met by the use of hypothermia during surgery without untoward incident and with complete protection of the brain. If the external carotid artery is stenotic on the side of an internal carotid artery occlusion, this vessel is endarterectomized with intraluminal shunt protection and autogenous vein patch angioplasty where needed. This is done preceding surgery upon the remaining stenotic internal carotid artery in order to secure as much protection by collateral circulation as possible.

Innominate artery occlusion has been treated by patch-graft angioplasty of the innominate artery through a sternal splitting incision and appropriate carotid endarterectomy, if indicated. The proximal left subclavian lesion has been corrected by endarterectomy during aortic arch exclusion clamping, or by descending thoracic aortic to left subclavian bypass through a limited left third intercostal space thoracotomy.

Discussion

Carotid artery arteriosclerotic disease is the most common source of cerebrovascular insufficiency. It is particularly suitable for surgical correction since it tends to be extracranial segmental and stenotic rather than occlusive. Patients having these lesions may have transient symptoms suggestive of cerebral ischemia with unilateral manifestations. There may be episodes of dizziness or instability, blurring of vision, extremity weakness, numbness, or lack of coordination and deterioration of memory and mental acuity. Clues regarding the presence of extracranial vascular disease are murmurs heard or abnormalities palpated in the arteries. Arteriography will clarify the anatomical situation. Rarely are stenotic lesions of the extracranial portions of the carotid arteries found in the absence of systolic murmurs. These should be distinguished from venous hums. Surgical correction of carotid stenotic lesions has been well established with excellent results. However, rarely can circulation be restored if the carotid artery is occluded for a period of time because of distal thrombosis and obliteration of the internal carotid artery.

Less well defined is the management of patients with acute, progressive neurologic deficit due to extracranial vascular disease. Hunter and associates[3] reported 26 patients who had an acute neurological deficit due to partial or total obstruction of the carotid artery bifurcation and were operated upon during this phase. Results were good in 20; poor in 5 patients. One patient died of cerebral hemorrhage following restoration of cerebral blood flow. They concluded that operation is contraindicated in patients with hemiplegia and that patients with cerebral emboli from ulcerated carotid atheromata are not benefited by operation. It has been preferred to allow these patients to go on to completion of their stroke unless the stroke is progressing slowly or is very recent.

Although the signs and symptoms of carotid insufficiency are clear and tend to be unilateral, this is not true in vertebral-basilar disease. There may be an overlap of symptoms of these two systems, the vertebral involvement tending to be bilateral. Blindness, dimness, or cloudiness throughout the visual fields, double vision, dysarthria, dysphagia, and most notably, vertigo, often with nausea and vomiting, occurs much more frequently in the vertebral-basilar system. If one vertebral artery is found patent on arteriography it is assumed that blood flow to the basilar system is adequate. If both a vertebral and a carotid lesion exist, one should correct the carotid lesion first, since there is so much overlapping of symptoms. This is especially true where the posterior communicating artery is prominent and acts as a satisfactory collateral. Frequently it is possible to correct both lesions during the same operation.

The subclavian steal syndrome has not been encountered frequently. The diagnosis requires a stenotic or occlusive lesion in the

innominate or subclavian artery. The constant physical findings are a significant blood pressure differential between the upper extremities and decreased or absent arterial pulsations in the involved arm. Confirmation is by arteriography, showing reversal of flow to the involved extremity. This favors the siphoning effect which is aggravated by exercise in the affected extremity leading to cerebral ischemia. Correction of this lesion has been by restoration of innominate or subclavian flow.

Aortic arch involvement of the innominate and left subclavian arteries is usually detected by a disparity in blood pressure in the upper extremities accompanied by supraclavicular systolic murmurs. These patients have variable symptoms depending on the extent of interference of cerebral blood flow; opportunity for correction is most favorable.

Whenever there has been a coexistent vascular lesion such as aorto-iliac occlusive disease, the cerebral stenotic lesion is operated upon first unless the coexisting vascular lesion presents an overriding emergency.

Results of Operation

Arterial patency was restored in all but two patients. Seventy-three were improved, eleven were the same, three were significantly worse, and nine died. Thus, 84 of the 96 patients were benefited by this procedure either by improvement or prevention of further damage. Postoperative thrombosis may occur after carotid endarterectomy, and failure of the patient to promptly recover from anesthesia is a definite indication for immediate arteriography and reoperation if occlusion is demonstrated. The dominant side appears to be especially vulnerable and careful attention to detail is required during the operative experience. Four patients were operated upon again for postoperative thrombosis and circulation was restored.

There were nine deaths in this group of patients, five in patients with progressing strokes, three from myocardial infarction, and two related to the operative intervention; one developed an infected carotid patch graft and after several attempts to control bleeding, required ligation of the internal carotid artery followed by stroke and death. One developed an aneurysm of the carotid artery four years after successful surgery and because of delay in returning for surgery presented a remarkably difficult technical problem. An internal carotid artery thrombosis developed postoperatively and was corrected by reoperation but resulted in a fatal stroke.

Summary

1. One hundred twenty-three arteries have been operated upon in 96 patients with definite improvement in 73 and prevention of further damage in 11.

2. The surgical treatment of stroke is definitely indicated in the asymptomatic patient and the patient with temporary ischemia (intermittent).

3. The most hazardous group has been the patients with progressing stroke. Surgical correction preceded by arteriography may be highly beneficial in selected patients having progressing stroke, where the stroke is seen early or is developing slowly. Otherwise, the stroke should be allowed to go on to completion.

4. Patients with completed strokes should be operated upon for stenotic lesions, after stabilization, in order to prevent further damage rather than with the hope that the procedure will cause significant reversal of a stroke, although such a favorable development may occur.

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Marfan's Syndrome With Coarctation and Aneurysms of Aorta

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A report is presented of a case of Marfan's syndrome associated with coarctation of the aorta and pre- and post-stenotic aneursymal dilatation. A more frequent occurrence of coarctation of the aorta with the syndrome has been noted recently.

CASE REPORTS

MARFAN'S SYNDROME, a hereditable disease of connective tissue[1,2] frequently exhibits cardiovascular abnormalities. Aneurysm of the aorta with or without dissection, aneurysm of the sinuses of Valsalva, septal defects,[3,4] and valvular deformities[3,4,5] are the most frequently recognized defects. Others include aneurysmal dilatation of the pulmonary artery with or without dissection, cardiac hypertrophy, and pulmonary hemorrhage. More recently, the association of coarctation of the aorta has been noted,[6] prompting this report.

Case Report

A 58-year-old woman of Mexican lineage was hospitalized in the US Army Hospital at Fort Gordon, Ga., for cardiac evaluation in October, 1963. Surgical correction for a thoracic aortic aneurysm was being contemplated.

The illness began in 1936, with easy fatigability and tiredness. At that time, auscultatory findings were not remarkable. A roentgenogram of the chest revealed an aneurysmal dilatation of the ascending aorta. Physical activity was restricted and her condition remained clinically static until 1948, when she experienced severe excruciating precordial chest pain with syncope. A gradual recovery ensued during which an aortic diastolic murmur was detected for the first time. A similar episode occurred in 1954. A left brachial retrograde aortogram, performed at the Brooke Army General Hospital in 1956, showed a multilocular aneurysm of the ascending aorta, extending from its root to the left subclavian artery, as well as a moderate degree of aortic insufficiency. The lesion was considered inoperable and medical management was advised. The clinical condition remained unchanged until May, 1963, when the patient experienced a "tearing" substernal pain with radiation to both arms, resulting in hypotension and coma. Supportive therapy led to recovery. Studies during this hospitalization again disclosed the presence of the aneurysm and left ventricular hypertrophy. She was discharged on a strict regimen but continued to experience angina and progressive loss of cardiac function. Signs of tracheal and esophageal compression developed, and she was readmitted to the hospital.

The patient had had one pregnancy at age 20, without complications.

Her mother, maternal grandfather, and two maternal aunts had died from ruptured aortic aneurysms, and had no symptoms prior to their sudden deaths.

Physical examination disclosed a tall, slender woman with disproportionately long limbs, "spidery" fingers, and sparse subcutaneous tissue. Blood pressure in the right and left arms was 80/80 and 80/40, respectively, and 120/60 and 110/40 in the right and left lower extremities. The radial pulses were imperceptible. She had worn glasses since childhood. The eyes were "sunken" in a deep orbit. No ectopia lentis was present. The palate was highly arched. The neck veins were distended 5 cm. above the clavicle at 45 degrees and vigorous carotid pulsation was present. There was anterior displacement of the sternum which was expansile in nature and which coincided with a palpable thrill over the left parasternal area. A grade 2, short ejection systolic murmur was followed by a decrescendo, high-pitched diastolic murmur. This was best heard over

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the aortic area and radiated to the left sternal border and apex. An apical protodiastolic murmur was also heard. Basilar, inspiratory rales and tracheal stridor were present. The liver was palpable 3 cm. below the right margin and gave a positive hepatojugular reflux. Femoral pulses were strong and equal. No peripheral edema was present.

Pertinent laboratory studies included a chest roentgenogram which confirmed the presence of an aneurysm of the ascending aorta with calcium deposits within its wall. Generalized cardiac enlargement and pulmonary congestion were also present (Fig. 1). The electrocardiogram showed evidence of left ventricular hypertrophy and nonspecific T-wave changes. The hospital course was characterized by progressive cardiac decompensation, refractory to conventional measures. Acute pulmonary edema preceded the patient's death on the fourth hospital day.

At autopsy, the heart weighed 600 gm. and was hypertrophied and dilated, especially in the left ventricle. Immediately above the aortic valve, a 12 by 9 by 4 cm. aneurysm was present, which compressed and deviated the esophagus, as well as the left main stem bronchus. This aneurysm extended to the ostium of the left subclavian artery, at which point a 1.2 by 0.6 by 0.3 cm. coarctation was present. Immediately beyond the coarctation, the aorta was once again aneurysmally dilated (11 by 9 by 4 cm.). Within each aneurysm was an incomplete, thick fibrous septum. The walls of the aneurysms were involved with grade 2 atherosclerosis (Fig. 2). The ring of a tricuspid aortic valve was slightly dilated. The lumens of the initial 2.5 cm. of the innominate and left subclavian arteries were severely compromised with atherosclerotic plaques and thrombi.

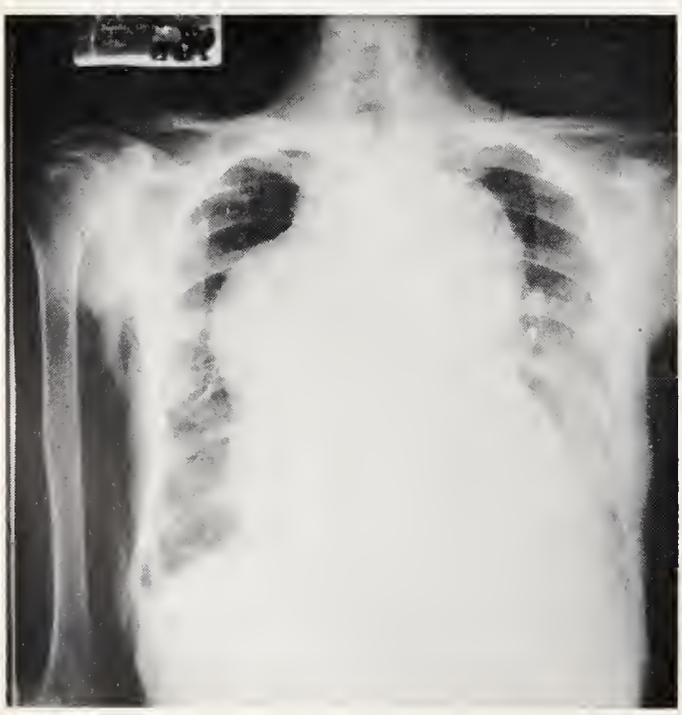

Fig. 1. Chest rodiograph showing generalized cardiac enlargement with pre-stenotic and post-stenotic aneurysm of the thorocic aorto. Calcification within the walls of the oneurysm is evident. The left main stem bronchus is displaced to the right and indented by the aneurysm. Pulmonary congestion is also present.

Microscopic examination disclosed fragmentation of the elastica and medial necrosis of the walls of the aneurysms. The media was split, the outer portion of which remained in the sac wall, while the inner portion contributed to the incomplete septa. Other findings included pulmonary edema and fibrosis, chronic pyelonephritis, a unilocular renal cyst, and a small psammomatous meningioma.

Discussion

This patient presented many of the features associated with Marfan's syndrome. The cardiovascular abnormalities of coarctation of the aorta, pre- and post-stenotic aneurysmal dilatation and aortic valvular insufficiency further supported the clinical diagnosis. The absence of ectopia lentis, the "ocular hallmark" of the entity in otherwise classic cases of Marfan's syndrome have been described before.[1,6] Repetitive aortic dissections which, perhaps, were also complicated by acute aortic insufficiency "crises"[12] may have accounted for the occurrence of the several episodes of cardiovascular collapse experienced by the patient. The presence of pre- and post-stenotic aneurysms in an otherwise uncomplicated coarctation of the aorta is infrequent.[7] The premortem diagnosis of coarctation of the aorta was not anticipated in view of the strong femoral pulses and negative angiocardiographic studies. This has also been the experience of others.[6] Augmentation of the femoral pulses in the pre-

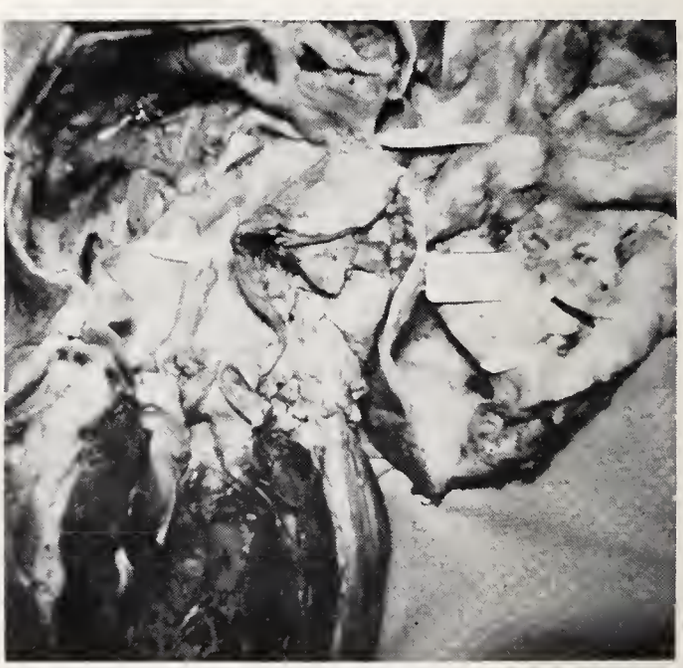

Fig. 2. Close-up view of the sagittol section of the heart and oortic aneurysm showing massive hypertrophy of the left ventricular wall. The site of the oortic coorctotion is marked by a white arrow. Pre-coarctation ond post-coorctation are also seen. Pseudo-septa and arteriosclerotic changes within the wall of the aneurysm are olso evident.

sence of long-standing coarctation of the aorta may result from either increased collateral circulation to the lower aortic segment or by a not very well understood hemodynamic phenomenon associated with aortic insufficiency. Under the circumstances, palpation of the dorsalis pedis pulses may be a more reliable sign in the detection of decreased perfusion to the lower extremities. Also, normotensive and hypotensive blood pressures may occasionally be recorded in the upper limbs, perhaps, secondary to compromise of the ostia and/or lumens of the subclavian arteries by either the dissection or the aneurysm.

There is a higher incidence of coarctation of the aorta associated with Marfan's syndrome (1:42)[6] than in the general population (1:3,000). The presence of coarctation of the aorta in Marfan's syndrome further increases the possibility of dissection[8,9,10] as would be expected since both conditions are predisposing factors for the development of aortic dissection.

Summary

A case of Marfan's syndrome with coarctation of the aorta and associated pre- and post-stenotic aneurysmal dilatation has been presented and discussed. This is the 13th known recording of similar cases.[6]

Addendum

Subsequent to this case report, another case of Marfan's syndrome with aneurysm and coarctation of the aorta has been described in a series of five cases of aneurysm of the aorta studied by Wheat and associates.[11] This brings to 14 the total number of known cases presenting this cardiovascular-anatomic complex.

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HEW Announces New Fund Regulations

New regulations permitting the use of Hill-Burton funds for constructing, expanding, or modernizing psychiatric units in general hospitals and certain other mental hospital facilities stipulate that Hill-Burton funds may be used for psychiatric beds or units in general hospitals only if the state's allotment under the Community Mental Health Centers Construction Act (P.L. 88-164) has been exhausted. This restriction stems from the "nonduplication provision" included in P.L. 88-164. In addition, any psychiatric units of general hospitals approved for Hill-Burton funds must conform to the state plan approved under the Community Mental Health Centers Program.

Thrombotic Thrombocytopenic Purpura: Temporary Remission Induced by Prednisone

CASE REPORTS

IN 1925, Moschowitz[1] first described the triad of hemolytic anemia, thrombocytopenic purpura, and bizarre central nervous system changes. He noted the postmortem pathologic vascular changes which are now accepted as characteristic of thrombotic thrombocytopenic purpura (TTP). This disease has been more frequently recognized and its scope of definition expanded into a quintad with the addition of fever and renal abnormalities, as outlined by Lukes and coworkers.[2]

Major problems associated with this disorder include the difficulty in securing adequate tissue for an antemortem tissue diagnosis and the lack of any consistently effective therapy. The following is a report of a patient with thrombotic thrombocytopenic purpura in whom the diagnosis was made by histologic examination of a needle biopsy (Vim Silverman) of the bone marrow, and who experienced a complete remission when treated with large doses of steroids and mercaptopurine.

Case History

The patient, a 46-year-old Negro longshoreman, was well until Dec. 13, 1963 when he first noticed malaise, fatigue, and dry cough unassociated with any pain, chills or fever. He consulted his private physician who told him that he was anemic and prescribed a seven-day course of unidentified medication which the patient took with no apparent improvement. During late December, generalized weakness ensued. On Jan. 1, 1964, gingival pain and bleeding developed. Within the next 24 hours his weakness intensified and he became disoriented. He was admitted to the Galveston County Hospital on Jan. 2. At that time he was semicomatose.

Laboratory studies revealed a leukocyte count of 8,200 per cu. mm., packed cell volume of 20 percent, hemoglobin of 6.2 gm. per 100 ml., bleeding time of 16 minutes, and Lee-White clotting time of 6 minutes and 40 seconds. A peripheral blood smear examined by a hematologist on Jan. 5, 1964, showed anisocytosis, poikilocytosis, and thrombocytopenia. Examination of the bone marrow revealed erythroid hyperplasia and a large number of megakaryocytes.

At that time treatment was started with aqueous penicillin, 200,000 units intramuscularly, every four hours. The patient received transfusion of five units of fresh whole blood, and begun on dexamethasone, 9 mg. daily, but failed to improve. He remained in Galveston County Hospital until Jan. 13, 1964, during which time his white blood count remained normal and the thrombocytopenia persisted. The patient was transferred to the Houston Veterans Administration Hospital on Jan. 13, 1964. Admission physical examination revealed a stuporous, muscular, Negro man with a supine blood pressure of 130/82 mm. of mercury, pulse 100, and normal respirations. The rectal temperature was 99.0F. The pupils were equal in size and reacted to light. The eyes appeared to be deviated to the left and a slight horizontal nystagmus with the fast component to the left was present. There was no jugular venous distention. Ecchymoses were noted in the buccal mucosa, and old blood was present in the mouth. The liver edge was palpated one fingerbreadth below the right costal margin. Several large ecchymotic areas were present over both arms. Rectal examination revealed feces which reacted weakly positive to guaiac testing. Neurologic examination revealed weakness of the right side of the face, right arm, and right leg, but no flaccidity or hypertonia. Deep tendon reflexes on the right side were less brisk than on the left. There was a slight, brief clonus upon testing the Achilles reflex of the right ankle.

Results of laboratory studies were as follows: Hemoglobin, 8.5 gm. per 100 ml. packed cell volume, 22 percent; leukocytes, 14,350 per cu. mm., with differential of 60 percent mature neutrophils, 1 percent metamyelocytes, 2 percent myelocytes, 27 percent lymphocytes, 6 percent monocytes, and 3 percent eosinophils. The platelet count by a direct technique was 46,000 per cu. mm. The erythrocyte sedimentation rate (Wintrobe) was 45 mm. per hour. On Jan. 14, 1964, the total bilirubin was 1.9 mg. per 100 ml.; serum electrolytes were normal and a urinalysis revealed proteinuria (2+) with 20 erythrocytes and 8 to 10 leukocytes per high power field. A stool guaiac test was trace positive. Examination of a peripheral blood smear (Fig. 1) revealed anisocytosis and poikilocytosis; many of the erythrocytes were bizarre in shape. Helmet cells and schistocytes were noted. A polymorphonuclear leukocytosis was present. A paucity of platelets was noted. Examination of the sternal bone marrow revealed erythroid hyperplasia. Myelopoiesis was normal. Numerous

In a case of thrombotic thrombocytopenic purpura, confirmed by Vim Silverman needle biopsy, a temporary remission associated with administration of large doses of corticosteroids and mercaptopurine was noted. The patient developed hepatitis while receiving mercaptopurine; the condition resolved rapidly after the drug was discontinued.

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megakaryocytes were present. On Jan. 14, a Vim Silverman needle biopsy of the iliac crest bone marrow was performed. Examination of the paraffin sections three days later revealed numerous blood vessel changes consistent with thrombotic thrombocytopenic purpura (Fig. 2).

Because of the association of hemolytic anemia with thrombocytopenia and bizarre central nervous system disease, a diagnosis of probable thrombotic thrombocytopenic purpura was made. Methylprednisolone, 10 mg. every six hours by intramuscular injection, was prescribed. On Jan. 16, the packed cell volume was 30 percent and the reticulocyte count was 7.7 percent. On Jan. 21, the corticosteroid regimen was changed to prednisone, 50 mg. every six hours by the same route. Serial determinations of the platelet counts and the packed cell volume were made (Fig. 3). On the day that prednisone was instituted at 200 mg. per day, the platelet count was 34,000 per cu. mm. Within two days, the platelets increased to 104,000 per cu. mm. The packed cell volume prior to the increase in the steroid dosage was 20 percent. This increased rapidly and within 10 days (Jan. 31) was 41 percent.

Mercaptopurine, 50 mg. by mouth every six hours, had been added to the treatment program on Jan. 25. The patient's mental status did not change appreciably until Jan. 30, but between this date and Feb. 4, his confusion cleared. Neurological examination on this latter date showed him to be mentally alert, to have normal deep tendon reflexes, and no pathologic reflexes. Prednisone was continued in the same dose until Jan. 30, after which the dose was gradually reduced (Fig. 3).

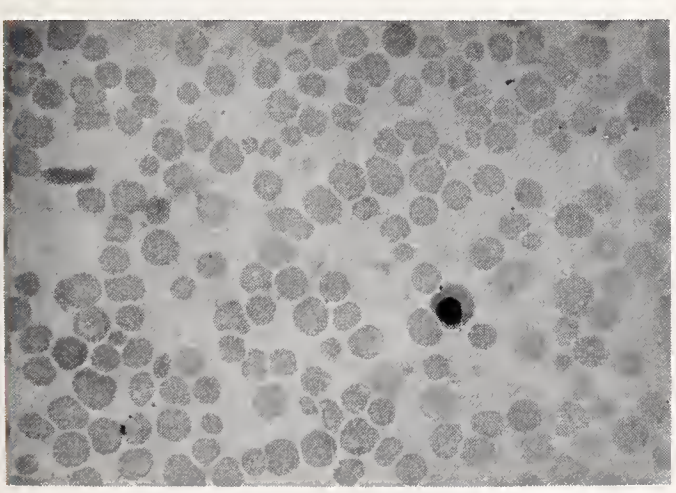

Fig. 1. Peripheral blood smear at time of admission.

On Feb. 10, tenderness developed in the right upper quadrant. The liver was enlarged and tender, and extended 5 cm. below the right costal margin in the midclavicular line, and mild scleral icterus was also noted. Stools were of normal color and the

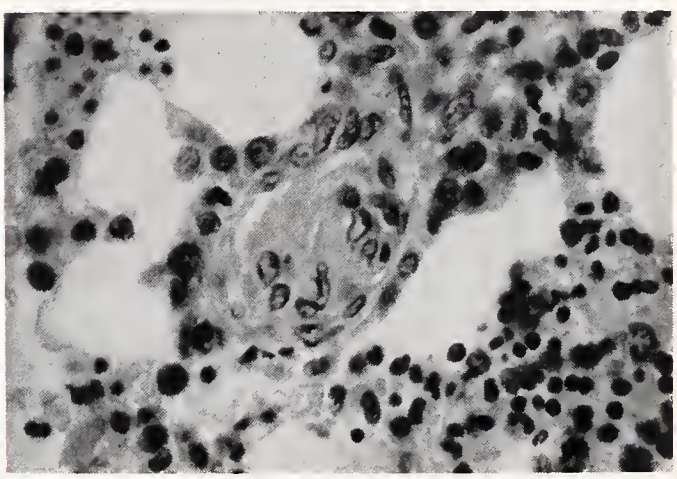

Fig. 2. Bone marrow biopsy obtained prior to treatment.

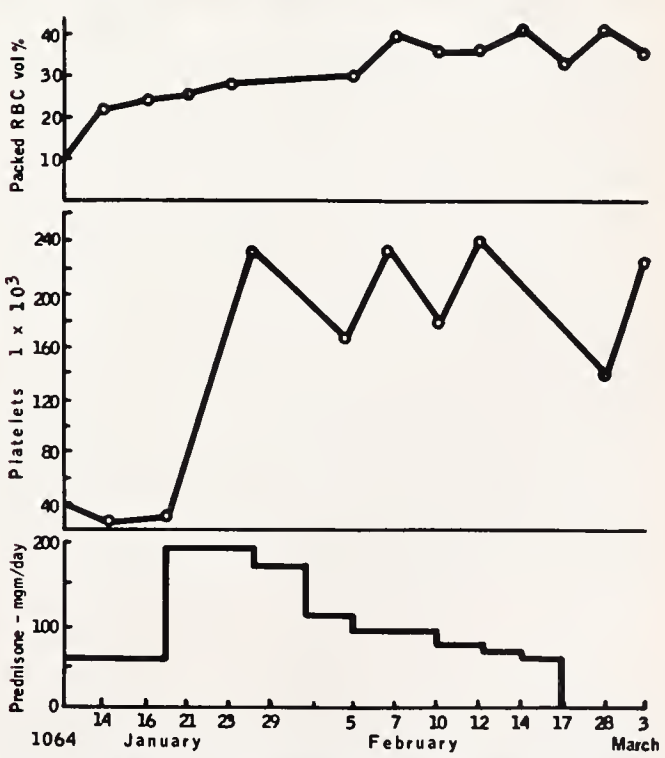

Fig. 3. Comparison of platelet count, reticulocyte count and packed red cell volume during hospital course.

urine was moderately positive for bilirubin. Laboratory tests revealed a serum glutamic pyruvic transaminase of 200 units (normal, less than 40 units) and total bilirubin 3.7 mg. per 100 ml., of which 1.4 mg. per 100 ml. was direct reacting. The alkaline phosphatase was 4.6 Bodansky units. A cephalin cholesterol flocculation test was negative and the thymol turbidity was 1 unit. The hemoglobin, packed cell volume and reticulocyte determinations were within normal limits.

The patient had no history of jaundice prior to this hospital admission. He had received 2,500 ml. of blood in the month preceding onset of his liver disease. He was, in addition, continuing to receive mercaptopurine, a drug previously reported as causing hepatitis in subjects with acute leukemia.[3,4] The mercaptopurine was discontinued on Feb. 12. By Feb. 21, the right upper quadrant distress had disappeared as had the icterus and hepatomegaly. The serum bilirubin gradually decreased and on March 2, the total serum bilirubin was 0.8 mg. per 100 ml. with no direct reacting fraction. The cephalin cholesterol flocculation and thymol turbidity tests remained within normal limits.

On Feb. 25, the patient began to complain of numbness involving the digits of the left hand with associated localized weakness. These findings persisted, and four days later were followed by development of recurrent grand mal seizures and a left hemiparesis. Therapy with phenobarbital and diphenylhydantoin was instituted but failed to control the seizures. On March 2, mercaptopurine was reinstituted at 200 mg. per day. On March 3, the leukocyte count was 8,700 per 100 ml. with packed cell volume, 45 percent; platelet count, 112,000 per cu. ml.; and normal electrolyte determinations. On March 4, the patient died suddenly.

At autopsy the most prominent findings were in the brain. There was an organized thrombus at the junction of the superior longitudinal sinus and the transverse sinus. The gyri over the right cerebral hemisphere were flattened. Encephalomalacia of the mammillary bodies and right cerebral hemisphere was observed on the cut section. On microscopic examination an occasional perivascular lymphocytic infiltration was seen. The lung parenchyma was noted to bulge on cut section and marked consolidation with associated hemorrhage was noted in the dependent portions. The liver weighed 1,960 gm.; the surface was of normal color and was smooth. On microscopic examination, the only abnormal findings were a small amount of brown granular pigment in the Kupffer cells and sinusoidal engorgement with erythrocytes. Gross and microscopic examination of the spleen, lymph nodes, kidneys and bone marrow revealed no significant abnormalities. Specifically, small blood vessels in each of these organs were noted to be normal.

Discussion

Three features of this case warrant discussion: (1) diagnosis by Vim Silverman needle biopsy of the iliac crest bone marrow, (2) a complete remission associated with the administration of corticosteroids and mercaptopurine, and (3) hepatic disease developing during the period of mercaptopurine administration and subsiding after its withdrawal.

Moschowitz[1] in 1925 noted postmortem pathologic changes in a case which is now designated as thrombotic thrombocytopenic purpura. In 1952, Cooper and associates[5] demonstrated that the characteristic hyaline thrombosis and arteriolar fusiform aneurysmal dilatation of thrombotic thrombocytopenic purpura were present in the small blood vessels of marrow particles acquired by aspiration. Prior to this study, histologic diagnosis of this disease had rarely been made before death. Ruffolo and associates[6] have subsequently found these classical changes in particles of marrow from six to eight subjects with this disorder. Other investigators[7] have been unable to confirm these studies, possibly because of the difficulty in collection and preparation of the marrow particles. McFarland and Dameshek[8] more recently have shown that good biopsies of marrow can be obtained with a Vim Silverman needle, and others[9] have demonstrated the value of such biopsies in the diagnosis of disorders involving the bone marrow.

Each specimen obtained in the case reported here had several vessels present and even serial sections of some of these vessels were available. Because of the frequent involvement in the bone marrow in thrombotic thrombocytopenic purpura, an attempt at diagnosis by means of Vim Silverman needle biopsy is indicated in all suspected cases.

Cahalane and Horne,[10] in a review of 116 cases, noted that 98 patients succumbed to a rapid febrile illness within weeks. However, Lukes and others[2] have expanded the disease spectrum by reporting that patients may demonstrate a chronic form of thrombotic thrombocytopenic purpura with intermittent signs and symptoms occurring for many years prior to the development of "acute" terminal disease. Our patient was clearly affected by the acute form of this disease and, accordingly, most unlikely to experience spontaneous remission.

Our patient had a complete remission which was temporally related to an increase in prednisone dosage from 70 mg. to 200 mg. per day. Burke and Hartman[11] had a similar experience with one of their cases. In this instance, a complete remission occurred two

days following a change in drug program from corticotropin, 80 mg. per day, to prednisone 100 mg. per day. These authors observed remissions in two other cases of thrombotic thrombocytopenic purpura treated with high doses of prednisone. Hill and Loeb[12] first described beneficial effects from large doses of steroids, but each of their subjects had a splenectomy in addition to steroid therapy. Steroids have been used in at least 39 reported cases, but remissions have been described in only 6 cases.[11-16] We have been unable to relate the development of a remission to the dose of steroid employed.

Multiple other modes have been employed in the therapy of thrombotic thrombocytopenic purpura, but remissions have been noted only in patients receiving heparin (one case) and splenectomy. The tendency for temporary spontaneous remission in the chronic form of this disease complicates any interpretation of therapeutic response, but steroids in large doses seem to be the drugs of choice in most patients with thrombotic thrombocytopenic purpura.

Therapy with mercaptopurine was chosen because of the proposed autoimmune nature of thrombotic thrombocytopenic purpura.[17] No statement as to the efficacy of this medication in this case can be made, however, as the patient was improving prior to the initiation of mercaptopurine therapy.

Hepatic disease associated with mercaptopurine administration has been noted in patients with acute leukemia.[3,4,18] Right upper quadrant tenderness has been described by Clark and associates[3] as the first evidence of liver disease. Boggs and others[18] have described a cholangiolitic type of hepatitis. Clark and others[3] reported two cases which developed centrolobular necrosis and bloody diarrhea. Boggs and others[18] noted the complete disappearance of evidence of liver disease after the drug was withdrawn. They also demonstrated that the drug could be reinstituted in some cases of acute leukemia without recurrent hepatitis. Our patient had right upper quadrant tenderness as his first manifestation of liver disease. The laboratory studies showed evidence of parenchymal disease as demonstrated by the elevation of serum glutamic pyruvic transaminase, but the clinical and laboratory manifestation of hepatitis cleared rapidly after discontinuance of mercaptopurine. No hepatic biopsy was obtained because of the prolonged prothrombin time during the period of hepatic disease. The postmortem liver specimen was unremarkable. To our knowledge, this is the first reported case of hepatitis related to mercaptopurine in a patient without leukemia.

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Hippocampectomy for Relief Of Intractable Pain

RESEARCH & INVESTIGATION

THE HYPOTHALAMUS, the anterior thalamic nuclei, the gyrus cinguli, the hippocampus, and their connections were believed by Papez[1] to constitute a harmonious mechanism which may elaborate the functions of central emotion as well as participate in emotional expression. A more wide-ranging concept was expressed by Herrick,[2] who considered the limbic system an activator of all neocortical functions, facilitating or inhibiting not only affective behavior, but memory and learning as well. Although the specific influences exerted individually by the various elements of the limbic system have not been clearly defined, it is probable that the limbic system, the predecessor of the more recent neocortical structures, modulates all functions of the central nervous system and is deeply concerned in, or controls, the reactions of the organism where survival of the individual and the species is concerned.

Many authors regard the hippocampus itself as the "hub" of the limbic system. Papez speaks of the "central emotive process of cortical origin" being "built up" in the hippocampal formation before relay to the mammillary bodies and the anterior nuclei of the thalamus. Grunthal[3] suggests that the hippocampus acts as a "catalytic activator" essential for the central affective and neocortical activity.

To gain a greater insight into the function of the hippocampus, a technique for subtotal hippocampectomy in the cat and monkey was developed.[4,5]

Following hippocampectomy, a taming effect was observed, similar to that described by Kluver and Bucy[6] and Schreiner and Kling.[7]

In addition, it was noted that the animals responded much less vigorously to pain stimuli than before hippocampectomy, though whether this was due to diminished perception or reactivity is still undetermined.

The laboratory findings provided an encouraging basis for further exploration of the effects of hippocampectomy on a clinical level. It appeared that the procedure might prove useful in psychosurgery and pain control. The transfer of a laboratory procedure to the clinical level is fraught with many unexpected pitfalls and it was decided therefore to use the procedure only for intractable pain due to widespread carcinomatosis, or pain states which had not responded to routine neurosurgical pain relieving procedures. For two years, this careful selection produced seven cases and thus the evaluation of hippocampectomy, on the clinical level, can only be considered to be in a preliminary stage, and the observations are presented more as a study of results than a proposal of a new technique with definite therapeutic indications.

Technique of Hippocampectomy

The technique of the procedure for clinical trial was adapted from the methods previously developed in the laboratory. Initially a series of twin-bladed retractors were made (Fig. 1). These were inserted into the middle part of the temporal lobe, between the su-

Fig. 1. Retractors for transventricular hippocampectomy.

Hippocampectomy can relieve pain, but because of the few cases in which it has been tried, clinical evaluation still is in a preliminary stage. A study of results in seven cases is presented.

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perior and middle gyri, just in front of the vein of Labbé. Through this transventricular approach, the hippocampus was removed by suction, using suction tips of varying shapes. The photographs illustrate the extent of the lesion. The transventricular approach in the human subject proved to be far less satisfactory than in the animal, and the illustrations show (1) the incompleteness of the resection, and (2) fairly extensive damage to the overlying neocortex of the temporal lobe (Figs. 2 and 3).

Hippocampectomy was next attempted by means of lesions produced by coagulation, using stereotaxic approaches through the posterior parietal lobe and the trigone of the ventricle, with the electrode running along the medial wall of the temporal horn in the hippocampus to the tip of the temporal horn. Multiple small coagulation lesions were made at 1 cm. intervals. The results of this procedure can be seen in the autopsy photographs.

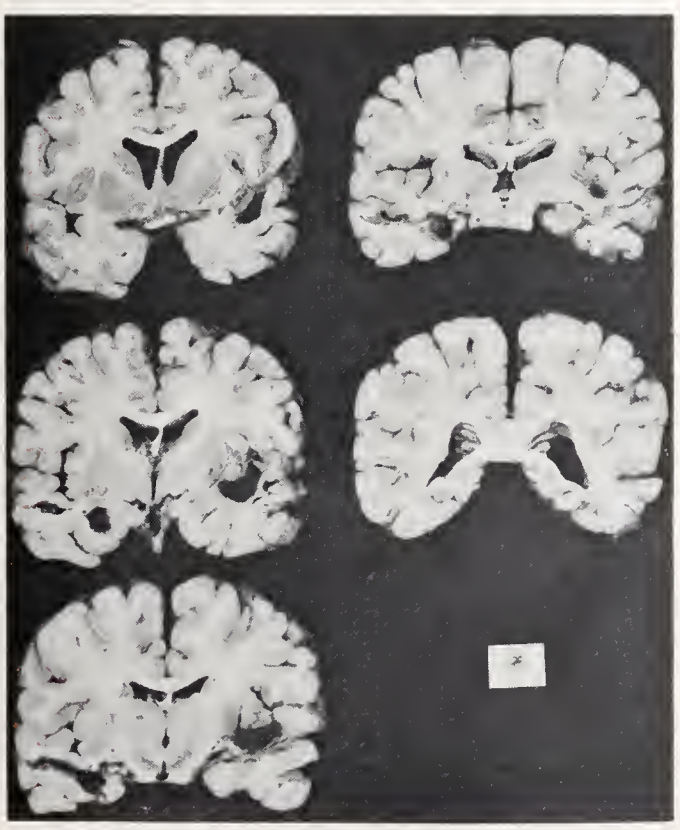

Fig. 2.

Case Histories

Case 1.—A man, age 27, was admitted to VA Hospital, Houston, Dec. 6, 1962, with a diagnosis of Hodgkins' disease, based on a biopsy in 1954. In

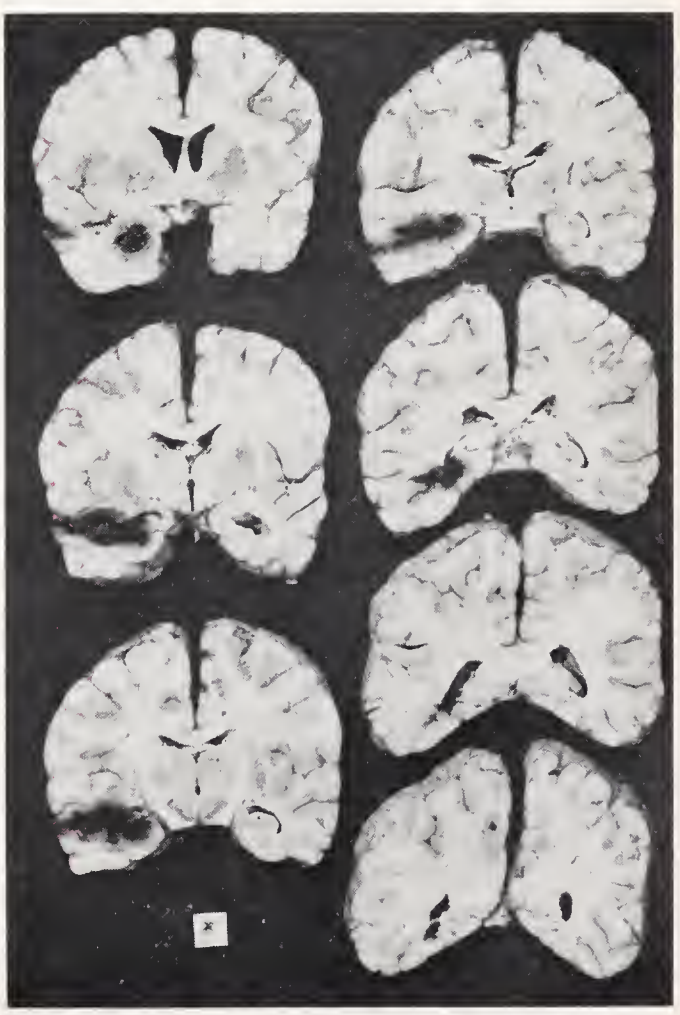

Fig. 3.

1962, bony lesions were found in the right ilium, lumbar spine and left femur. The lesions were treated with irradiation and vincristine. A week prior to his admission he developed increasingly severe lumbar pains, paraparesis, dysuria and constipation.

On examination he was found to have widespread adenopathy, diffuse tenderness of the spine from T-11 to the sacrum, severe flaccid paraparesis and a sensory level at T-11, with hypesthesia below this level. X-rays demonstrated multiple bony lesions of the lower thoracic, all lumbar and sacral vertebrae and the pelvis. Myelographic blocks were present at T-10 and L-2.

A decompressive laminectomy was performed at the above sites without any significant improvement. Following decompression, morphine, 1/4 grain, was required every two hours to control pain due to spinal and pelvic lesions.

No psychological testing was possible in this ill, emaciated and drowsy patient who only woke up sufficiently to ask for more medication. Grossly he appeared to be fairly well oriented though semi-illiterate and dull.

On Dec. 11, a right hippocampectomy was performed through a one-inch incision between the upper and middle temporal gyri, just in front of the vein of Labbé, removing an estimated 80 to 90 percent of the right hippocampus.

Postoperatively all narcotics were discontinued. Mild disorientation was noted in the first postoperative week, but this cleared completely. During all this time the patient never complained of pain and showed a remarkable unconcern for painful stimuli.

By Dec. 24, the patient had some return of pain but this was controlled by one Darvon every four hours and he was able to get up in a wheel chair. Psychological testing on Dec. 27 showed an IQ of 72, no disorientation, a slight memory impairment and signs of indifference.

The patient remained at home and did not come and he remained at home for approximately two months, by which time the pain had become too severe again to be controlled by Darvon.

On Feb. 21, a left hippocampectomy was performed in the same fashion and extent as before, and again all analgesic medication was withdrawn. After three weeks he was discharged home in a wheel chair with a supply of Darvon tablets which controlled satisfactorily the residual discomfort in his spine. The last psychological evaluation at the time of discharge showed a Wechsler and verbal IQ of 66 and comparable performance on performance subtests. The patient was fairly well oriented in time and space. He knew where his home was but could not remember the house number. He knew the year but not the date, and he could not name the president.

The patient remained at home and did not come to the clinic for follow-up. He died at home on June 9, and no autopsy was obtained.

Case 2.—A man, age 73, was admitted to VA Hospital, Houston, Dec. 31. In April 1963, this patient was discovered to have carcinoma of the prostate with metastases to the spine and pelvis. In July, 1963, a decompressive laminectomy of T-5 was performed. Shortly afterwards a right mid-thigh amputation was performed for gangrene of the foot due to vascular insufficiency. This operation was followed by severe pains in the stump.

On examination the patient was found to have metastases in his right femur, pelvis, lumbar and thoracic spine and one scapula.

In the week prior to hippocampectomy, the patient's medication had to be changed from Demerol, 75 mg., and Phenergan, 25 mg., to morphine, 1/8 grain, four to six times a day.

On Jan. 20, 1964, a series of lesions were made stereotactically, by means of a leucotome, throughout the length of the left hippocampus; this was followed by a series of coagulative lesions at 1 cm. intervals in the same area. Fig. 4 shows the extent of the lesion. Fig. 5 shows placement of the electrode in relation to the temporal horn which was outlined with Pantopaque.

Following the operation the patient was dysphasic and would speak only in Spanish, although prior to hippocampectomy he spoke fairly fluent English. He complained of no pain and all narcotics were stopped. By Jan. 28, he was up in a wheel chair responding to simple commands, talking a mixture of Spanish and English. By Feb. 10, he was alert, active, and never complained of pain until Feb. 15, when he suddenly became unresponsive and without a palpable pulse. He died soon afterward.

Although a preoperative psychological evaluation was performed, the patient died before a postoperative performance plateau was attained and psychological data are therefore incomplete.

Autopsy showed no definite immediate cause of death, only widespread carcinomatous deposits in an old and emaciated individual; there was cerebral atrophy. Serial sections showed disruption and degeneration in the left hippocampus and the loss of bulk is clearly evident when the left and right hippo-

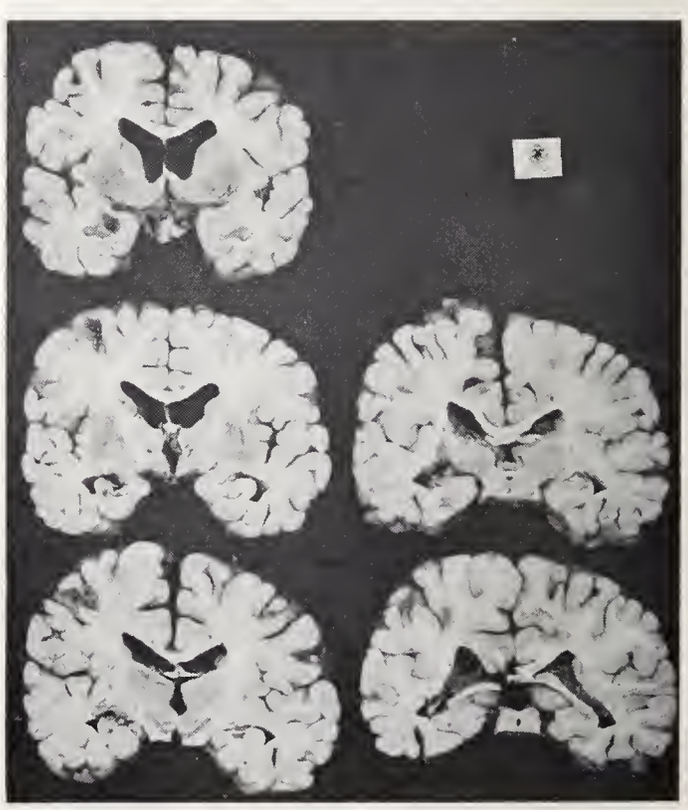

Fig. 4.

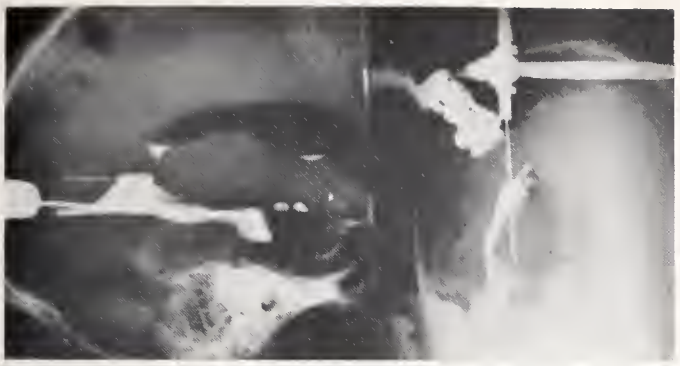

Fig. 5. Pantopaque in the temporal horn shows outline of hippocampus with electrode in situ.

campus is compared. The microscopic estimate of tissue destroyed was 50-60 percent of the left hippocampus.

Although some extension of the lesion beyond the hippocampus can be seen, the volume of extrahippocampal tissue affected is quite small.

The case histories of five other patients showed similar features. Satisfactory pain relief until death (up to two months) was achieved in three further cases with bilateral hippocampectomy. Partial or temporary relief was attained in the remaining two patients with unilateral hippocampectomy. All patients showed a relative unconcern for pain.

Discussion

No mention has been made in the literature about the function of the hippocampal formation in the perception of pain and therefore great interest attaches to the finding that destruction of even part of the hippocampal masses can control intractable pain. If we accept the theory that the limbic system is concerned with the preservation of the individual and species, then it will not be surprising to find that destruction of any part of the limbic system will affect these prime goals of the system. Pain can be considered as perhaps the most important warning signal to the organism that its well-being and existence are threatened. Although no cortical projection areas for pain sensation have ever been described, it appears not impossible that after traversing the thalamus, the first cortical awareness of pain is at the level of the paleocortex of the limbic system. Disruption of the function of the hippocampus could then explain the apparent lack of awareness of the patients of their pain.

The relation of the hippocampus to memory recording, or more exactly to acquisition and retention, requires further elucidation. At the present time, considerable evidence has been presented by Penfield, Scoville and Milner[8,9] that the hippocampal formation and the hippocampal gyrus play a large and essential part in the recording of recent experiences. However, it can be shown that "memory recording" cannot be assigned so simply to one part of the central nervous system. In Wernicke's syndrome a severe memory defect is present yet no hippocampal or temporal lobe lesions can be found, most of the changes being noted in the diencephalon and the hypothalamus.

In our cases, the memory deficit varied with the size of the lesion and appeared to be more influenced by the neocortical than the paleocortical lesions; however, which of these definitely played a major part in the memory deficit cannot yet be stated with certainty. Certainly, bilaterally extensive hippocampal lesions did not produce the extreme memory deficits described by Penfield, Scoville and Milner.[8,9]

Whether hippocampectomy will play a role in pain control surgery is not clear at this time. The procedure is admittedly complex and evaluation of the behavioral effects and defects is not easy. The material remains restricted by humanitarian considerations and much further work lies ahead. Cryogenic methods with stereotaxic procedures will be explored and it is hoped that a further clarification, not only of the value of the procedure in pain control and psychosurgery will be obtained, but also a more detailed understanding of the fascinating activities of the limbic system as a whole.

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Trends in Anesthesia Coverage in Texas

REVIEW ARTICLES

MANY EVENTS in recent months have prompted anesthesiologists to do an introspective analysis of their specialty. The attempts of legislatures to brand anesthesiology as a "hospital-based specialty" and, as such, to impose upon anesthesiologists the controls associated with hospital medicine was one such event. Other events, perhaps more damning, were the release of the Philadelphia report and the appearance of articles such as "Anesthesiology Just Wasn't for Me."[1] Still other events such as the departure of fully trained Texas anesthesiologists to residencies in other fields have caused many of us to pause to examine our image. We have attempted to examine both retrospectively and projectively the situation in Texas.

Certified Registered Nurse Anesthetists

The American Association of Nurse Anesthetists requires formal training in anesthesia and examination for certification and membership. The group's original requirements were established in 1931 and schools are approved by the parent organization. The total number of schools in 1965 was 172, each filled to capacity, a total of 1,026 students; Texas had three civilian schools and three military schools. Texas, under the guidance of Miss Dorothy M. Hoadley, in 1935 became a component member of the national organization. Continued improvement of these schools has been evident in the product.

Presented to the Texas Society of Anesthesiologists on April 17, 1966, during the Annual Session of the Texas Medical Association.

Table 1 indicates the increasing time of training required beyond the RN diploma for nurse anesthetists. Completely to the credit of pressure from within their own group, great strides in curriculum improvement and case requirements for nurse anesthetists have been instituted to parallel the increased time requirements. The qualifying examination given candidates for certification as registered nurse anesthetists (CRNA) at this time would put stress on most practicing anesthesiologists and even the well-informed resident or newly graduated anesthesiologist. When we examine the number of CRNA's in Texas as compared to the national membership, we observe parallel curves indicating that the Texas ratio of CRNA's is following the national average very well when only active members are considered. The number of nurses cited represents only actively practicing nurse anesthetists in Texas whereas the national total number of nurse anesthetists is presented. One factor we feel should contribute greatly to potential stabilization of available nurse anesthetists is the greater number of males who have become CRNA's in recent years. Without the handicaps associated with obstetrical leave, inactivity while husbands move to new jobs, or the need for a baby sitter for the nurse to work, job stability and dependability are likely to be greater. The

TABLE 1.—*Required Length of Training for CRNA's.*

1933-1939	4 months
1939-1947	6 months
1947-1951	8 months
1951-1963	12 months
1963-Present	18 months

A survey of the availability of qualified anesthetists—nurse and M.D.— in Texas is presented. Figures are compared to national statistics.

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geography and population distribution of our state will favor in the future even greater use of male nurse anesthetists in Texas as they become available.

Anesthesiologists in Texas

While membership in the Texas Society of Anesthesiologists (TSA) does not require training or proof of competence in anesthesiology, neither does lack of membership in the TSA signify lack of training or incompetence in the practice of anesthesiology. We feel safe to assume that membership indicates interest in the practice of competent anesthesia. Probably few physicians in Texas who devote as much as 50 percent or more of their practice to anesthesia or list anesthesia as their primary specialty with the AMA are not members of the Texas Society of Anesthesiologists. Therefore membership signifies a competent anesthesiologist. Nationally, Texas is seventh in total number of anesthesiologists and one out of 31 physicians in Texas is an anesthesiologist (349 out of 10,889) which is one point better than the national average (1 out of 32).[2] We offer training in Texas for 90 residents in 11 programs in anesthesiology, whereas nationally there are 213 programs offering 1,818 positions.[4] The number of anesthesiologists in Texas has at least kept pace with or slightly exceeded the national growth of anesthesiologists. Our only indication of data below national averages is in the number of residents in training in anesthesiology. Nationally, the resident programs are 68 percent filled (1965) while in Texas only 57 percent of residencies available are filled.

Texas Anesthesia Supply and Demand

From the preceding analysis of personnel data and projections of parallels to national averages, Texas, statistically projected, will follow national averages. From these projections and application of previously published data[5] we may conclude Texas anesthesia coverage breaks down reasonably close to the following statistics. A breakdown of surgical coverage in Texas indicates that approximately 15 percent of the anesthesia is administered by persons of unknown qualifications. Physicians (not anesthesiologists) provide 11 percent, R.N.'s 3 percent, and, in some cases, laboratory technicians or even, in one case, Red Cross volunteers are used to give anesthesia making up 1 percent of the total anesthesia given in this "unknown" category. In smaller hospitals, the situation is not this good, for example, in the hospital of 0-49 beds, approximately 60 percent of the anesthetics are given by persons of unknown qualifications, often by the operating surgeon or anyone who can be spared at the moment including even the lay hospital administrator. The possession of an M.D.'s license is definitely not a qualification for competency in administering anesthesia. As bad as this may appear on the surface, these figures represent only a small percentage of the total, and unquestionably the situation is far better in Texas than in many other areas.

The problem of obstetrical anesthesia coverage in Texas, however, is another story. Of the 209,908 babies born in 1963 in Texas hospitals, 120,080, or 53 percent, were born in hospitals having more than 1,000 deliveries per year representing 51 Texas hospitals.

All of these hospitals have more than 250 beds and from these figures we can apply the projection that 57 percent of these anesthetics were given by members of TANA whereas only 19 percent were given by TSA members. R. N.'s without other qualifications gave 9 percent of these anesthetics and persons who were neither nurse nor doctor gave 2 percent. When we get down to the hospital of 0-49 beds that provides for obstetrical deliveries, only 37 percent of the obstetrical coverage is by either TSA or TANA members. While these figures are probably not completely accurate, they give an indication of the extent of coverage of anesthesia by qualified persons. To one familiar with the potential problems of the unprepared obstetrical patient, these figures may be alarming but in general the good health enjoyed by our young mothers seems to be reflected in our maternal mortality statistics in Texas.

Discussion

Our use of the term "qualified" may be repugnant to some who feel all anesthetists should be physician anesthetists but an elementary knowledge of arithmetic will show if we were to simply replace the present nurse anesthetists in Texas with physicians, all graduates of all the Texas medical schools for three straight years would have to enter anesthesiology—instead of the 3 percent that normally find their way into the study of anesthesia. Again, at the present rate of approximately 57 percent capacity in our residency training programs, it would take all of our Texas-trained residents for the next 12 years just to replace the nurse anesthetists in Texas in 1966 without future needs considered. It is not the purpose of this paper to discuss the relative merits of the CRNA but, rather, to apply to Texas some recent information by direct means and statistical inference. It is worthwhile noticing, however, that our colleagues in obstetrics face a similar situation and are at this time admitting, perhaps more honestly than are anesthesiologists, that there is a definite place for the technician to supplement the technologist. Economically, I am sure that even the most severe critic of the qualified nurse anesthetist will admit complete coverage by anesthesiologists will never be a reality. One factor is the geographic distribution of population in Texas. Since we have established the fact that 1,000 anesthetists who are qualified in 1966 (637 TANA; 363 TSA) cover 85 percent of the surgical anesthesia and considerably less than that of the obstetrical anesthesia in Texas, then it becomes evident that we need at least 200 more qualified anesthetists in Texas. By comparison to 1955 we have come far. On the surface this may appear to be a drastic need but if we examine the situation for a moment and size up other areas, we quickly see we are not doing badly at all. In the USA in 1965, there were more than 25,000,000 operations,[6] most of which required anesthesia. We have every reason to expect this figure to increase yearly as in the past, and Texas' medical growth will be no exception. Our attraction to all anesthetists has been slightly better than the nation's average in the most recent years while our attraction to training in anesthesiology has been 9 percent less than the national average. The increases in qualified personnel of both physicians and nurses have been parallel and have slightly exceeded the ratio of new hospitals constructed in Texas in recent years. Hospital numbers and even personnel numbers do not, however, give the total picture. The fault in analysis can be seen in part in the vast expansion of the role of the anesthetists and the anesthesiologists. The ever increasing requests for assistance in areas of cardiopulmonary resuscitation, pulmonary therapy, metabolic and endocrine crises, drug-induced depressions and prophylaxis, and greater demands for anesthesia for non-operative diagnostic and therapeutic procedures further depletes the number of available anesthetists and anesthesiologists. If this were not enough, increasing obligations of and demands for anesthesiologists as key members of committees and boards where their special knowledge is of esteemed value further cut down on the time they can give to actual administration of anesthesia. This time is certainly not misused but it is obvious that there is a necessity for accepting the increasing use of the anesthetist for less difficult cases and, consequently, eventually a rearrangement of the economic and "ethical" arrangements now in vogue. With no intent to degrade the value or ability of the qualified nurse anesthetist, it should be categorically stated that no trained anesthesiologist should ever find himself in the position of competing with a nurse anesthetist for a living in his specialty. If by ability, knowl-

edge, attitude, and practice, his services can not establish their value with his medical colleagues and patients, then his training as a physician has been wasted. To be in competition with nurse anesthetists is likened to an obstetrician competing with a licensed midwife. Too often, I fear, apparent competition explained as the "hard facts of economy" is a shabby attempt to cover a technician-type practice. There can be no doubt of the tremendous need in the forseeable future for both the CRNA and the diplomate of the American Board of Anesthesiology and all levels in between. Our greatest need now is in recruitment of new qualified personnel to administer anesthesia both in Texas and nationally. The increase of slightly more than 2,200 anesthetists, physicians, and nurses, annually does not keep up with attrition and increased demands for anesthesia. On a state level we are doing well, but only by constant recruitment can we hope to continue. Anesthesiologists must offer by example the professional practice of anesthesiology. We must actively support and recruit for the widest use possible qualified anesthesia technicians to be guided by adequate and competent medical supervision.

Summary

Qualified anesthetists in Texas are generally available within economic support capabilities of the medical committees concerned. National averages are paralleled and presently less than 15 percent of surgical anesthetics are given by persons of "unknown qualifications." The continuation of this picture is dependent upon two primary factors (1) the expertness of practice and attitudes of the anesthesiologist and the anesthetist and (2) the increasing support and understanding of those dependent upon our knowledge and skill.

ACKNOWLEDGMENT

Mrs. Florence McQuellin,[3] Executive Director, American Association of Nurse Anesthetists, and Mr. Jim Kerley,[3] secretary of the Texas Association of Nurse Anesthetists (TANA), supplied information on nurse anesthetists. Mrs. Mary Jones assisted with information on the Texas Society of Anesthesiologists.

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Women in Health Services

In this country, over 3 million persons—between 4 and 5 percent of the total civilian labor force—work in health services. About 1,800,000, or 60 percent of these workers are women. The proportion of women workers varies by occupation, but they tend to be employed at levels lower than for men. In 1960, only 7 percent of the physicians and 1 percent of dentists were women. On the other hand, women constitute 85 percent of the medical technicians, as well as the overwhelming majority of nurses, social workers, laboratory workers, dental hygienists, assistants, aides, and clerical employees. Current forecasts indicate that an increasing percentage of the total labor force will be women.

—MARION B. FOLSOM, in speech before the Tenth Congress of Medical Women's International Association.

Meniere's Disease

Diagnosis and Treatment

REVIEW ARTICLES

ONE OF THE real misconceptions in medicine is that "all that is dizzy is Meniere's disease." Meniere's disease is a true pathological entity which has a definite set of symptoms, known physical signs, and well-documented histopathological changes. Although the etiology and pathogenic and histochemical changes are not known, the clinical picture is clear-cut and well established. This discussion will deal with the diagnosis and management of this clinical entity.

Diagnosis

Meniere's disease, as presently understood, develops a pattern of symptomatology as the result of certain histopathological changes in the inner ear. It is a chronic disease, usually starting in the third or fourth decade of life and lasting many years. There are acute exacerbations of dizziness, lasting several hours, which are the most dramatic and dreaded experiences of the entire malady. It is usually unilateral, although in approximately 10 percent of patients, it may be bilateral. The incidence is equally divided between men and women. It is a disease of the white race, seldom occurring in the Negro. In a series of 160 labyrinthectomies performed in 22 years in my office, only one patient was Negro.

The patient is usually tense and filled with emotional problems. Most of them admit their emotional problems and agree that the acute attacks are often initiated by some psychic trauma. To evaluate the role of the emotional factor, patients both with and without Meniere's disease were interviewed. Although most persons in both groups admitted they had some personal or work problems, those with Meniere's disease appeared less able to cope with these emotional challenges. It is not the emotional problems themselves—which seem to be universal—but rather the inability to cope with these problems which lays the foundation for development of the so-called "adaptive diseases."

The classical triad of symptoms—hearing loss, tinnitus, and dizziness—originally was described by Prosper Meniere[1] in 1861. This symptomatology still prevails; however, two other symptoms have been added: (1) fullness in the ear and (2) diplacusis binauralis dysharmonica. The disease has this characteristic set of symptoms:

I. HEARING LOSS.—The hearing loss is cochlear in type; that is, it is of an inner ear or perceptive type and has the following characteristics:

A. *Fluctuation of hearing.*—The hearing fluctuates early in the course of the disease. It becomes more severe just before the onset of an acute attack and remains so for several days following the attack. Late in the course of the disease, the hearing loss remains constant and usually at a level below 30 decibels but usually does not become complete.

B. *Discrimination.*—The patient has relatively good discrimination; that is, the ability to understand speech. This ability to understand is involved to a lesser degree in Meniere's disease than it is in the true nerve lesions such as in acoustic neuromas. As such, it is an important differential diagnostic point.

Understanding the emotional problems seen in Meniere's disease is as important as medical management. Surgery, however, should not be performed until all other medical methods of management have been exhausted, cautions the author.

RALPH J. CAPAROSA, M.D.

C. *Type.*—The hearing loss is recorded on the pure-tone audiogram as a flat sensorineural type. This, too, is an important diagnostic point.

The hearing loss usually starts before the vertigo since this disease is believed to be of cochlear rather than vestibular origin. In approximately 20 percent of the cases, the deafness may persist without vertigo. In this situation, great care must be taken to rule out cerebellar-pontine angle lesions. In rare instances, the vertigo may occur without deafness; and this presents a real diagnostic challenge.

II. TINNITUS.—The tinnitus is low-pitched and roaring. The intensity varies, and usually is more severe just prior to an acute attack. It abates slightly several hours after the acute attack. The patient may confuse pitch with intensity and may feel that the ringing is "high pitched" when actually he means loud intensity. The true pitch can be elicited by having the patient compare the sound of a 500 cycle per second tuning fork with one of 1,000 cps.

III. VERTIGO.—Vertigo is one of true rotation and is explosive and violent in character. It is exaggerated by any attempts at movement, and absolute rest and immobility offer the maximum relief from the dizziness. The involved ear is usually upward; however, in some cases, the patient will prefer a complete supine position with the eyes forward. It is extremely unusual to have the patient position himself with the involved ear downward. Nausea and vomiting do occur and reflect the severity of the attack. During the acute attack, the patient's hearing loss usually becomes more severe and the tinnitus more intense. In some situations, however, the concern with the vertigo is such that this change in the hearing level and the tinnitus is not noted subjectively.

IV. FULLNESS.—The feeling of fullness in the ear occurs in many patients and frequently may be the first symptom. Many patients have had repeated eustachian catheterizations and politzerizations when in reality the middle ear was normal. When there is a hearing loss of the perceptive type and the middle ear structures are normal, Meniere's disease must be suspected.

V. DIPLACUSIS DYSHARMONICA.—This is the hearing of the same sound differently in the involved ear than in the uninvolved ear. This is characteristic of Meniere's disease. Unfortunately, patients often confuse loudness with difference in tone. People with musical backgrounds have no difficulty with this; however, others frequently do, thus limiting the value of the symptoms.

The diagnosis of Meniere's disease can usually be made when any three of the previous symptoms are present. The differential diagnosis must include:

1. Cerebellar and cerebellar-pontine angle lesions.
2. Vascular spasms, insufficiency, and occlusion.
3. Anxiety states.
4. Otosclerosis.

Each of the previous diagnoses has signs and symptoms which are characteristic. The real difficulty arises in those infrequent cases of Meniere's disease in which the episodic

vertigo does not occur—the cochlear Meniere's disease.

1. Cerebellar-pontine angle (CPA) tumors usually start with an insidious loss of hearing which is slowly progressive without the fluctuations noted in the early stages of Meniere's disease. The special battery of audiological tests including the speech and discrimination scores, Bekesy, short increment sensitivity index, recruitment, and tone decay tests serve well to differentiate the two. The dizziness of CPA tumors is not an early characteristic symptom. When it does occur, it is usually in the form of an unsteadiness and is more or less present continuously. The explosive episodes of Meniere's disease do not occur.
2. Vascular spasm, insufficiency, and occlusions occur more frequently in older persons and in people who have associated vascular disease. Vascular insufficiency is especially common in the older age groups when motion of head or changes of position occur too rapidly. Occlusive vascular disease in the brain stem is usually associated with other brain stem signs.
3. The anxious patient may present almost any pattern. He usually, however, does not present the episodic vertigo, and the hearing loss is erratic. Patience is necessary, and the evaluation must be complete enough to rule out organic disease. A complete evaluation helps to assure the patient that "everything possible is being done" as well as assuring the physician that an organic lesion is not present on top of an anxiety state.
4. The otosclerotic patient, not infrequently, has a "fragile" labyrinth and may have an associated hydrops-type symptomatology. The incidences of patients with true labyrinthine vertigo attacks is probably higher in the otosclerotic population than in the general population. Good audiological studies will, of course, include the possible diagnosis of otosclerosis. The prognosis for surgical success is less favorable in the otosclerotic patient with a history of labyrinthine vertigo attacks.

Treatment

I. MEDICAL.—The disease is one of long duration with periods of lengthy remissions between series of attacks even without therapy. Any enthusiasm engendered for a single mode of treatment must be tempered with good judgment in evaluating the final results. The chronicity of the disease makes an accurate statistical follow-up difficult. The medical management of Meniere's disease is divided into the therapy for the acute and chronic states.

A. *Acute Attacks.* — The experiences which the otolaryngologists have with acute attacks is usually limited since the patient often is unable to seek help immediately. The attack usually lasts from one-half hour to three hours, and the patient recovers completely from the attack with no sequelae. Any attack of true vertigo which lasts longer than half a day must be looked upon with suspicion as being caused by some disease other than Meniere's disease. The drug of choice in the acute attack is Nembutal suppositories, 200 mg. (3 grains), administered by the patient or by a member of his family. This does not alter in any way the pathological-physiological derangement which produced the symptom but it does put the patient at rest. This permits a time lapse which is necessary for the attack to subside. The patient gains a great deal of security by knowing that the medications to alleviate this severe acute unsteadiness is readily available. This is most reassuring and allays a great deal of needless anxiety.

B. *Chronic Care.*—The chronic stage of the disease has been managed in many different ways. The following are some of the methods:
   1. Fluid restriction
   2. Sodium restriction
   3. Diuresis
   4. Diet restriction
   5. Vitamin administration
   6. Hormone administration
   7. Desensitization
   8. Vasodilation

   Good results have been reported with all of these methods. Some form of them has been used at one time or another in our office. At present, however, histamine desensitization

forms the basis of our office treatment. Any long-term treatment must be simple, inexpensive, and relatively successful in relieving the vertiginous episodes. Over the past 22 years this method has proved really reliable. It is certainly not a "cure" but it has been reliable enough to avoid destructive surgery in a large percentage of patients.

A mechanism of Meniere's disease has been suggested as being an "autonomic dysfunction" producing increased fluid volume as the result of the "spastic-atonic" state of a vascular bed. Histamine desensitization[2] has been shown clinically to help prevent the development of this labyrinthine "hydrops."

Pure histamine crystals are used to make the serial dilutions. A 1:1,000 dilution is used for an intradermal skin test. Reactions are graded from 1+ to 4+ depending on the size of the wheal, the number of pseudopods, and the surrounding erythema. Reactions of 4+ and 3+ are started at the treatment dilution of 1:1,000,000. Reactions of 2+ and 1+ are started at the treatment dilution of 1:100,000. The schedule starts with 0.1 cc. given weekly, and increased by 0.1 cc. each week for ten weeks. At the end of ten weeks, either the next dilution is serially increased or else the patient is maintained on 0.1 cc. of the next dilution given once a week without increasing the dose. The decision depends upon the clinical response. The number of injections may be continued at once a week or may be increased to two or three times a week. In rare situations, the patient may require injections daily.

Because the risk of reaction is small, early attempt is made to have the patient or a member of his family assume the responsibility of giving the injections. This, of course, reduces the cost of the treatment as well as lessening the inconvenience.

Those who do not respond to the desensitization mechanism are suspected of having an allergic problem and are subjected to an allergen survey.[3] Offending foods are eliminated, and the appropriate inhalant desensitization program is established. This further allergy management adds significantly to the number of patients which can be controlled without surgery.

Concomitantly, a course of Pro-Banthine with Dartal is given for its anticholinergic and sedative properties. This is prescribed three times a day after meals, and it may be continued for six weeks. It appears to be especially effective when first given. Repeat courses of this, however, appear to be less and less effective. A large number of patients can be controlled with this method of management.

II. SURGICAL TREATMENT.—For those who continue to have vertiginous episodes, surgery can control the attacks. At present, destructive labyrinthectomy is the procedure of choice. The partial labyrinthectomy, either mechanical or ultrasonic, and the shunt procedures have fallen short of the goal of complete control. Complete control is assured in a significantly larger percentage of patients with total labyrinthectomy. Surgery, however, should not be performed until all other medical methods of management have been exhausted.

Summary

The following impressions can be gained from evaluating the management of Meniere's disease:

1. Patients treated early while the disease is in the cochlear state tend to respond much more favorably than those seen in the late vestibular stages.
2. Many patients' symptoms can be controlled with histamine injections. In addition, others can be controlled with management of allergy.
3. Attention to the patient's emotional problem through kind and sympathetic understanding is probably as important as any other single factor of therapy. This cannot be emphasized too much. An attitude of desiring to understand the patient's emotional problems is a prerequisite in managing these patients successfully.
4. Assuring the patient that treatment will succeed is extremely important.
5. Further assuring him that surgery also can be performed if necessary is also of extreme importance and helps to allay the patient's fear that this is an "incurable malady."

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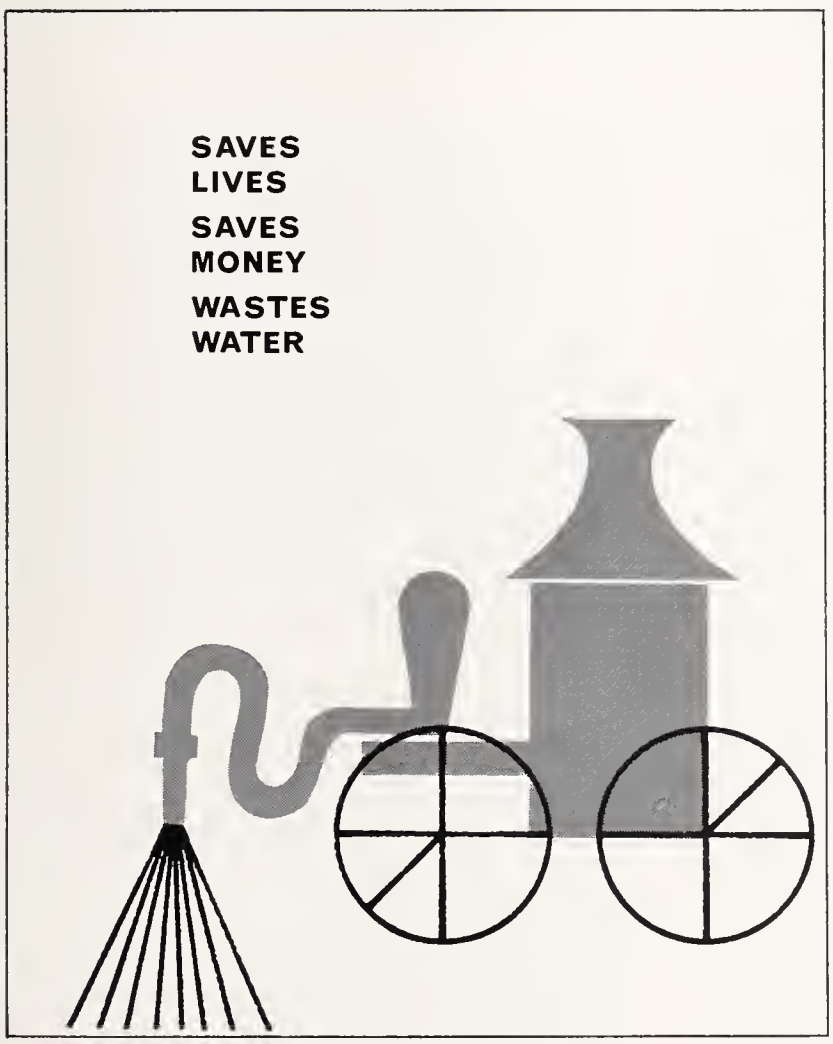

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Clinicopathologic Conference

Dudley Jones, M.D.; Roberto Melgar, M.D.; Fred S. Brooksaler, M.D.;
Peggy Sartain, M.D.; and Heinz F. Eichenwald, M.D.

DR. MELGAR: A girl weighing 2,050 gm. was born to a 14-year-old Negro, gravida 1, para 0, on Nov. 27, 1965. The mother's last normal menstrual period was March 27, and her expected date of confinement was Jan. 1, 1966. The mother had been examined regularly in the Outpatient Obstetrical Clinic since early October, 1965, when a routine serologic test for syphilis was positive to 16 dilutions. The mother was instructed to go to the venereal disease clinic for immediate therapy; however, the VD Control Center has no record of a visit by the infant's mother, and apparently she was never treated. The mother has B positive blood. The labor and delivery were normal, with a cephalic, low forceps delivery under local anesthesia. Total duration of labor was 18 hours, and the fetal membranes ruptured 30 minutes prior to delivery. The mother received 25 mg. Demerol and 25 mg. Phenergan 10 hours prior to delivery. The placenta was described as grossly normal.

The infant, after delivery, had a three to four minute delay before respirations were established by tracheal intubation. She was given an Apgar rating of 1. The fingernails were stained by meconium, and blood-stained mucus was aspirated from the endotracheal tube. Inspiratory and expiratory rales were heard over both lung fields. Respirations were difficult and shallow with some intercostal retractions. The heart rate was 90 to 100 per minute, and no murmurs were heard. The infant's abdomen was distended, and a fluid wave was present. The liver extended below the umbilicus, and the spleen was palpable 7 cm. below the left costal margin. Many vessels were prominent over the head, abdomen, and all extremities except the right leg. Multiple ecchymoses were present in the skin of the abdomen and extremities. The infant was pale and had generalized edema and hypotonia.

Shortly after birth the infant became jaundiced, and blood cultures were drawn from the right femoral vein; oozing of blood into the surrounding soft tissues continued for approximately 1½ hours despite external pressure on the vessel. Because of this, no other specimens for laboratory work were drawn. Penicillin and Kanamycin were given intramuscularly just after birth. Shortly thereafter, the infant experienced several apneic episodes. Despite vigorous resuscitative measures, she died 3 hours and 20 minutes after birth.

This is the fifth in a series of clinicopathologic conferences from The University of Texas Southwestern Medical School, Dallas, which cooperated with the Children's Hospital of Texas to prepare this case. Guest editor for the series is Dr. Bruce D. Fallis, professor of pathology. Participants this month are Dr. Dudley Jones, resident in pathology, Dr. Roberto Melgar, fellow in pediatrics, Dr. Fred S. Brooksaler, professor of pediatrics, Dr. Peggy Sartain, instructor of pediatrics, and Dr. Heinz F. Eichenwald, professor and chairman, Department of Pediatrics.

Clinical Discussion

DR. FRED S. BROOKSALER: If we had discussed congenital syphilis ten years ago, we would have discussed a chapter of medical history, because at that time, we were sure that syphilis, and especially congenital syphilis, had vanished from the United States. About 30 years ago, one of the most frequented departments in our Outpatient Clinic

Today's intern or resident may never see a case of congenital syphilis; it involves multiple systems and mimics many conditions. Now, venereal disease is increasing, and review of this case will remind the attending physician that a sick baby may have lues.

was the Lues Clinic. Every week we had 30 to 40 patients. Unfortunately, to a slight degree, these "good old times" have reappeared. Since 1957, there has been a resurgence of syphilis and of congenital syphilis in this country.[1,2,6] The reason for this is uncertain. Possibly it is mobility of the population, or promiscuity, or failure of the mothers to show up for antisyphilitic treatment, or perhaps the complacency of doctors who say, "Alright, one shot of penicillin is enough to cure syphilis." Whatever the reason, we have congenital syphilis again. We also see occasional instances of acquired syphilis in older children.

The manifestations of congenital syphilis in the young infant, from two weeks to three months old, are quite varied, but many of the clinical manifestations are sufficiently characteristic to be all but pathognomonic. For example, there are the rhagades (fissures around the mouth) which strongly suggest the diagnosis of syphilis. Rhinitis (the snuffles) occurs in many cases, with noisy breathing and a purulent or blood-tinged nasal discharge. Striking changes occur in the bones—osteochondritis, osteomyelitis, and periostitis—and cause severe pain. Such children may stop moving the arms: Parrot's pseudoparalysis. This is not paralysis, but the result of severe bone pain. Osseous lesions are early manifestations and are already present at delivery in many infected neonates. Therefore, roentgenograms of the long bones should be done in all suspicious cases at birth and repeated in four to six weeks. Other findings include prematurity, anemia, generalized lymphadenopathy, hepatosplenomegaly, malnutrition, jaundice, pneumonia, and skin eruptions. The skin eruptions may be macular, pemphigoid, bullous, or of many other types. Syphilitic dermatitis in the infant may simulate pemphigus, impetigo, decubitus ulcers, diaper rash, and many other skin lesions.

The serologic test for syphilis is one of the most important diagnostic tools in the study of congenital syphilis. We used to do Wassermann reactions at each and every admission to the hospital along with the complete blood count and urinalysis. Unfortunately the results of the serologic tests for syphilis in a newborn baby, the positive as well as the negative ones, are not completely reliable. A mother who has been treated for syphilis may still have some reagin, and this reagin will be passively transferred to the child and cause a positive reaction to the serologic test for syphilis; such a false positive reaction will gradually disappear without treatment. There are also false negative serologic tests for syphilis, which are misleading unless special quantitative tests are done.

In congenital lues, any or all of the organs and tissues may be involved. In fact, we used to say if you did not know the differential diagnosis of any disease in a little baby, you should consider syphilis, because everything can be imitated by this disease.

Late congenital syphilis becomes manifest at about 8 or 10 years of age, often with the characteristic Hutchinson's triad, consisting of abnormal teeth, interstitial keratitis, and nerve deafness. The typical peg-shaped Hutchinson's incisor teeth occur only in the late dentitions, not in the first teeth. A variety of other lesions occur in late congenital syphilis: saber shins, thickened clavicles, Clutton joints, gummas, and so forth. Our hope now is that the current cases of congenital lues will be diagnosed and treated early in life, and we shall not see, again, examples of these late lesions.

By awareness in differential diagnosis, by treating adequately every infected child, and by using the confidential case finding services provided by our health departments, congenital syphilis can be eradicated.

DR. PEGGY SARTAIN: Prior to 1960, we were diagnosing congenital lues infrequently (less than one child per year). Beginning in 1961 we began to see it with increased frequency, and during the past several years we have seen about three infants per year with congenital syphilis. I would like to review the clinical findings in line

with what Dr. Brooksaler has just said. Snuffles, rhagades, mucocutaneous lesions, and pseudoparalysis were uncommon in our patients, who ranged in age from 1 to 21 days at the time of diagnosis. Rhinitis, skin lesions, and pseudoparalysis may not become evident until two or three months of age. Of nine reported patients,[5] five were initially thought to have some other disease: sepsis, ruptured spleen, congenital nephrosis, erythroblastosis, and congenital heart disease. In other words, the diagnosis was "stumbled on" after the results of the maternal serology became known or after roentgenograms of the long bones were taken.

Of nine patients, anemia was present in eight, thrombocytopenia in four, hepatosplenomegaly in eight, and jaundice in six. Anemia was seen in both term and premature infants and was apparently hemolytic. There was intense reticulocytosis (8 to 15 percent), and peripheral blood smears showed normocytic, normochromic erythrocytes with polychromasia and many nucleated red blood cells. Hemolytic activity was seen during the first two or three weeks of illness, and several infants required simple transfusions during this time. White blood cell counts varied widely, ranging from 5,000 to 40,000 per cu. mm., and there was variation in differential counts. One baby had a leukemoid reaction with immature white cell forms, suggesting congenital granulocytic leukemia to some observers. His bone marrow revealed merely granulocytic hyperplasia, and after treatment with penicillin the white count returned to normal. Thrombocytopenia is known to accompany severe generalized infection in the newborn, regardless of the organism. Four infants in our series had platelet counts less than 70,000 per cu. mm. By the time therapy was completed, the three surviving infants had normal platelet levels.

Jaundice was present in six babies and with one exception there were elevations of both conjugated and unconjugated bilirubin. The exception had only unconjugated bilirubin. He had a large hematoma of the scalp, and this probably accounted for a considerable bilirubin load. The five other infants with elevations with both conjugated and unconjugated bilirubin had significant hepatomegaly, and about half had elevated serum glutamic oxaloacetic transaminase (SGOT). Bilirubin levels rapidly returned to normal once therapy was begun.

Congenital syphilis must be returned to the differential diagnosis of the "sick" newborn. Woody and associates[6] have commented that missed diagnoses may be common in an era when house officers go through their entire training period without observing a single case of congenital syphilis. Because congenital syphilis involves multiple systems, it may masquerade as sepsis, congestive heart failure, or congenital nephrosis. Exactly the same clinical and hematological picture may result from toxoplasmosis, cytomegalic inclusion disease, or the rubella syndrome. The infant with congenital lues resembles the erythroblastotic infant in many respects, both conditions being responsible for anemia, nucleated erythrocytes in the peripheral blood, hepatosplenomegaly, and foci of extramedullary hematopoiesis.

DR. HEINZ F. EICHENWALD: It is important to emphasize that the routine screening method used for the detection of reagin is a nonspecific test. Whether one uses one of the various precipitation tests or one of the complement fixation tests, the test is still a nonspecific one, and a positive reaction to the Wassermann, Mazzini, or Kolmer test does not necessarily mean the patient has syphilis. There are several interesting aspects of the immunology of congenital syphilis which have received attention recently. It has been known for a long time that a mother with a positive serologic test for syphilis will transmit reagin to the fetus and that the fetus will then have a positive serclogic test for syphilis whether or not he, in fact, has syphilis. If the mother has serologically resistant syphilis and has been adequately treated, the baby may have a positive Wassermann reaction in the cord blood. Does this mean the infant should be treated? Most of us feel that if the mother has had adequate therapy, and if the baby has a positive Wassermann reaction but all other indications of syphilis (clinical examination, x-rays, positive spinal fluid reaction) are absent, this baby should not be treated but should simply be observed. There is a fairly rapid decay in the infant's reagin titer if the infant does not, in fact, have congenital syphilis.

Another interesting aspect, suggested a few years ago by Dr. Silverstein,[4] was that it was possible for the fetus to form antibodies as early as the 22nd or the 24th week of gestation. He based his suggestion on examinations of stillborn fetuses with congenital syphilis. He found many clusters of plas-

ma cells, which appeared to be synthesizing protein and, therefore, probably were making antibodies. This was the first suggestion that the human fetus could form antibody and is at variance with most of the previous theories of immunology. We now know that the fetus, at some point, probably starting after the 18th week, is capable of producing antibody.[3] The congenital syphilitic infant is born with not only passive antibody derived transplacentally from the mother, but also active antibody which he himself has formed. Because of this the infant with congenital syphilis maintains his antibody titer and, in fact, increases it once he is separated from the mother. If the mother had been adequately treated during the late stages of pregnancy and the infant is asymptomatic with normal x-rays and normal spinal fluid, the infant should be watched. The only infants that should be treated are those whose mothers had not been treated or those with clinical signs of syphilis. It is often stated that the spirochete does not affect the fetus until the last half of gestation. This is now known to be incorrect. Therefore, the mother should be treated as early as possible in her pregnancy; this is rarely before the third month, because women usually do not go to a doctor prior to this time. As soon as the diagnosis of maternal syphilis is made, the mother should be treated, and her reagin titer should then be followed in serial fashion.

Another problem that arises is the problem of reinfection. It is possible for the mother to be reinfected from the same source from which she received her initial infection and to have another active case of syphilis after she had been treated two or three months before. However, if the woman is being followed closely, this sort of thing can be excluded, particularly if her initial contact is identified and treated.

Pathological Findings

DR. DUDLEY JONES: The lesions of congenital syphilis of the newborn consist of perivascular infiltrates of lymphocytes, plasma cells, and macrophages; diffuse interstitial fibrosis; and nonspecific retardation of development. These lesions may occur in any organ or tissue.

At necropsy, the infant weighed 2,000 gm. and was deeply jaundiced. The abdomen was moderately distended. There was a large, subcutaneous hematoma in the region of the femoral vein puncture, and ecchymoses were present over the entire body. However, there were no cutaneous syphilitic lesions. The cutaneous lesions of syphilis are not usually present at birth but appear weeks or months later. The peritoneal cavity contained 60 ml. of serosanguineous fluid.

The heart weighed 15 gm., and the myocardium was pale and flabby. Petechiae and ecchymoses were present in the pericardium, epicardium, and myocardium. Histologic study revealed a mononuclear cell perivascular infiltrate in the myocardium, and special stains revealed a few spirochetes.

The lungs were the seat of typical pneumonia alba. They weighed 50 gm. and were firm, poorly aerated, and whitish-yellow. There were multiple ecchymoses and petechiae in the visceral pleura and in the pulmonary parenchyma. Microscopic examination revealed an interstitial mononuclear cell infiltrate and slight interstitial fibrosis (Fig. 1). The lungs were incompletely developed and consisted of alveolar ducts with relatively few true alveoli. The alveolar ducts were poorly expanded and were lined by plump alveolar epithelial cells (nonexpansion atelectasis). They contained many keratin squames from the amniotic fluid, indicating intrauterine aspiration, probably the result of intrauterine anoxia. Spirochetes were identified around the small blood vessels and within the alveolar septa.

The liver weighed 104 gm. and had a normal shape. Externally and on cut surfaces the liver parenchyma was mottled and yel-

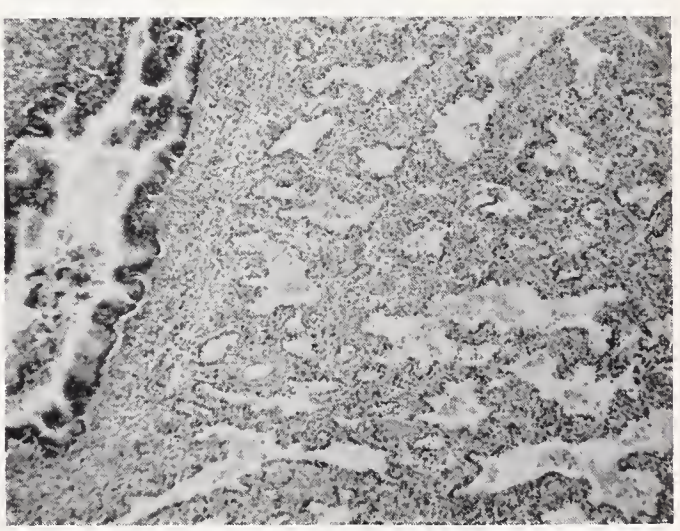

Fig. 1. Photomicrograph of lung. Hematoxylin and eosin stain. Note the inflammatory infiltrate in the wall of the bronchiole (left) and in the alveolar septa. The alveolar ducts are dilated and contain some pale amniotic debris. They are lined by plump cuboidal epithelial cells and are separated by cellular fibrous tissue with many chronic inflammatory cells. X 80.

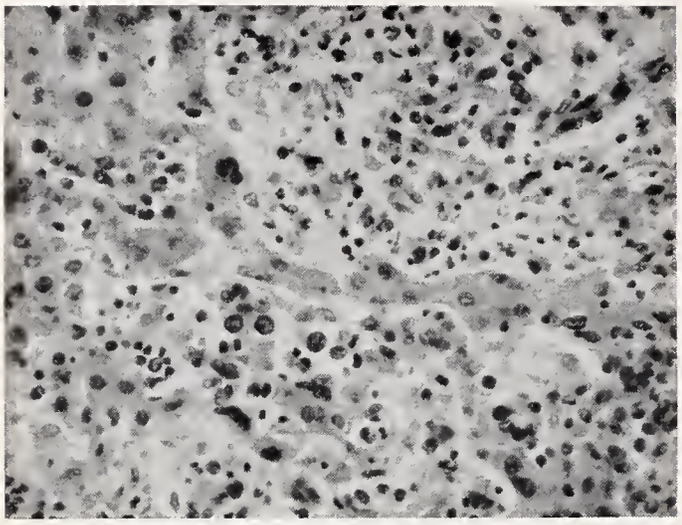

Fig. 2. Photomicrograph of liver. Hematoxylin and eosin stain. The hepatic cords are disrupted by an infiltrate of acute and chronic inflammatory cells. Note the multinucleated liver cell (upper left). X 344.

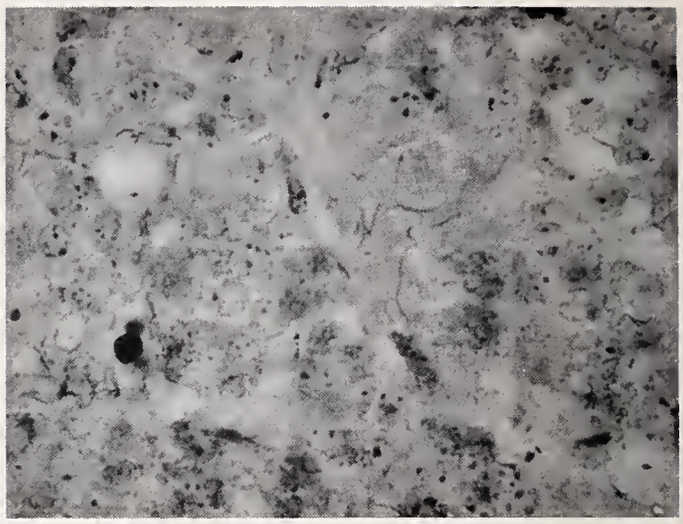

Fig. 3. Photomicrograph of adrenal. Warthin-Starry stain. Many spirochetes are visible. X 776.

lowish-tan, but no increase of fibrous tissue was apparent. Microscopically the lobular architecture was disrupted, and the liver consisted of small, haphazardly arranged clusters of liver cells separated by many mononuclear inflammatory cells (Fig. 2). Some multinucleated liver cells were present. There was a minimal increase of fibrous tissue, especially periportally. Hemosiderin-laden macrophages and many foci of extramedullary hematopoiesis were also present.

The pancreas was normal grossly, but microscopic study revealed loss of parenchyma, mononuclear cell infiltrate, and widespread interstitial fibrosis.

The spleen was enlarged and weighed 20 gm. A fibrinous exudate covered its serosal surface. Histologic study revealed a slight increase of macrophages and interstitial fibrosis in the red pulp.

Special stains revealed a few spirochetes in the liver, pancreas, and spleen.

The adrenal glands were of normal size but contained extensive foci of hemorrhage. Although inflammatory infiltrate was minimal, special stains revealed numerous spirochetes (Fig. 3).

The brain weighed 240 gm. Small subependymal and intraventricular hemorrhages were present.

Cut sections of the long bones revealed classic syphilitic osteochondritis. In the metaphysis of each involved bone there was a broad, irregular band of opaque yellow tissue demarcated toward the diaphysis by a red band of hyperemia and a gray-white zone of fibrous tissue. Histologic study revealed disorganization and retardation of endochondral ossification, mononuclear cellular infiltrate, and abundant granulation tissue. Numerous spirochetes were present. Subacute periostitis was also present.

At the time of delivery, the placenta was stated to be normal by the attending obstetrician. Characteristically, the placenta of congenital syphilis is enlarged, firm, and pale. However, the placenta in this case was unremarkable grossly. Nonetheless, microscopic study revealed slight inflammation and fibrosis of the placental villi and umbilical cord, and a few spirochetes were present. It should be emphasized that a grossly normal placenta does not exclude congenital syphilis.

In summary, this infant had typical congenital syphilis with pneumonia alba, myocarditis, hepatitis, splenitis, fibrinous perisplenitis, hemorrhagic necrosis of the adrenals, osteochondritis, periostitis, and placental infection. There was also a hemorrhagic diathesis with generalized petechiae and ecchymoses, pallor of the viscera caused by anemia, jaundice, ascites, subependymal and intraventricular hemorrhage in the brain, and slight prematurity.

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The authors, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas 75235.

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References: (1) Based on 23 clinical papers, 1512 cases. Bibliography on request. (2) Bush, I. M., Orkin, L. A., and Winter, J. W., in Sylvester, J. C.: Antimicrobial Agents and Chemotherapy—1964, Ann Arbor, American Society for Microbiology, 1965, p. 722.

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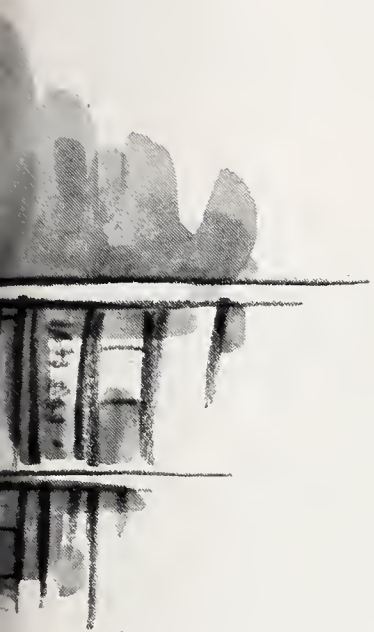

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Physician's Image, Government Control Concerns of Speakers at PR Conference

How to strengthen the image, individually and collectively, of the physician and the medical profession, and how to stem the flow of governmental control over the "private sector" of life today in the United States were the primary concerns of a series of speeches and panel discussions heard during the Texas Medical Association's 12th annual Public Relations Conference and Orientation Program.

Meeting in a one-day session Saturday, Oct. 1, at the TMA Headquarters Building in Austin, more than 671 Texas physicians, wives, and guests heard a group of outstanding speakers from New York, Los Angeles, Baton Rouge, Chicago, St. Louis, and Alexandria, Va.

"It's time you got off your hands and went forth to do battle," Roy Pfautch of St. Louis, president of Civic Service, Inc., told the gathering at the day's closing session, echoing the challenges set out by other speakers during the day, urging the medical profession to take an active part in the public and private domains of their communities.

Besides Mr. Pfautch, whose St. Louis organization specializes in institutional, religious, educational, and political public relations, fund raising, and opinion research, guests during the afternoon session heard John J. Henry of Alexandria, Va., editor of *Babson's Washington Forecast Letter.*

Morning speakers were John J. Powers of New York, president of Chas. Pfizer & Co., Inc.; Robert B. Wolcott, Jr., of Los Angeles, president of the Public Relations Society of America; and Dr. J. A. Sabatier of Baton Rouge, member of the American Medical Association's Committee on Quackery.

Mr. Powers

The importance of a clear division of responsibility among the three participants in health research—industry, universities and government—was stressed by Mr. Powers in his speech opening the first session.

He called his message "an attempt, in a time when there is so much to do, to suggest the need for each group to concentrate on the thing it does best."

Mr. Powers pointed out that the country recently has undergone an information explosion in the life sciences, thus vastly increasing our fund of basic knowledge.

"This in turn has given a new impetus to medical product research which has enormous significance for the practicing physician," he said. "Indeed, I believe it is no exaggeration to say that we are in the midst of a revolution in medical product research."

He traced the changing scene of the technical side of medical research from the days of empirical research, when hard work resulted in medicinal products to relieve symptoms and deficiencies caused by disease and which eventually evolved the sulfa drugs and antibiotics and the first successful attacks on the causes of disease. This was followed by the more sophisticated structure-activity work of the biologist and the medicinal chemist, which now has provided a body of information and developed laboratory techniques that are helping to launch the next era of research in the life sciences—the era of molecular biology.

The politico-economic atmosphere of medical research, just as the technical side, has changed, too, Mr. Powers emphasized.

Traditionally, he said, most government-supported work has been devoted to the development of basic knowledge and the training of young scientists.

"But there has been an increasing tendency for government to work on or support work directed to the discovery and development of specific products," he added. "Concurrently, industry has found it necessary to supplement its traditional product discovery and development role with an increasing amount of basic knowledge to help fill some of the gaps in knowledge.

"The traditional lines of demarcation between government and industry in medical research are becoming blurred."

Mr. Powers urged that the respective roles of the three partici-

Dr. Charles D. Reece of Houston chats briefly with Dr. J. A. Sabatier of Baton Rouge, La., Public Relations Conference speaker and member of the American Medical Association Committee on Quackery.

Mr. John J. Powers, president of Chas. Pfizer & Co., Inc., Conference speaker, and Dr. Milford O. Rouse of Dallas, President-elect of the American Medical Association, pick up materials at registration desk.

Mr. John J. Henry, editor of *Babson's Washington Forecast Letter*, addresses the afternoon session of the Conference.

Mr. Robert A. Wolcott, Jr., president of the Public Relations Society of America, Conference speaker, in session with Dr. J. L. Cochran of San Antonio.

Dr. David Wade of Austin, immediate Past President of TMA, engages Conference speaker, Mr. Roy Pfautch in conversation before the afternoon session.

pants in health research be re-examined, not only for political and economic factors, but for methods of achieving the greatest efficiency in the effort to reach the health goals put forth by the present administration.

"It is important to know who is going to do what work," he said, "and the extent and kind of interplay there should be between government, the universities, and industry."

While he acknowledged the efficacy in some instances for joint action, Mr. Powers expressed the need for government to continue—indeed, to increase—its support of the amassing of basic knowledge and of training people in the health research field, by such means as financing university projects.

"In doing so it releases the energies of the private sector to do what it does best—to discover new drugs and to develop their application in the practice of medicine," he said.

The one factor to be singled out for emphasis, he said, is the suggestion that government should pay primary attention to the training of scientists.

"And this means strong support of the universities," he said.

"A program for the development, principally by government and the universities, of a broader infrastructure of basic knowledge, made freely available to anyone, and of trained scientists, will create the platform for a tremendous forward thrust in new-drug discovery and development beyond anything heretofore imagined.

"And to accomplish the latter is the primary responsibility and the special skill of industry, operating in a competitive climate," he pointed out.

Speakers porticipating on the ponel session, "New Federol ond Stote Medical Progroms and the Physician," gather before session's beginning. Porticipants are, left to right, Dr. John F. Thomas of Austin, Dr. Chorles Mox Cole of Dallas, Mr. C. Lincoln Williston of Austin, Mr. Fred R. Higginbotham of Dollas, and Dr. Howord R. Dudgeon, Jr., of Woco.

Another ponel session considered "Reaching the Public Through Positive Public Relotions." Speokers were Dr. Joseph T. Ainsworth of Houston, Mr. Jim Reed, Mr. Leo E. Brown, and Mr. C. Darrell Coover, oll of Chicogo.

Mr. Wolcott

Putting medical public relations on a personal level by recognizing the "role of the individual doctor in the total human relations and public relations picture of the medical profession" was urged at the morning session by Mr. Wolcott.

He told the doctors that "all of the magnificent public relations and communications work of the AMA—and your county society as well—will fall on hollow ears if the individual in the neighborhood is unfavorably handled by his local doctor."

He gave the group several positive suggestions for improving public relations at ground level, beginning with taking stock of the human relations process in the doctor's own office.

"No matter how small or large the organization, policy—and in this case public relations attitudes policy—stems from the top," he said.

He suggested that the doctors check their own attitudes as well as those of their staffs, in order to maintain the human relationship factor at its best.

He also urged the doctors to make a real contribution to their communities through participation in such positive activities as urban renewal.

Finally, he urged that "your public posture be one that largely is in favor of propositions, but is not one that is always against everything."

He pointed out that his impression of the AMA, undoubtedly an unfair but an honest one, has been one of a "defensive organization that has been fighting change, but has not necessarily been in favor of progress."

Conversing during morning coffee break at the Conference are Dr. James D. Murphy, Texas Medical Association President, Dr. James A. Hallmark, both of Fort Worth, Dr. A. J. Gill and Dr. Frank Harrison, both of Dallas.

To counteract such opinions, he suggested that doctors take positive stands and make positive contributions to their communities.

"If people think you have the public interests at heart—and not just the doctors'—you can go a long way towards building respect and recognition," he said.

Dr. Sabatier

Some of the results of a study of chiropractic by members of the American Medical Association's Committee on Quackery were presented at the morning session by Dr. Sabatier, a member of the committee.

In his slide presentation of excerpts from chiropractic textbooks, news clippings, testimony, and the like, Dr. Sabatier revealed some of the basic philosophies and methods of chiropractic training and practice.

"Medicine and the medical profession have an obligation to the general public in ascertaining health practices and standards," he told the group. "This is accomplished in evaluating new methods of diagnosis and treatment as well as in evaluating health legislation."

He pointed out that chiropractic presents itself as having competence in the broad scope of diagnosing and treating all human disease.

"We believe that it is the obligation of chiropractic to be submitted to at least the same degree of scrutiny continuously applied to medical methods," he said.

Urging that the tenets of chiropractic be revealed to the general public, Dr. Sabatier pointed out that "at this time the success of chiropractic is based upon testimonials rather than testimony."

Mr. Henry

In a witty, tongue-in-check presentation, Mr. Henry discussed the trends of paternalistic government: "Can We Afford It Financially or Spiritually?"

"All of us have aspired to a place in society—and many of us thought that we'd never make it," he said. "But now—suddenly—with no effort on our part, and without even learning whether we're supposed to eat caviar with a fork or a spoon, we have not only been catapulted into society—but into the Great Society."

In a more serious vein, he told the group: "In return for . . . paternal benevolence, the American citizen has been willing to trade measure after measure of his personal freedom. He has become willing to conform to the master plan—to become a number in the computer of life.

"He has surrendered his freedom of choice to a dominating government which prescribes the exact method by which he may seek the type of welfare and happiness which that government feels is best for him. It is becoming the same sort of welfare and happiness that is available to everyone else—no more, no less.

"Each new milestone in the path toward the Great Society takes more from the 'haves' and distributes it to the 'have-nots'—places more burdens on the 'can-do' individuals in order to give more benefits to the 'couldn't-care-less' boys."

Mr. Henry warned the medical group that "we haven't reached the end of this parade." Urging the doctors to work personally to stop the growing regimentation, he said, "The longer we fail to realize what we are doing to ourselves and to our nation, the more difficult it will become to lift ourselves up by the bootstraps from the mire of personal and national mediocrity."

He pointed out that motivation to alter "this collision course with a fate of collective insignificance" must come from the people.

Fired by the inspiration of our forefathers, he went on, "We must dedicate ourselves to a rebirth and remolding of the great American image—pledging our efforts—and working tirelessly to attract the efforts of others—to the task of transforming our nation once again into a land of industrious, responsible citizens who neither need nor want to live by government handout."

Until government subsidy and controls over private decisions and actions are checked, he concluded that "we will not restore the priceless American freedom which we are now unwittingly selling—piece by piece—in return for the shoddy security of public charity."

Mr. Pfautch

Another call to the physicians to take an active part in the politics, economic planning, and legislation of the United States was made by Mr. Pfautch, the day's final speaker.

"The authors of our Constitution provided for the evolution of democracy, accepting the changing nature of men," he pointed out. "They built in a process of amendment, with such power residing in the people at the state level."

"It is this process of evolution by the will of the people which is most disregarded today," he said.

"Instead, in an uncanny and unforeseen union of executive and judicial power, the legislative which rests as the final hallmark of the people has fallen before a tightening of the compact of government."

He cited as marks of deterioration of representative government such factors as the upset of the traditional balance of power originally built into the Constitution, and the power of the press

Physicians who have served in Vietnam in volunteer civilian capacities were honored and presented with plaques at the Public Relations Conference by Dr. James D. Murphy, TMA President, third from right. Doctors present to receive recognition were, left to right, Dr. Billy Jim Blankenship of Galveston, Dr. Robert Franken of San Angelo, Dr. Wayne Truax of Galveston, Dr. Edward B. Singleton of Houston, Dr. Jerry W. Bains of Galveston, and Dr. Kurt Lekisch of Midland.

"to sway and to mold the opinion of a majority based on their own interpretation rather than necessary facts in some instances."

"The final extension of central power," he continued, "has been the take-over of the economic sphere of our life by Executive Government."

He pointed to a Congress "passively (accepting) the nature of this change," but added that "on the other hand the Congress in recent months has indicated its restlessness at the hands of the Federal leash."

Emphasizing that the 1966 Congressional elections become vital stakes, Mr. Pfautch urged the physicians to take an active part in campaigning for candidates of their choice.

"First of all we can each accept the responsibility to examine just what our position is in the structure of our life—that is, in the world around us," he said.

He challenged the doctors to examine their role and to take stock of their influence within their communities, in order to use that influence most wisely. He also suggested practical ways in which the physicians could participate actively in the upcoming elections.

"Because we are willing to look with cold hard eyes at the government we love and to see it in its strength and in its weakness, its glory and its failure, because of this we can rededicate ourselves to the concern which is American democracy. Our way of life is on trial this fall," he concluded.

Panel on Positive Public Relations

Three members of the American Medical Association staff from Chicago participated in an afternoon panel session on "Reaching the Public Through Positive Public Relations."

Moderated by Dr. Joseph T. Ainsworth of Houston, chairman of the TMA's Council on Public Relations and Public Service, the panelists included Jim Reed, director of the AMA's Communications Division; Leo E. Brown, assistant to the executive vice-president of the AMA; and C. Darrell Coover, director of the AMA's Community Relations Department.

Discussing ways to develop good news media relations, Mr. Reed pointed out that sound PR is built of good works and sound communications practices.

Of the two kinds of PR programs—preventive and remedial—he emphasized that "the preventive course is the one to follow in PR as it is in medicine."

Preventive PR, he said, denotes a "long-range program with well-defined and established objectives. Its main purpose is to maintain continuous cordial relationships with your many publics by precluding any sources of misunderstandings or friction."

Basically, publicity is news, Mr. Reed told the doctors. "It is your job to find the news in your society and to present it in such form as will qualify it for publication or broadcast."

Getting acquainted with the editor of the newspaper and the managers of the radio and television stations was cited as perhaps the most important step in getting news disseminated.

In discussing methods of handling unfair attacks on the medical profession, Mr. Reed stressed maintaining good rapport with the news media.

"If your medical society has good programs in the public interest, if the physicians in your community are practicing good medicine, and if both are practicing good PR, you'll not have to worry too much about attacks on the medical profession," he said.

Mr. Brown outlined methods of setting up a speakers' bureau as part of county societies, which he called a "must" for every county medical society public relations, public education, or communications program.

After competent physicians have been recruited to make up the roster of the speakers' bureau, Mr. Brown urged that at least minimal training in the art of public speak-

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ing be provided. He also stressed the importance of promoting the speeches through news media, particularly if the host organization does not provide its own publicity.

In explaining the need for speakers' bureaus, Mr. Brown said: "Much has been written and said about the weaknesses and the shortcomings of the medical profession in recent years. A certain amount of such criticism is to be expected. No organization or profession which exerts the influence that medicine does can expect to exist in today's society without being attacked . . . Freedom of the press and freedom of speech are fundamental freedoms of which the medical profession must take advantage just as medicine's detractors do."

A sound community relations program and its importance was explained by Mr. Coover.

He urged that physicians and their wives become actively involved in community organizations in such a way that they will be able to exert genuine influence in these groups.

As active participants, they will be able to get speakers each year on health subjects and distribute literature from the county society or the AMA on various health subjects.

Mr. Coover also pointed out that within other organizations, groups may be encouraged to adopt needed health projects, misconceptions about the medical profession may be cleared up in informal conversations, positive resolutions on health matters may be initiated, and detrimental resolutions may be killed.

"All of you have been asked why the image of medicine is poor," Mr. Coover said. "In my opinion, all the ad campaigns and PR devices in the world will not do any good—unless MDs and their wives are effectively involved in every aspect of the community at all levels."

At the morning panel session, "New Federal and State Medical Programs and the Physician," Dr. Howard R. Dudgeon, Jr., of Waco, President-elect of the TMA, presided. Speakers included C. Lincoln Williston of Austin, TMA executive secretary; Dr. Charles Max Cole of Dallas, Speaker of the House of Delegates; Dr. John F. Thomas of Austin, chairman of the TMA's representatives to the State Coordinating Committee for Planning Regional Programs in Heart Disease, Cancer and Stroke; and Fred R. Higginbotham of Dallas, director of public relations for Blue Cross-Blue Shield of Texas.

Dr. Rouse To Be Headliner At Southern Medical Meeting

Dr. Milford O. Rouse of Dallas, President-elect of the American Medical Association, will speak at the president's luncheon of the Southern Medical Association, Nov. 15. The president's luncheon is one of the activities highlighting the annual meeting of the Southern Medical Association, convening this year in Washington, D.C., Nov. 14-17.

Other activities will include general symposia, 22 specialty section meetings, color television presentations, scientific exhibits, informal gatherings of alumni groups from various medical schools, and a dinner dance.

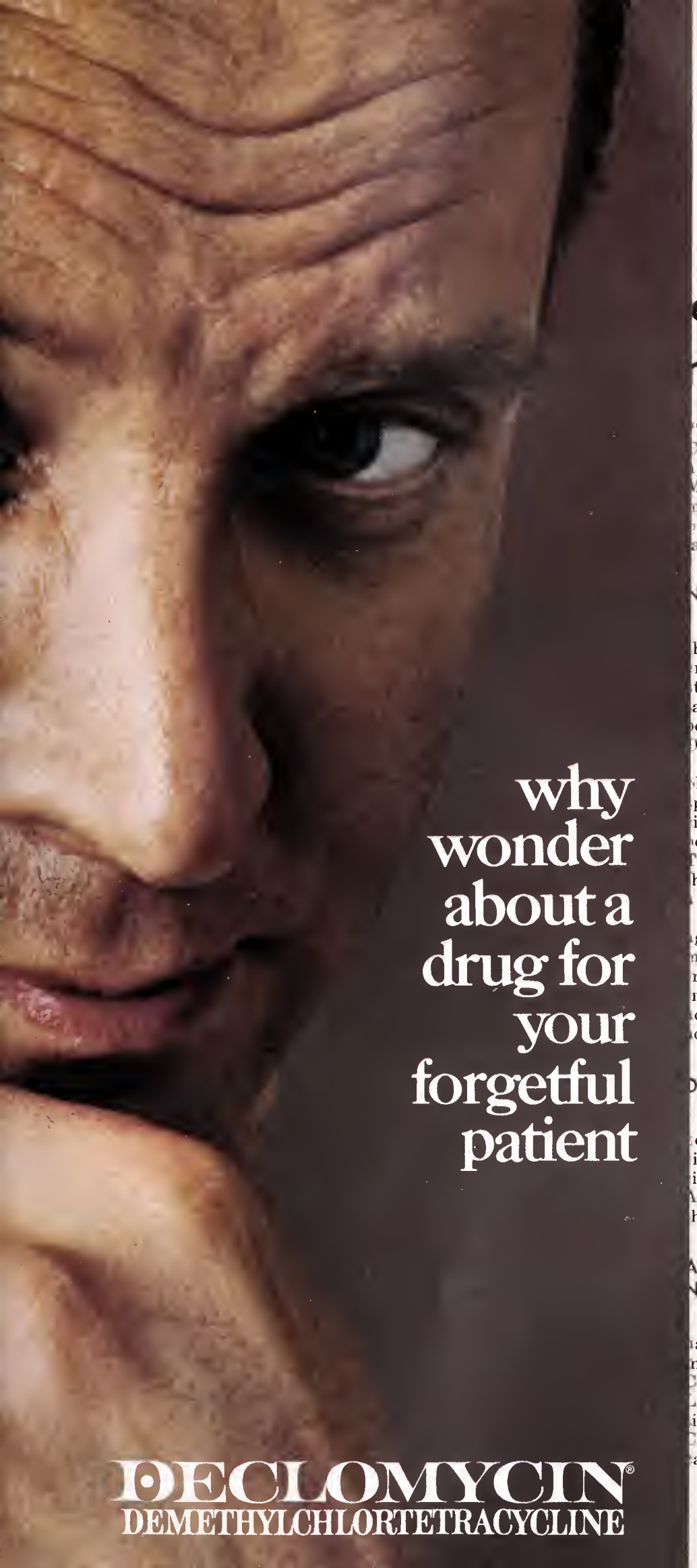
Meeting conjointly with the SMA will be the southern chapter of the American College of Chest Physicians, the College of American Pathologists, the Flying Physicians Association, the Radiological Society of North America, the Southern Gynecological and Obstetrical Society and the Woman's Auxiliary to the Southern Medical Association.

The SMA annual meeting will feature a large number of guest speakers. These guest speakers and the sections they will address include Dr. Lowell L. Henderson, of Rochester, Minn., consultant in internal medicine and allergy, allergy; Dr. Louis R. Orkin of New York City, chairman of the Department of Anesthesiology, Albert Einstein College of Medicine of Yeshiva University, anesthesiology; Dr. Carl T. Nelson, New York City, chairman of the Department of Dermatology, Columbia University College of Physicians and Surgeons, dermatology; Dr. Thomas C. Chalmers, Jr., of Boston, professor of medicine at Tufts University School of Medicine, gastroenterology; and Dr. Samuel L. Raines of Memphis, Tenn., chairman of the Department of Urology, University of Tennessee College of Medicine, general practice.

Other guest speakers and their sponsoring sections are Dr. James H. Nelson of New York City, director of Gynecological Tumor Service, Downstate Medical Center, State University of New York, gynecology; Dr. Leon J. Warshaw of New York City, medical director of Paramount Pictures and United Artists Corporation, industrial medicine and surgery; Dr. Mac Roy Gasque of New York City, corporate medical director of Olin Mathieson Chemical Corporation, neurology and psychiatry; Dr. Roy Hertz of Washington, D.C., professor of obstetrics and gynecology, George Washington University School of Medicine, obstetrics; Dr. John M. McLean of New York City, ophthalmology; and Dr. Donald B. Slocum of Eugene, Ore., assistant professor of orthopedic surgery, University of Oregon Medical School, orthopedic and traumatic surgery.

Also speaking to specialty sections will be Dr. W. Likely Simpson of Memphis, Tenn., professor emeritus of otolaryngology, University of Tennessee College of Medicine, otolaryngology; Dr. William David Tigertt of Washington, D.C., commanding officer at Walt Reed Army Institute of Research, pathology; Dr. Alan D. Perlmutter of Boston, instructor in surgery, Harvard Medical School, pediatrics; Dr. Donald L. Rose of Kansas City, Kan., chairman of the Department of Physical Medicine, University of Kansas Medical Center, physical medicine and rehabilitation; and Dr. James Barrett Brown of St. Louis, professor of clinical surgery, Washington University School of Medicine, plastic and reconstructive surgery.

Additional guest speakers in specialty sections are Dr. Thomas S. Moulding, Jr., of Denver, staff physician and coordinator, Tuberculosis Training Program, National Jewish Hospital, preventive medicine; Dr. Maus W. Stearns, Jr., of New York City, president of the American Proctologic Society, proctology; Dr. John A. Campbell of Indianapolis, Ind., chairman of the Department of Radiology, Indiana University Medical Center, radiology; Dr. Richard D. Brasfield of New York City, associate attending surgeon, Memorial Hospital, surgery; and Dr. Wyland F. Leadbetter of Boston, chief of urology, Massachusetts General Hospital, urology.



egislation, n Considered

"The Board of Trustees of the Community Health Centers shall appoint a duly licensed Doctor of Medicine as a director of the community center, if feasible." In the present Act, the director may be a lay person.

Nursing Home Accreditation

The Executive Board observed that nursing homes should be accredited and inspected by a single state agency and that an accreditation and inspection section should be set up within the Texas State Department of Health.

The Board further noted that a physician should direct each inspection team and that the physician does not necessarily have to be on the payroll of the State of Texas. It further recommended that a registered nurse should be a member of the inspection team.

Also recommended was encouragement of nursing schools and medical schools to provide for some training time to be spent in geriatric nursing and medicine in an accredited or approved nursing home or extended care facility.

Distinguished Service Award

Dr. G. V. Brindley, Sr., was nominated by the Board of Counselors for the Texas Medical Association's Distinguished Service Award which will be presented at the next Annual Session.

AMA Delegates To Nominate Dr. Tenery

Dr. R. Mayo Tenery of Waxahachie, who recently received an interim appointment to the AMA Council on Constitution and By-Laws, will be nominated for election to a full term at the 1966 Clinical Convention of the American Medical Association in Las

Vegas, Nov. 27-Dec. 1. Texas Delegates to the AMA will make the nomination, endorsed by the Executive Board.

Physicians Placement Service

After reviewing a thorough study of the physician manpower problem in Texas, the Trustees recommended redefining the objectives and organization of the Association's Physicians Placement Service in order to meet existing needs. The primary effort will be directed toward recruitment of more physicians for opportunities which are available, and more information will be circulated more often regarding the opportunities.

More staff time will be given to the service and to assisting committees concerned with physician manpower.

New Trust Fund

The Board of Trustees of the Association will serve as trustee of a new gift which will be available for loans to medical students and to individuals in paramedical fields, for bequests through the Physicians Benevolent Fund, and for other worthy support programs.

The new fund was the basis for the establishment of the Texas Medical Education and Research Foundation.

Disaster Medical Care

The Executive Board approved a resolution submitted by the Committee on Disaster Medical Care and the Council on Public Relations and Public Service which protested a proposed transfer of administration of the packaged disaster hospital program from the state to the federal government, bypassing the established state and and local civil defense health department, and medical society functions with regard to the program.

The Board agreed that the program could better function with the coordination of the Texas State Department of Health, the Texas Department of Public Safety, the Governor's office, and other Texas agencies.

Dr. Gill Commended

Dr. A. J. Gill, dean of the University of Texas Southwestern Medical School, who has recently extended his resignation as dean to return to full-time teaching, was commended by the Board for his many accomplishments at the school.

Opposition To Price-Anderson Bills

The Executive Board heard the recommendation of the Committee on Nuclear Medicine that the Association should oppose H.R. 16920 and S. 3722 on Uniform Radiation Exposure-record Keeping and Federal Workmen's Compensation Standards. Instead, the committee suggested that, in lieu of federal legislation, other measures such as an educational program for employers and employees regarding radiation monitoring and general safety programs, development of uniform workmen's compensation laws at the state level and careful follow-up-observation of personnel known to be involved in radiation accidents would be helpful.

The recommendation was referred to the Council on Medical Jurisprudence.

Tuberculosis

The Committee on Tuberculosis pointed out the importance of chest x-rays of patients on, or shortly before, admission to hospitals and nursing homes, and recommended that the information be transmitted to the Texas Hospital Association.

UND Form 1

The Council on Medical Service and Insurance noted its approval of UND Form 1 provided by the Health Insurance Council as a standard form for health insurance claims. The Council noted that if a member does not wish to use this form, he may attach a narrative report, charging a reasonable fee, and this will be acceptable to the insurance companies.

Separation of Fees

The Council on Medical Service and Insurance lent its full support to the program of separation of fees from hospital billing and called upon cooperation of the health insurance industry to provide all policyholders, at the time of contract renewals, with adequate information on the meaning of separation of fees, noting which policies provide for separation of fees and which do not, and to make such policies available.

The Board of Councilors noted that it is proper and preferable to effect the separation of professional fees from hospital bills and recommended the institution of an educational program immediately. The Councilors noted that, ethically, no new contracts should be written which do not afford the separation of fees by all physicians who are presently receiving remuneration from hospitals for professional services rendered. They urged immediate renegotiation of all present contracts which do not afford separation of professional fees and reaffirmed their position, noting that they did not object to a physician's bill being collected by employees of a hospital provided that there was no merging of hospital and doctor's bills, that the method of billing clearly stated to whom the charge was owed, and that no fee was charged for collection other than the actual cost to the hospital.

The Councilors reiterated the decision of the AMA Judicial Council that it is unethical for a physician to participate in commercial directories outside the profession. Councilors noted also that use of medical credit cards by patients is unethical.

The Councilors reminded members of the Association that the use of the term "Admission Fee" on itemized bills is misleading inasmuch as a fee for the mechanics of obtaining admission of a patient to a hospital is unethical. A charge may be made for history and physical examination, initial medical treatment, and such phases of treatment which more clearly designate that all of the services are medical or professional.

"A victim of mouth-to-mouth resuscitation."

Commemorative Painting To Highlight 1967 Centennial Annual Session

A painting of the first meeting of the Texas Medical Association will be a central feature of special celebration activities at the Centennial Annual Session in Dallas May 4-7, 1967.

The painting, to be produced by artist Robert A. Thom who painted the founding of the American Medical Association, will depict the first meeting of TMA. Parke-Davis & Company will defray costs of the original oil painting. The painting is expected to be approximately three feet by four feet and will be displayed during the 100th Annual Session. Later, it will be hung in the Headquarters Building.

Consideration is being given to having prints of the painting made. These prints could be distributed to all members of the Association, or to all in attendance at Dallas as a memento of the 100th meeting.

Special Centennial Features

The format of the 1967 meeting is similar to that followed for the 1966 Session, but several features have been added to commemorate the Centennial Session.

A centennial emblem has been developed which has been printed on TMA stationery and envelopes. "Stick-on" type foil labels displaying the emblem have also been printed. Journal covers, brochures, and other items will display the emblem throughout the centennial period.

A special historical issue of *Texas Medicine* will be developed for March, 1967. Included in this issue will be special articles on the first meeting of TMA, highlights of the 100 annual meetings of the Association, historical articles on related groups, and a special journal cover.

The *Dallas Morning News* and the *Dallas Times Herald* will devote special editions to the 100th Annual Session on Sunday, April 30 just prior to the Session's opening.

An abbreviated history of TMA and other historical highlights will be placed in the Annual Session printed program.

County medical societies will be asked to honor Texas residents who attain age 100 during the centennial year. Wherever possible, the centenarian's private physician will be invited to attend the ceremony.

The Committee on Medical History is planning a special dinner program during the 1967 Session to honor outstanding persons in Texas medicine.

Special Programs

In addition to the special centennial features, several special programs are being planned for the Dallas Session.

Smith Kline & French Laboratories will present four medical color television programs during the Session. Dr. Vernie A. Stembridge of Dallas, as television chairman, will supervise and arrange the scientific content of the programs.

Ten Curbstone Consultations roundtable discussions have been planned. Topics of these discussions will be (1) stroke, (2) skin tumors—clinical diagnosis and treatment, (3) cancer of the thyroid, (4) management of thromboembolic disease, (5) sensitivity chest disease, (6) respiratory insufficiency, (7) adolescent amenorrhea, (8) management of chronic infantile diarrhea, (9) management of the post-coronary patient, and (10) evaluation of the patient complaining of sinus disease.

United Cerebral Palsy of Texas, Inc., will sponsor a Symposium and luncheon on Saturday, May 6. A program similar to that of the 1966 session, "Children's Learning Problems Due to Neurologic Dysfunction," will be given at the 1967 session.

The American Cancer Society, in conjunction with its Executive Board meeting and the TMA Annual Session, will offer a cancer conference entitled "Current Concepts in Etiology and Diagnosis of Cancer" on Wednesday, May 3.

The Texas Academy of General Practice will sponsor a seminar on Thursday, May 4.

The Texas Commission on Alcoholism will offer a Symposium on Alcoholism on Thursday, May 4.

The TMA Committee on Cardiovascular Diseases in cooperation with the Texas Heart Association will conduct a Symposium of Cardiovascular Diseases on May 4.

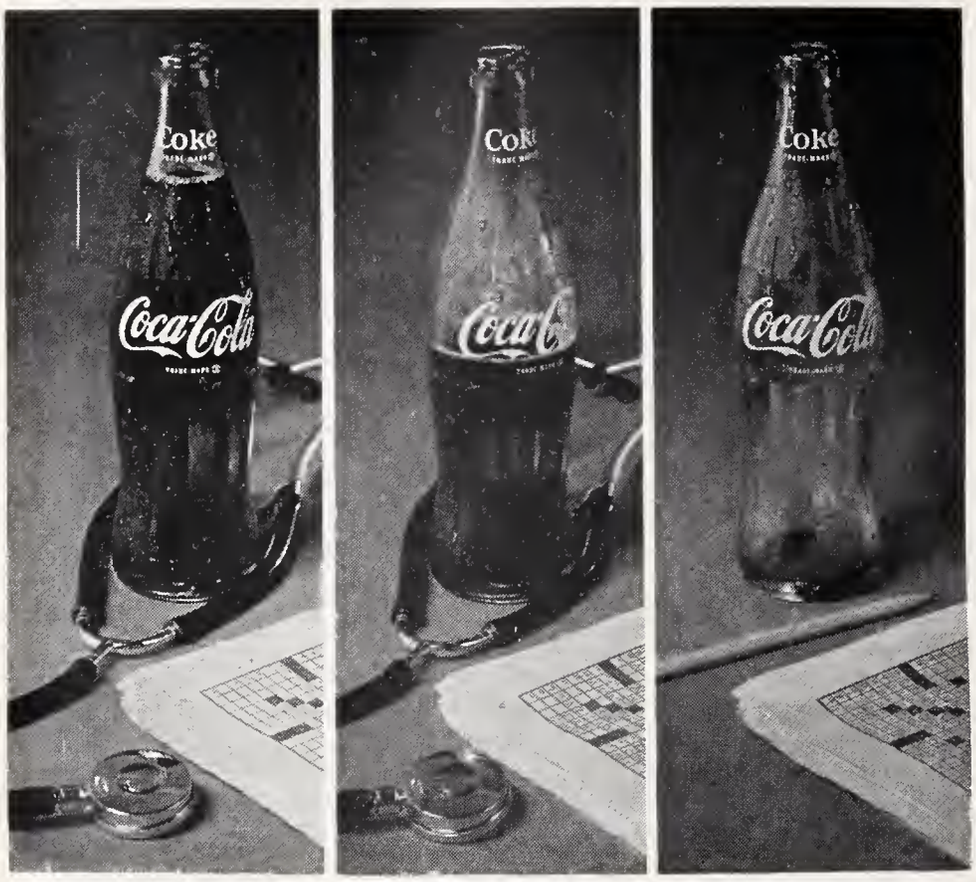
The TMA Committee on Nuclear Medicine is planning a Seminar on Food Preservation and Irradiation on Friday, May 5.

The Texas Nurses Association and the TMA Committee on Nursing have outlined plans for the First Texas Conference on Physicians and Nurses.

The TMA Committee on School Health will sponsor a conference on Physicians and Schools, Friday, May 5.

Housing Accommodations

TMA has made housing commitments for more than 1,700 first-class hotel and motel rooms. The following facilities have rooms reserved for those attending Annual Session: Adolphus Hotel, Statler-Hilton Hotel, Sheraton-Dallas Hotel, Baker Hotel, Marriott Motor



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Dode Mae Hanke, M.D.
Maurice S. Green, M.D.
Doyle I. Carson, M.D.
Stanley L. Seaton, M.D.
Thomas H. Allison, M.D.

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Mary Russell, B.S., Music

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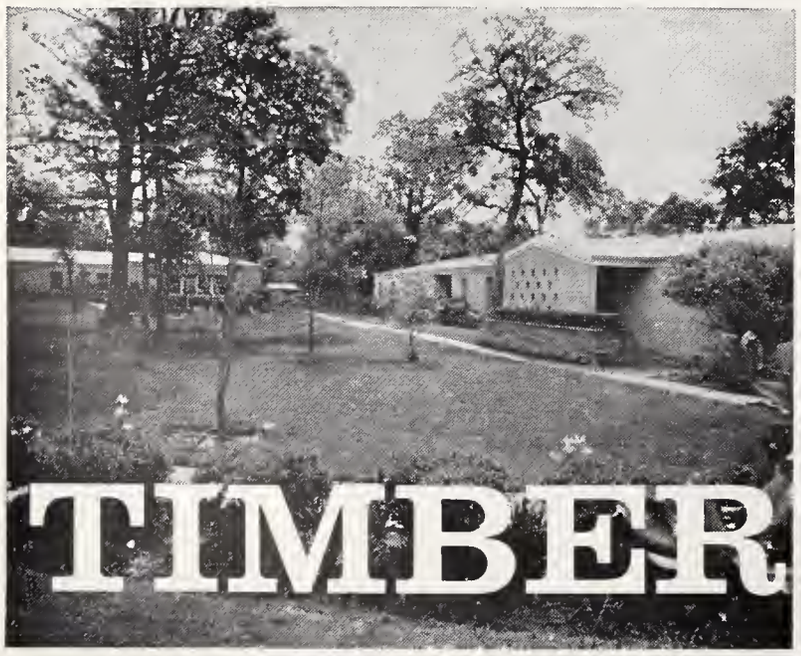

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Housing procedure will be the same as has been established in the past. Physicians and guests will be invited to write directly to the hotel or motel of choice. Facilities will be asked to confirm reservations directly. When the hotel/motel exhausts its allocation of rooms, it will refer requests to the Dallas Convention and Visitors Bureau. The bureau will assume the responsibility of securing reservations in suitable facilities for the individuals involved.

Session Headquarters

Scientific meetings and exhibits will be concentrated at the Dallas Memorial Auditorium. Some scientific sessions will be placed in the Adolphus Hotel, headquarters hotel for scientific meetings.

The Statler-Hilton Hotel has been designated headquarters for business sessions. All House of Delegates' sessions, and board, council, and committee meetings will be scheduled in that hotel.

The Sheraton-Dallas Hotel has been designated Auxiliary Headquarters.

Exhibits will be quartered in the Exhibit Hall of the Memorial Auditorium. A cocktail party for exhibitors will be held on Thursday, May 4.

Walter Jetton of Fort Worth will cater luncheons in the Auditorium on Friday and Saturday, May 5 and 6.

Twenty-nine out-of-state speakers have been secured with five or six more expected.

Orthopedic Surgeons Plan Dallas Trauma Symposium

The Committee on Injuries of the American Academy of Orthopedic Surgery in cooperation with the University of Texas Southwestern Medical School will sponsor a second Symposium on Trauma in Dallas, Nov. 2-5.

Registration fee for the course is $75 which includes lunches and one evening's entertainment. Headquarters for the symposium will be the Marriott Motor Hotel.

Out-of-state participants on the program will include Dr. Marcus J. Stewart of Memphis, Tenn.; Dr. Ian MacNab of Toronto, Canada; Dr. J. Neill Garber of Indianapolis, Ind.; Dr. Edwin F. Cave of Boston; and Dr. William A. Larmon of Chicago.

Faculty members of the University of Texas Southwestern Medical School participating in the symposium program will include Dr. A. J. Gill, Dr. Charles F. Gregory, Dr. Vernon M. Bryant, Dr. Russell B. Graham, Dr. Harold A. Mattson, Dr. Edward R. Richardson, Dr. Marvin P. Knight, Dr. Livius L. Lankford, Dr. F. Leon Ware, Dr. Adolph H. Giesecke, Dr. G. Thomas Shires, Dr. Charles R. Baxter, Dr. Robert N. McClelland, Dr. William A. Cook, Dr. Malcolm Perry, Dr. William C. Garre, Dr. Robert G. Grossman, Dr. Robert V. Walker (D.D.S.), Dr. William C. Head, Dr. Jack Reynolds, Dr. Ronald C. Jones, Dr. G. Truett James, Dr. Louis H. Paradies, and Dr. L. Ray Lawson.

Participants from other Texas medical faculties include Dr. Frank Parrish of Baylor University College of Medicine in Houston and Dr. E. Burke Evans of the University of Texas Medical Branch in Galveston.

1966 AMA Clinical Convention Set Nov. 27-30 in Las Vegas

The American Medical Association's Clinical Convention, the largest educational enterprise of its kind, will open its 20th annual meeting Nov. 27 and continue through Nov. 30 in Las Vegas, Nev.

More than 12,000 persons are expected to attend, including 5,000 physicians.

Discussion topics at Las Vegas will include "The Problem and Potential of LSD," the controversial drug, and "An Agonizing Reappraisal of Cancer Chemotherapy," an assessment of the effectiveness of drugs in treating various kinds of cancer. Doctors will discuss these and other topics at breakfast round tables, before the day's regular teaching sessions begin.

Scientific presentations will be given on 18 specialized topics, including juvenile diabetes, aerospace medicine, psychiatry, pediatrics, and diagnostic and therapeutic tools of the future. Scientific and industrial exhibits will be displayed in the newly expanded Las Vegas Convention Center. Closed-circuit color television programs and more than 25 medical motion pictures will be presented. The AMA's House of Delegates also will meet.

The Eighth National Conference on the Medical Aspects of Sports will be held in conjunction with the Clinical Convention. Sponsored by the AMA, the day-long program will include discussions of problems faced by team physicians at all levels of athletic competition. The Conference will be Sunday, Nov. 27, at Caesar's Palace.

Gastroenterology Researchers To Gather Nov. 4 in Chicago

The program of the Gastroenterology Research Group fall meeting will feature the study, "Animal Models in the Study of Gastrointestinal Diseases of Man." The meeting will take place on Nov. 4 at the Drake Hotel, Chicago.

Subjects to be covered by program speakers include metabolic aspects of digestion in the ruminant, transmissible gastroenteritis in swine, granulomatous colitis in the boxer, and intestinal disaccharidase deficiency in the California sea lion.

Medical Women To Convene In Washington Nov. 2-5

"Sexual Problems in Clinical Practice" will be the subject of the Scientific Symposium during the annual meeting of the American Medical Women's Association (AMWA) in Washington, D.C., Nov. 2-5.

The physicians will install Dr. Elizabeth A. McGrew of Chicago as the 1967 AMWA President, succeeding Dr. Margaret J. Schneider of Cincinnati. Dr. Mary E. Sears of Houston is currently serving AMWA as councilor.

International Inn, 14th & M Sts., NW, is the headquarters hotel for the business and scientific meetings. A tour of the White House, a reception and banquet, and a dinner-theater party are planned.

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Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

Indications: DIUTENSEN-R may be employed in all grades of essential hypertension. **Dosages:** Usual dose is 1 tablet twice daily, at morning and evening meals. However, adjustment of dosage to suit individual circumstances may be required. Please refer to package insert for full particulars. **Side effects and precautions:** The side effects observed with patients on DIUTENSEN-R have been of a mild and nonlimiting nature. These include occasional urinary frequency, nocturia, nasal congestion, muscle cramps, skin rash, joint pains due to gout symptoms and nausea and dizziness which have been reported for the individual components. Most of these symptoms disappear while the drug is continued at the same or lower dosage level. The concomitant use of digitalis and DIUTENSEN-R may increase the possibility of digitalis-like intoxication. If there is evidence of myocardial irritability (extrasystoles, bigeminy or AV block), dosage of DIUTENSEN-R should be reduced or discontinued. Nocturia in patients with marginal cardiac status and salt and fluid retention can be effectively controlled by limiting the time of administration to early afternoon. DIUTENSEN-R should not be used in patients with a known intolerance to reserpine. Package inserts furnish a complete summary of recommended cautions related to each of the ingredients of DIUTENSEN-R.

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Considine, Ball Set To Speak At Nursing Home Convention

"The Future Belongs To Those Who Prepare" will be the theme of the 16th Annual Convention of the Texas Nursing Home Association in November. Approximately 700 persons will attend the meeting Nov. 9-12 at the Hotel Texas in Fort Worth.

Harold Baumgarten, professor of administrative medicine, School of Public Health, of Columbia University will use the meeting theme as his keynote address topic. Other featured speakers will be newspaper columnist and radio personality Bob Considine and Social Security Administration Commissioner Robert M. Ball.

Medicare and nursing home participation will be a topic of speakers from the state certifying agency; the Office of Equal Health Opportunities; regional office of the Social Security Administration; and the fiscal intermediary, Mutual of Omaha. Other speakers in the program will discuss such legislative matters as minimum wage and unemployment compensation legislation, the medical vendor program for welfare patients, in-service training, medical records and charts, insurance programs, and other topics.

The American Nursing Home Association will hold its convention Nov. 3-5 at the Chase Park Hotel in St. Louis. For further information write Mr. Al Ercolano, Executive Director, American Nursing Home Association, 1101 17th St. NW, Washington, D.C.

Academy of Pediatrics Plans National Meeting in Chicago

More than 4,500 pediatricians, their families, and guests, are expected to attend the 35th annual meeting of the American Academy of Pediatrics, Oct. 22-27 in Chicago.

The meeting, to be held in the Palmer House, will feature a diverse scientific program, 26 roundtable discussions, 11 seminars, and 164 technical and scientific exhibits.

Subjects to be covered in scientific papers include growth hormone and disorders of growth, society's stake in responsible parenthood, clinical trial of live attenuated rubella virus vaccine, the unmet needs of children, and language development and disorders.

Papers will be presented during general session meetings, and during meetings of the sections on surgery, diseases of the chest, cardiology, military pediatrics, anesthesiology, child development, and the committee on urology.

Chest Physicians To Convene

The American College of Chest Physicians will conduct its interim meeting Nov. 26-27 at the Flamingo Hotel in Las Vegas.

Program items include presentation of scientific papers, roundtable luncheons, and fireside conferences. All physicians are invited to attend; however, there is a $15 registration fee for non-members of the college.

Mental Health Meeting Set

The Texas Association for Mental Health will hold its annual conference and delegate convention Oct. 27-29 at the Commodore Perry Hotel in Austin.

For additional information, contact Mr. R. W. Shafer, Jr., Executive Secretary, 4004 N. Lamar Blvd., Austin.

Cytology Society To Meet

The American Society of Cytology will hold its 14th annual scientific meeting Nov. 10-12 at the Hotel Pfister, Milwaukee, Wis.

Further information can be obtained from Dr. Warren R. Lang, Secretary-Treasurer, 1025 Walnut St., Philadelphia 19107.

Auxiliary Speakers Stress Need for Medical Personnel

"Excellence Equals Enthusiasm Times Efficiency Squared" was the School of Instruction theme of the Woman's Auxiliary meeting Sept. 30. The school was preceded by an Executive Board meeting on Sept. 29.

Four speakers stressed the urgent need for more medical personnel. Dr. Robert Moreton of Houston, chairman of the TMA Advisory Committee to the Woman's Auxiliary, stated that within the next 20 years Texas will become the third largest state and will need more doctors and nurses to serve the population adequately. Mr. C. Lincoln Williston of Austin, TMA executive secretary, said that Medicare will cause a greater need for more general medical doctors and nurses. Mrs. R. C. L. Robertson of Houston, chairman of the AMA Auxiliary's Committee on Rural Health, stressed recruitment for medical careers. Dr. James D. Murphy of Fort Worth, TMA President, also stressed the immediate need for greater medical manpower.

Two non-medical speakers highlighted the programs. Mr. Paul Boesch of Houston, author and professional wrestler, spoke on "The Womanly Art of Self-Defense." Col. Vincent I. Hack, information officer of Brooke Army Medical Center, San Antonio, spoke on "The Psychology of Color."

Eight Medical Assistants Gain National Certification

Eight Texas medical assistants have recently received national certification after taking an examination in June. This addition to the number of Texas certified medical assistants gives Texas a total of 16 who have received certification.

Those who have received certification since the June examination and the doctors for whom they work are Pauline Coplin of Lubbock, employed by Dr. William G. Evans; Mildred Crawford of San Antonio, employed by Drs. Albert W. Hartman, William J. Hills, and William L. Sammis; Vera St. John Crim of Waco, employed by Dr. C. G. Catto; Thelma Cullins of Midland, employed by Drs. Joe B. Hilliard and Ralph G. Greenlee; Elizabeth F. Goodman of El Paso, employed by Dr. L. C. Feener; Alice M. Staples of Marshall, employed by Dr. Richard G. Granbery; Lotena M. Van Deventer of Marshall, employed by Dr. J. P. Graves; and Gladys T. Vance of Bedford, employed by Drs. Aubrey E. Guthie II, and Philip Sheinberg of Hurst.

First *Texas Medicine* Award Given at Fort Worth Meeting

The first annual *Texas Medicine* award for scientific writing by a Texas medical student was presented to Dr. Charles F. Johnson III, an intern at John Peter Smith Hospital in Fort Worth, at a meeting of Tarrant County Medical Society Tuesday night, Oct. 4.

DR. JOHNSON

The award was given to him for an article written while he was a medical student at the University of Texas Medical Branch, Galveston.

Dr. Johnson was awarded a $300 check by Dr. James D. Murphy, President of the Texas Medical Association, and Dr. James A. Hallmark, a member of the *Texas Medicine* Board of Publication.

Dr. Johnson received his award for excellence of scientific work and competence in the writing craft, based upon an article he had written under the direction of Dr. Charles T. Ashworth, chairman of the Department of Pathology at the University of Texas Southwestern Medical School, Dallas. At the time, he was studying under a grant from Dr. F. L. Jennings, chairman of the Department of Pathology at the University of Texas Medical Branch.

The winning article, "The Effects of Acute Ethanol Intoxication on Hepatic Protein Synthesis: A Biochemical and Morphologic Study," will be published in the November issue of *Texas Medicine*.

The award was established this year by the Board of Publication and the Journal Advisory Committee for *Texas Medicine* to encourage student excellence in scientific writing. Members of the Journal Advisory Committee, served as judges in the competition. The award is again being offered medical students attending Texas Medical schools. Entry deadline for the new award is April 1, 1967.

Dr. Johnson's mother, Mrs. Norma F. Johnson, a registered nurse, serves as a school nurse in Karnes City, Texas. Formerly of San Antonio, Dr. Johnson completed high school and junior college in Victoria, and received a bachelor of arts degree in zoology from the University of Texas, Austin, in 1962, before entering medical school.

Personals

Dr. Edward R. Thompson of Galveston recently donated a rare collection of early-day surgical instruments to the University of Texas Medical Branch. The instruments were owned by the late Dr. James E. Thompson who was a member of the founding faculty at the Medical Branch and served as professor of surgery there from 1891 until his death in 1927. The instruments were donated in memory of the late Dr. John Thompson, a brother of Dr. Edward Thompson and a practicing physician in McGregor. Dr. John Thompson, who died in July, had inherited the collection when his father died.

Dr. Truman G. Blocker, Jr., left, executive director, University of Texas Medical Branch, receives a rare collection of early-day surgical instruments from Dr. Edward R. Thompson, Galveston. The gift to the Medical Branch was in memory of the late Dr. John Thompson of McGregor.

Dr. Truman G. Blocker, executive director and dean of the Medical Branch, said he was unable to place a value on the instruments, but believed the well preserved instruments "might possibly be 100 years old." Dr. James Thompson had acquired the instruments from a Dr. William Schmoeller who died in 1917 and had been a resident of Houston.

Dr. James E. Thompson had four sons to graduate from the University of Texas Medical Branch and become doctors. The other two, besides John and Edward, are Dr. James E. Thompson, Jr., and Dr. Frederick R. Thompson, both of New York. A grandson, Dr. Peter Kempner Thompson, the son of Dr. and Mrs. Edward Thompson, completed his medical studies at the Medical Branch in 1964 and is now practicing in San Francisco.

Dr. Don W. Micks (Sc.D.) has been appointed chairman of the Department of Preventive Medicine and Public Health at the University of Texas Medical Branch. He had served the department as acting chairman since 1961. He joined the faculty as assistant professor in 1949 and became a full professor in 1959.

Dr. Robert K. Bing, director of occupational therapy at the University of Texas Medical Branch, has been assigned additional duties as planning coordinator for the proposed School of Allied Health Sciences at the Medical Branch. The formative program at Galveston has been made possible by a US Public Health Service grant to study the nation's burgeoning regional medical programs against heart disease, cancer, and stroke.

Dr. Carl F. Shaffer of San Antonio has been appointed Director of Medical Education for the Santa Rosa Medical Center in San Antonio. **Dr. Wade H. Lewis** has received appointment as chairman of the Department of Psychiatry of the Santa Rosa Medical Center.

Dr. Joseph G. Murphy of Galveston has been unanimously elected to the Supreme Council of the Knights of Columbus.

Dr. Robert E. Sullivan, Jr., of La Marque was named "Boss of the Year" by members of the Galveston County Medical Assistants Society at their Annual Bosses' Night Banquet in September.

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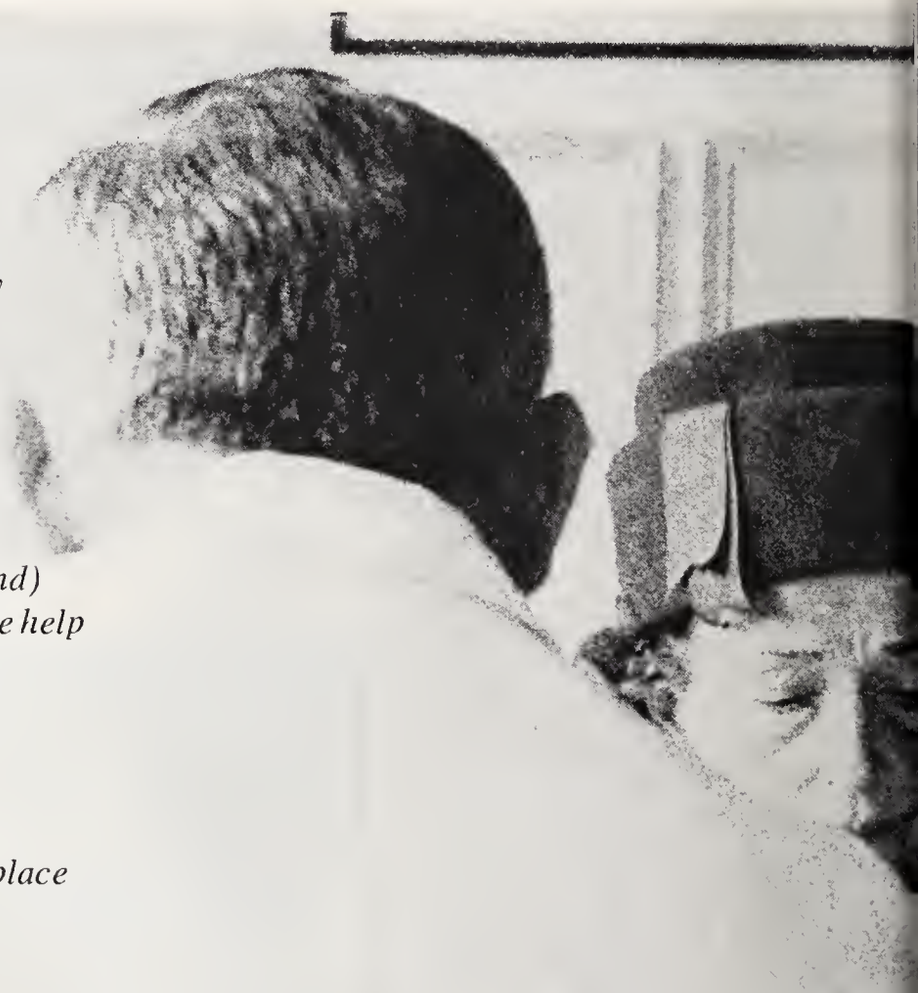

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McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

"Mediatric (steroid-nutritional compound) capsules, one a day, seem to give definite help to debilitated patients."

Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

"Nutritional and hormone bolstering of function in the aged may have a useful place in geriatrics."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"In diets which for any reason are restricted in calories, enough of these substances (B vitamins) may not be supplied . . . The use of B and C vitamin supplements may then be justified and indeed may be necessary."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"Intensive nutritional therapy is necessary, especially in elderly people, to correct dietary deficiencies created by large losses of protein, vitamins and other nutrients."

Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.

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Thiamine mononitrate	10.0 mg.
Riboflavin	5.0 mg.
Niacinamide	50.0 mg.
Pyridoxine HCl	3.0 mg.
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Ferrous sulfate exsic.	30.0 mg.
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*Orally active, water-soluble conjugated estrogens derived from pregnant mares' urine and standardized in terms of the weight of active, water-soluble estrogen content.

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Dr. R. Lee Clark, director, University of Texas M. D. Anderson Hospital and Tumor Institute, begins committee report to representatives of news media at press conference in Texas Medical Association Headquarters Building on Sept. 8. The distinguished committee of 32 was appointed by Gov. John Connally to study the medical aspects of the Charles J. Whitman shooting incident from the University of Texas Tower which took place Aug. 1.

Governor's Committee Recommends Health, Safety Measures for UT Campus

IN THE AFTERMATH of the Whitman case, 32 Texas and nationally recognized "blue-ribbon" M.D.'s and Ph.D.'s quietly, upon appointment, put their heads together to study the specific case itself and to view the whole problem—the reasons that such tragedies occur and the prevention of future cases.

Gov. John Connally issues statement concerning committee report at conference.

Out of their study came several proposals for improvement of campus health care and safety measures. These were submitted to Gov. John Connally by a committee he had appointed to study the circumstances surrounding the Aug. 1 University of Texas shooting incident involving Charles J. Whitman.

The committee of 32 included these recommendations in a report released to the public at a Sept. 8 press conference held at Texas Medical Association Headquarters Building in Austin. A final pathologic diagnosis of the physical condition of Whitman and conclusions reached by the group regarding the possible origins of Whitman's behavior were revealed.

Recommendations Made

Emphasizing that the recommendations specifically referred to the University of Texas, the consultants stated that the general principles could well apply to all colleges and universities. To aid health and safety at the University of Texas, they recommended:

1. Development of a University health program for students and faculty. Responsibility for development and plans for such a program for all divisions of the university would be placed in the hands of the Vice-Chancellor for Health Affairs.

2. Development of a mental health program for students and faculty. All appropriate university resources should be utilized in planning such a program, including perhaps the Hogg Foundation.

3. Expansion and strengthening of a student counseling service. Each student should be provided counseling of such depth and scope as to achieve the personalization of his abilities and educational and life goals.

4. Study and strengthening of safety factors within the University of Texas System. Safety factors of the individual campuses would be studied in order to anticipate and devise solutions to problems that may arise. Necessary additional laws for protective measures should be introduced at the next session of the Texas Legislature. A study should be made of safety measures for the continued use of the Tower building on the main University of Texas campus.

The committee also made some general recommendations.

Committee members felt that an Office of the Medical Examiner should be established on a statewide basis. Studies of the effects of news media in the presentation of violence in human behavior, relearning process for combat-trained military personnel, and brain function as related to violent and aggressive behavior were recommended. Other recommendations concerned aid to victims and commendation of those who had acted heroically on the occasion.

Pathologic Study

The members of the group were provided with the following material which formed the basis of the pathologic study conducted at the University of Texas M. D. Anderson Hospital and Tumor Institute: (1) Autopsy protocol including the microscopic findings; (2) various specimens of brain tissue; (3) paraffin block containing two pieces of brain tumor; (4) two stained and two unstained sections of brain tumor; (5) two kidneys; (6) a slice of liver with the gallbladder attached, and (7) the stomach.

Five factors necessarily limited the examination in depth and scope. (1) The autopsy was not performed until approximately 24 hours after death. (2) The body had received arterial and trocar embalming before the initial examination. (3) Many parts of the brain were damaged by the penetrating fragments of bone which had been created by the gunshot wounds. (4) All of the pieces of the brain were not recovered for the examination. (5) The brain had been sectioned at the time of the autopsy.

The final pathologic diagnosis was as follows:

1. Multiple contusions and lacerations of brain, with bilateral focal hemorrhages in the frontal, parietal and temporal lobes, with multiple small fragments of bone in the white and gray matter (history of multiple gunshot wounds of the head and face).

2. Recent subarachnoid hemorrhage of both frontal and parietal lobes and the pons.

3. Recent multiple contusions of white matter in the frontal, parietal and temporal lobes, and the brain stem.

4. Glioblastoma multiforme (sections of two pieces of tumor reportedly removed from the right temporo-occipital white matter by Dr. Coloman de Chenar on Aug. 2).

5. Flattening of cerebral convolutions, slight.

6. No significant pathologic change in liver, kidneys, and stomach.

7. Multiple wounds of the body.

The malignant tumor removed from the brain by Dr. de Chenar microscopically exhibited the features of a glioblastoma multiforme with a remarkable vascular component of the nature of a small congenital vascular malformation. It contained widespread areas of necrosis with palisading of cells characteristic of glioblastoma multiforme.

No evidence of other disease or previous trauma was observed but gunshot wounds destroyed so much of the brain that examination of all the major nerve tracts and nuclei was impossible.

Specialists at the Armed Forces Institute of Pathology in Washington, D.C., reviewed formalin-fixed and embalmed specimens of tissue from the brain, kidney, stomach, and liver for toxicological analysis. There were no findings of basic drugs in the kidney, stomach, or brain; no findings of barbiturates in the liver; no findings of neutral drugs in the kidney.

The data obtained provide no evidence of clinical neurological abnormality, and there is no evidence from the pathological reports that the tumor interrupted pathways leading to detectable neurological signs.

Psychiatric Study

Consultants on the psychiatric portion of the study were concerned with arriving at reasonable conclusions as to the possible origins of Whitman's behavior within the limitations imposed by the lack of opportunity to interview the subject himself. The group reviewed all available written material, including police records, service records, school records, personal diaries and letters, and interviews with Whitman's friends and acquaintances, and press reports.

A summary of the psychiatric findings follows:

Charles J. Whitman was an intelligent, intense, driven young

Dr. William H. Sweet, professor of surgery, Harvard Medical School, reports committee recommendations developing from study of the Whitman tragedy.

Listening to the committee report is committee member Dr. Kenneth M. Earle, chief, Neuropathology Branch, Armed Forces Institute of Pathology. Dr. Earle is a Texan who was formerly dean of the University of Texas Medical Branch.

man living under conditions of increasing personal stress from which he felt he could not escape or master. He experienced profound personal dissatisfactions, seeming to have a poorly formulated image of himself resulting in a deep sense of unrest. He was prone to impulsive action and loss of control at times. He had not chosen academic pursuits for which he was best equipped and experienced much difficulty in the abstraction of ideas and organization of his studies. He was deeply concerned over the marital discord and recent separation of his parents, having strong inconsistent feelings of hostility toward members of his family, especially his father. He was known to express concern about physical symptoms frequently, but he was not known to have consulted a physician about these symptoms in the last four months of his life.

"It is the opinion of the task force that the relationship between the brain tumor and Charles J. Whitman's actions on the last day of his life cannot be established with clarity. However, the highly malignant brain tumor conceivably could have contributed to his inability to control his emotions and actions. Without a recent psychiatric evaluation of Charles J. Whitman, the task force finds it impossible to make a formal psychiatric diagnosis."

Committee Members

Members of the committee and invited consultants are as follows:

From the University of Texas M. D. Anderson Hospital and Tumor Institute in Houston: Dr. R. Lee Clark, director, surgeon-in-chief, and professor of surgery; Dr. Joseph A. Jachimczyk, senior consultant in forensic pathology; Dr. Tate M. Minkler, assistant pathologist and medical systems analyst; Dr. Robert D. Moreton, assistant to the director and professor of radiology; and Dr. William O. Russell, head, Department of Pathology, and chief, the Section of Anatomical Pathology.

From the University of Texas Medical Branch at Galveston were Dr. Donald Duncan (Ph.D), chairman, Department of Anatomy; and Dr. Hamilton Ford, chairman, Department of Neurology and Psychiatry.

From the University of Texas Southwestern Medical School in Dallas: Dr. Frank Harrison, professor of anatomy and associate dean; and Dr. Robert L. Stubblefield, chairman, Department of Psychiatry.

From Baylor University College of Medicine in Houston: Dr. Stuart L. Brown, assistant professor of psychiatry; and Dr. Shervert H. Frazier, Jr., chairman, Department of Psychiatry.

From the Main University of Texas campus at Austin: Dr. Maurice D. Heatly, staff psychiatrist, Student Health Center; Dr. Ira Iscoe (Ph.D.), professor of psychology; Dr. Gardner Lindzey (Ph.D.), chairman, Department of Psychology; Dr. Jack Otis (Ph.D.), professor and director, Graduate School of Social Work; Dr. Robert Sutherland (Ph.D.), Hogg Professor of Sociology and director, Hogg Foundation for Mental Health; and Dr. Paul L. White, director, Student Health Center.

From Austin: Dr. Coloman de Chenar, pathologist, Austin State Hospital.

From out-of-state: Brig. Gen. Joe M. Blumberg (M.D.), director, Armed Forces Institute of Pathology, Washington, D.C.; Dr. Kenneth M. Earle, chief, Neuropathology Branch, Armed Forces Institute of Pathology; Dr. Fred Elmadjian, (Ph.D.), chief, Biological Sciences Section, Behavioral Sciences Training Branch, National Institute of Mental Health, Bethesda, Md.; Dr. Jack R. Ewalt, director, Massachusetts Mental Health Center, Boston; Dr. Dana L. Farnsworth, superintendent of university health services, Harvard University, Cambridge, Mass.; Dr. Justin M. Hope, psychiatrist to the Wiswall Hospital, Wellesley, Mass.; Dr. Horace W. Magoun (Ph.D.), professor of anatomy, University of California at Los Angeles; Dr. Jeff Minkler (Ph.D.), associate clinical professor of pathology, University of Colorado Medical School; Dr. Alan R. Moritz, provost, Western Reserve University, Cleveland, Ohio; Dr. Augustus Rose, professor of neurology, University of the City of Los Angeles; Dr. Paul I. Yakovlev, clinical professor emeritus of neuropathology, Harvard University Medical School; and Dr. Harry M. Zimmerman, chief, Laboratory Division, Montefiore Hospital, and professor of pathology, Albert Einstein College of Medicine of Yeshiva University, New York City.

Baylor Graduate Achieves Highest Examination Score

Dr. Carlos Robert Hamilton, Jr., of Baltimore, Md., made the highest grade on the certification examination given by the Texas State Board of Medical Examiners on June 20-21. Dr. Hamilton is a graduate of Baylor University College of Medicine in Houston. His grade was a general average of 93.5.

Second and third ranks on the examination were achieved by Dr. Dwain Gordon Fuller, of Charlottsville, Va., with 93.3, and Dr. Byron James Bohnn of Wichita Falls, with 93.2. Dr. Fuller is a graduate of Baylor University College of Medicine, Dr. Bohnn of the University of Texas Medical Branch.

Texas Blue Cross-Blue Shield Announces New Appointment

Charles D. Scott, life insurance executive, has become associated with Blue Cross-Blue Shield of Texas. His responsibilities will include general managership of the life insurance operation, as well as serving on the Blue Cross-Blue Shield executive staff.

MR. SCOTT

Mr. Scott, a fellow in Life Management Institute, has been in the insurance industry since 1930 and since 1948 has been affiliated with Great American of Dallas Insurance Companies in various executive positions, including the office of president.

Mr. Scott has actively participated in life and health insurance activities on the local, state and national levels. He has served as chairman of the Hospital Relations Committee of the Health Insurance Council, chairman of the Texas Committee for Health Insurance Council, vice-president of the Texas Life Convention, and was one of the founding members of the Hospitals-Insurance-Physicians Joint Advisory Committee of Texas.

Prenatal Injury: Physicians Held Liable by Courts

FOR EIGHTY YEARS the courts of every jurisdiction in the United States followed the reasoning and rationale of Oliver Wendell Holmes concerning the physician's liability for prenatal injury as set out in the opinion he wrote in *Deitrich v. Inhabitants of Northampton*, 138 Mass. 14.

In that case the Massachusetts Supreme Court denied recovery to an administrator suing on behalf of a prematurely born child for injuries sustained while the infant was nonviable in his mother's womb. Holmes, speaking for the Court, theorized that a child during pregnancy was an integral part of its mother rather than a separate entity, and that not being a person, it had no rights and no cause of action could accrue in its favor.

The *Deitrich* case was the landmark decision to which courts turned when prenatal injury cases arose; and, at least until recently, it was generally followed. Thus, the view prevailed in this country that a child could not maintain an action for injuries sustained prior to birth, and that no legal duty was owed to a person not in existence.

Another and obvious reason for the ruling in that case was the difficulty of establishing a causal relationship between the physician's conduct and the child's injury. The Court felt that a ruling allowing such a claim would give rise to many fictitious allegations.

In 1946 a federal court held in *Bonbrest v. Kotz*, DC, 65 F. Supp. 138, that a child could maintain an action for prenatal injuries sustained through alleged malpractice as it was taken from the mother's womb. Since the decision in *Bonbrest* the courts almost consistently have rejected the arguments once relied upon and have said that a cause of action accrues for prenatal injuries.

Although there are no Texas decisions in this particular field of malpractice law, the majority of the jurisdictions in this nation which have appellate opinions rendered in lawsuits alleging prenatal injuries have adopted the view first espoused in the *Bonbrest* decision. When it is suggested that such an extension of tort liability may encourage false claims, the courts answer by saying that the issue of a causal connection between the injury to the newborn child and the negligence of another is one of proof which differs, if at all, only in degree from other medical questions which daily arise in negligence cases. In determining whether or not a physician can be held liable for prenatal injuries, the courts have generally held that such a cause of action does accrue to a child if that child is born alive and has suffered injuries sustained when viable in his mother's womb.

In recent years some courts have discarded the test of viability and in its stead have proclaimed that a right of action may accrue to the child from the moment of conception. The courts of various states, however, have not agreed unanimously with this. The trend, though, has been reinforced by the decision just handed down by the Rhode Island Supreme Court in the case of *Sylvia v. Gobeille*, June 17, 1966, 220 A 2d 222. The Rhode Island court ordered a physician to stand trial in an action brought by the father of an infant born with physical defects. The physician, the father alleges, had failed to prescribe gamma globulin for the mother despite the fact that he was aware that she had been exposed to German measles. In overruling past Rhode Island cases on prenatal injury the Court explained its decision in language that has broad legal implications:

> More importantly, however, we act for the reason that we deem it fitting and proper to protect a child's right to commence life unhampered and unimpaired by damage negligently caused to his body or mind by another.

The *Sylvia* decision also rules out the test of viability in the womb as the beginning point in time for the accrual of such causes of action. The court also held:

> There still remains the problem of whether the right to sue should turn on the child's viability at the time the injury occurred. That problem exists because there is a view that recovery may be had only for injuries sustained after a child becomes viable. For a child to be viable means that it is so far developed and formed that if born it could exist outside the mother's womb even if only in an incubator, and the term viability refers to that period in a child's fetal development when it becomes capable of independent life. In our judgment there is no sound reason for drawing a line at the precise moment of the fetal development when the child obtains the capability of an independent existence, and we reject viability as a decisive criterion.

The court states that this rejection of the viability concept is not based on any medical grounds but because "we are unable logically to conclude that a claim for an injury inflicted prior to viability is any less meritorious than one sustained after viability."

The court buttresses its opinion with conclusion that "justice requires that the principle be recognized that a child has a legal right to begin life with a sound mind and body" and that if "the wrongful conduct of another interferes with that right, and it can be established by competent proof that there is a causal connection between the wrongful interference and the harm suffered by the child when born, damages for such harm should be recoverable by the child." This holding is in line with other recent decisions from Pennsylvania, New York, Georgia, New Hampshire, Wisconsin, and Michigan.

The Rhode Island court explicitly states that since the situation before it involved the injuries sus-

tained by a child who was born alive, its decision must not be interpreted as a decision on whether or not the child must be born alive to maintain an action for injuries sustained while in the womb of the mother. The court states that:

> We decide only the issue before us and hold that a child born alive has a right of action in tort against a negligent wrongdoer for prenatal injuries.

As previously stated, the problems discussed herein have not been presented for decision to Texas courts. But with the erosion of the "locality" tests in malpractice and negligence cases and with the widespread publicity given to malpractice decisions, the probability of a Texas decision on a prenatal injury case is great.

Also, the Rhode Island case presents still another forceful reason for physicians to maintain good medical records since such records often provide the doctor's best defense in disproving non-meritorious malpractice allegations of patients. It is of the utmost importance in the field of prenatal injuries to remember that a plaintiff's cause of action is not barred by the statute of limitations until two years after the injured party's 21st birthday. Thus, theoretically, a physician could be sued for an alleged prenatal injury which took place 23 years, 8½ months prior to the filing of a lawsuit. Without proper medical records, a physician or his estate might be in a "swearing match" with a malformed or injured plaintiff. It certainly would not take an experienced trial attorney to predict where the jury's sympathy would lie in such a case.

—Philip R. Overton, LL.B.,
Sam V. Stone, Jr., LL.B.,
Austin, Legal Counsel,
Texas Medical Association.

Postgraduate Courses

Pharmacy Refresher Course, Austin, Oct. 28-29.—This two-day seminar will attempt to clarify the functions of the community pharmacist in providing pharmaceutical service to the nursing home and hospital. The emphasis will be on the practical application of methods and procedures for supplying the required services from the community pharmacies under Medicare.

The course is presented annually by the Pharmacy Extension Service of the University of Texas College of Pharmacy. Further information can be obtained from Mr. Bill D. Jobe, Director, Pharmacy Extension Service, University of Texas, Austin 78712.

Fractures and Joint Injuries, Denver, Colo., Nov. 2-4.—A three-day course, sponsored by the Division of Orthopedic Surgery, the Department of Surgery, and the Office of Postgraduate Medical Education, the University of Colorado School of Medicine, is acceptable for credit for members of the American Academy of General Practice. Tuition fee is $45, of which $10 is non-refundable in case of inability to attend.

Further information is available from the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver, Colo. 80220.

Gynecological Endocrinology, Houston, Nov. 16-17.—The University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education, will present a two-day intensive short course in the Texas Medical Center, Houston. The course will be given in collaboration with Baylor University College of Medicine and is designed for practicing physicians desirous of a comprehensive series of summaries concerning pertinent steroid gonadotropic studies and a review of the management of the common related gynecological problems.

"We're spending all our money to keep them from having what I had as a child."

The guest faculty will consist of Dr. Nichols Vorys and Dr. Paige K. Besch, Ohio State University College of Medicine, Columbus, Ohio; Dr. C. Donald Christian, associate professor of obstetrics and gynecology and endocrinology, Duke University School of Medicine, Durham, N.C.; and Dr. Joseph W. Goldzieher, chairman of the Department of Endocrinology, Southwest Foundation for Research and Education, San Antonio.

Further information may be obtained from the University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education, 102 Jesse Jones Library Building, Texas Medical Center, Houston 77025.

Annual Pediatric Postgraduate Symposium, Houston, Nov. 17-19.—The Division of Maternal and Child Health of the Texas State Department of Health and the Department of Pediatrics, Baylor University College of Medicine will present a symposium at the Texas Children's Hospital in Houston.

Guest speakers will include Dr. John D. Crawford, associate professor of pediatrics, Massachusetts General Hospital, Boston; Dr. F. Clarke Fraser, professor of genetics, Human Genetics Sector, McGill University, Montreal, Canada; Dr. Malcolm A. Holliday, professor of pediatrics, University of California School of Medicine, San Francisco Medical Center; Dr. Samuel Karelitz, director of pediatrics, Long Island Jewish Hospital, New Hyde Park, N.Y.; and Dr. Richard L. Naeye, associate professor of pathology, University of Vermont College of Medicine, Burlington, Vt.

Physicians interested in attending should contact Dr. Russell J. Blattner, professor and chairman, Department of Pediatrics, Baylor University College of Medicine, Houston 77025.

Pulmonary Function in Health and Disease, New Orleans, Nov. 28-Dec. 1.—The third annual postgraduate course on Pulmonary Function in Health and Disease will be sponsored by the American Thoracic Society, the Louisiana

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HP/Sanborn field offices can give you valuable help in system planning, installation and staff training — and provide continuing, local service. For details, send the coupon to Hewlett-Packard Company, Sanborn Division, Waltham, Mass. 02154. In Europe, H.P.S.A., 54 Route des Acacias, Geneva.

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Thoracic Society, the Tulane University School of Medicine, the Louisiana State University School of Medicine, and the Alton Ochsner Medical Foundation.

Tuition for the four-day course is $75 for members of the American Thoracic Society and for physicians whose tuition is provided by voluntary tuberculosis and respiratory disease associations; $100 for physicians not members of the society; and $25 for nurses and technicians. Residents will be enrolled tuition-free upon acceptance by the course committee.

Further information may be obtained from the Pulmonary Function Course Committee, Louisiana Thoracic Society, Suite 407, 305 Barronne St., New Orleans 70112.

Progress in Gastroenterology — 1966, Philadelphia, Nov. 28-Dec. 2. —This course, presented by the American College of Physicians (ACP) through the University of Pennsylvania School of Medicine, has been planned to present intensive and current reviews of selected problems in gastroenterology.

Tuition fee for ACP members is $60; for nonmembers, $100. Only ACP members will be accepted in courses immediately upon receipt of application. The names of nonmembers will be placed on waiting lists in the order in which they are received. Three weeks after the release of the course outline nonmembers will be notified whether sufficient room remains for them in the course.

Minimum course registration will be 100; maximum registration, 250.

Requests for information and registration forms should be addressed to Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine Street, Philadelphia 19104.

What the Internist Should Know About Cancer, New York City, Dec. 5-9.—Also an American College of Physicians-sponsored course, this postgraduate course will be presented by the College of Physicians and Surgeons of Columbia University.

The course is designed to offer a current view of medical oncology. Opportunities for ward rounds and seminars on selected topics will be provided on an optional basis.

Minimum course registration will be 50; maximum, 110. For further information concerning tuition fees and course registration, see *Progress in Gastroenterology—1966*, immediately preceding. Additional information for this course may also be obtained from Dr. Edward C. Rosenow at the Philadelphia address.

Microbiology, Atlanta, Ga.—A series of laboratory refresher short courses will be presented by the Laboratory Branch of the Communicable Disease Center of the US Public Health Service. Various courses in special subdivisions of microbiology are being offered over an 11-month period.

Courses with registration closing dates through November are Isolation of Salmonellae from Food Products and Animal Feeds, and Laboratory Methods in Medical Mycobacteriology, both to take place Jan. 9-20, 1967, and both with registration closing on Nov. 28.

Mycology Workshop, Harlingen, Dec. 6-9.—The Texas State Department of Health Laboratory personnel and personnel from the Harlingen State Tuberculosis Hospital will present a cooperative program on the systemic fungi and dermatophytes.

Applications for workshops at tuberculosis hospitals may be addressed to Dr. Robert B. Skinner, Director, Division of Tuberculosis Hospitals, Texas State Department of Health, 1100 W. 49th St., Austin 78756.

Texas Hospital Association Has New Assistant Directors

Robert L. English of New Braunfels and David L. Moore of Oklahoma City have been appointed as assistant executive directors of the Texas Hospital Association. Mr. English will work with public information programs, and Mr. Moore will work with accounting activities.

The announcement of the appointments was made by O. Ray Hurst, executive director of the association. In the newly created position, Mr. English's duties will be devoted primarily to a stepped-up public information program in conjunction with the association's council on public education.

Mr. Moore's major responsibilities will be in developing, coordinating and conducting accounting programs and activities for the benefit of member hospitals and for the association.

A native of Brownwood, Mr. English received his B.A. degree from Howard Payne College and his master's degree in hospital administration from Baylor University.

Prior to entering the hospital field, he was editor of the Brady *Standard* and state editor of the Wichita Falls *Record News*.

In the hospital field, he has held administrative positions in Germany, Canal Zone, Temple, and San Angelo.

Mr. Moore is a native of Waco and received his bachelor of business administration degree from Baylor University in 1961. He has been a staff certified public accountant with Ernst and Ernst, a national accounting firm, in both the San Antonio and Oklahoma City offices.

One Doctor for 954 People Is Current Ratio in Texas

At the present time, Texas has a ratio of one physician for every 954 people. The population of Texas is currently estimated to be 10,700,000. The number of physicians in Texas at the beginning of 1966 was 11,218. The 11,218 figure includes all holding M.D. degrees, even those not currently in actual practice.

The physician population is expected to grow larger at a faster rate in the very near future because of the opening of the new University of Texas South Texas Medical School in San Antonio. Admission of a first class this fall gives Texas four medical schools.

At the beginning of 1966 there were 292,088 physicians in the United States, or one physician for every 681 persons. Recent graduations from the nation's 88 medical schools have pushed this figure over the 300,000 mark. The number of physicians in the United States has been increasing faster than the general population for several years.

The demand for medical service will continue to grow. New medical programs, such as Medicare, will put an increased load on the present physician population. The situation in Texas will also bear watching because Texas is one of the major "growth" states.

New Books In TMA Library

American College of Surgeons, Committee on Trauma: *Emergency Care of the Sick and Injured*, Philadelphia, W. B. Saunders Co., 1966.

American Society for Microbiology: *Symposium: Spores*, L. L. Campbell & H. O. Halvorson (eds.) Ann Arbor, Mich., 1965, vol. 3.

Brachman, L., ed.: *Encyclopedia for Medical Assistants*, Milwaukee, Wis., Cathedral Square Publishing Co., 1965.

Fields, W. S.: *Neurological Diagnostic Techniques*, 13th Annual Houston Neurological Scientific Symposium: Springfield, Ill., Charles C Thomas, Publisher, 1966.

Forster, F. M.: *Synopsis of Neurology*, ed. 2, St. Louis, C. V. Mosby Co., 1966.

Graham, M. F.: *Prescription for Life*, New York, David McKay Co., Inc., 1966.

Haley, L. D.: *Diagnostic Medical Mycology*, New York, Appleton-Century-Crofts, 1964.

Havener, W. H.: *Ocular Pharmacology*, St. Louis, C. V. Mosby Co., 1966.

International Symposium on Antihypertensive Therapy: *Antihypertensive Therapy: Principles and Practice*, F. Gross, (ed.), New York, Springer-Verlag, 1966.

Nahum, A. M., ed.: *Early Management of Acute Trauma*, St. Louis, C. V. Mosby Co., 1966.

National Foundation: *Birth Defects, Structural Organization of the Skeleton: A Symposium*, vol. 2, no. 1, New York, National Foundation, 1966.

Oski, F. A., and Naiman, J. L.: *Hematologic Problems in the Newborn*, (vol. 4 in the series Major Problems in Clinical Pediatrics), Philadelphia, W. B. Saunders Co., 1966.

Redlich, F. C., and Freedman, D. X.: *Theory and Practice of Psychiatry*, New York, Basic Books, 1966.

Winner, H. I., and Hurley, R.: *Candida Albicans*, Boston, Little, Brown and Co., 1964.

Book Notes

Surgery of World War II: Thoracic Surgery, Vol. II. F. B. Berry, ed., prepared and published under the direction of Lt. Gen. L. D. Heaton, Surgeon General, United States Army. 615 pp., 222 illus., tables, Washington, D.C., Government Printing Office, 1965, $7.25.

ASSOCIATED in this effort were such notable individuals as Drs. Brian B. Blades, Lyman A. Brewer III, Thomas H. Burford, B. Noland Carter, Michael E. DeBakey, and Dwight E. Harken. These names are brought out because it is doubtful if a group of such competence could have been brought together in a unified effort by any organization other than one of the armed services which in effect represents the nation itself.

This volume deals with surgical chest conditions which include not only foreign bodies in the lung and pleural cavities and mediastiunum, but also in the heart and great vessels. Some remarkable advances in surgery were brought about by the specialized chest centers during World War II. Particular mention is made of the techniques developed for the removal of foreign bodies from the heart and great vessels and the return to full duty of many of these people.

The technique of handling chest injuries and avoiding development of pleural changes that subsequently required decortication is one of the fine advances in the handling of chest injuries over that of World War I. Prompt aspiration of blood, fluid or pus from the pleural cavities and improved management of infections materially reduced the mortality and morbidity as compared to previous periods in the history of the services.

The importance of this volume is that it presents a follow-up of material to analyze the effectiveness of management by the chest surgery centers during the period of war time activities. It is illuminating and instructive.

One would be quick to point out that this should be a continuing study, for there will be different lessons to be learned. Modifications of sound methods already developed will be necessary to bring into play the many scientific advances in medicine and its allied scientific fields to further improve results.

Tribute should be paid to the refinements developed in anesthesia which made possible the handling of both thoracic and abdominal injuries sometimes simultaneously with the many differences in technique and handling depending on the sides and the organs involved.

Many complications and their sequelae are presented together with the method of their prevention and the best immediate management should any one of them occur.

While this book is naturally slanted toward the care of conditions incident to trauma, so many basic principles are brought out in the proper management of lung conditions generally that it makes a valuable addition to the reference library of anyone who sees emergency chest conditions, traumatic or otherwise.

—JOE C. RUDE, M.D., Austin.

Current Therapy 1966. Howard F. Conn, ed. 857 pp. Philadelphia, W. B. Saunders Co., 1966, $13.

WITH THE RAPIDLY changing therapeutic methods from all corners of the world, the latest edition of *Current Therapy* continues to bring the busy physician concise information in one volume. Traditional therapy as well as modern advances are combined in a well-balanced treatise on the management of disease.

Recent Deaths

DR. G. T. DENTON, SR.

Dr. Guy T. Denton, Sr., 86, a Dallas general practitioner, died Sept. 2, in a hospital there. He practiced in Dallas from 1921 to 1956 when he retired.

Dr. Denton was born Feb. 21, 1880, in Robards, Ky., the son of L. D. and Mary E. Lee Denton. He attended school there, and was graduated from Vanderbilt University in Nashville, Tenn., in 1906. He received his medical degree from Vanderbilt University School of Medicine in 1913. While attending college, Dr. Denton served as a coach in Vanderbilt's physical education department, and after graduation he was an instructor in the physical education departments of Vanderbilt, George Peabody College for Teachers, and Belmont College, all in Nashville. He interned at St. Thomas Hospital in Nashville in 1916.

When he first arrived in Dallas he was a volunteer doctor for the Oak Cliff High School football team. During his early years there, he worked with the Red Cross in its first aid and water safety programs.

Dr. Denton was an honorary member of the Dallas County Medical Society, Texas Medical Association, and the American Medical Association. He also was a charter member of the Dallas Southern Clinical Society. He was one of the original staff members of Methodist Hospital and had served the staff as president.

A member of Tyler Street Methodist Church, he was a charter member of Oak Cliff Chamber of Commerce and the Oak Cliff Kiwanis Club.

Survivors include his wife, a son, Dr. Guy T. Denton, Jr., a daughter, Mrs. Frances Belk, all of Dallas; three brothers, Dr. Irving Denton and Dr. Rex Denton, both of Fordsville, Ky., and Max Denton of Robards, Ky.; a sister, Mrs. Clara D. Royester of Paducah, Ky.; six grandchildren and one great-grandchild.

DR. DENTON

DR. E. P. REED, SR.

Dr. Emil Patrick Reed, 61, died Sept. 7 in a Dumas hospital. He had been ill several months.

Dr. Reed was a medical officer in the US Army from November, 1940, until February, 1946. He was captured by the Japanese and was in the Bataan Death March in 1942. He was held prisoner until January, 1945. Before retiring from service as a lieutenant colonel, Dr. Reed received the Purple Heart medal.

The son of a physician, Dr. Horace Reed, and Frankie Patrick Reed, he was born in Guthrie, Okla., on March 10, 1905. His only brother, Karl A. Reed, is also a physician. On June 22, 1931, Dr. Emil Reed married Newell Cochran of Dallas in Oklahoma City.

He attended schools in Oklahoma City and Bell Buckle, Tenn. He was graduated from the University of Oklahoma in 1929, and from the University of Oklahoma School of Medicine in 1931. He interned at the University Hospital, Ann Arbor, Mich., and remained there to serve a surgical residency. He practiced in Oklahoma City and Brownsville, Tex., prior to entering the service. After his return from the war, Dr. Reed was associated with the Veterans Administration Hospitals in Biloxi, Miss., and Amarillo. He entered private practice in Amarillo for five years, and rejoined the Veterans Administration Hospital staff in 1953. He moved to Dumas in 1955, where he was on the staff of Memorial Hospital.

A diplomate of the American Board of Surgery, he was a fellow of the American College of Surgeons and a member of the Dallam-Hartley-Sherman-Moore Counties Medical Society, Texas Medical Association, American Medical Association, and the Southwestern Surgical Congress. A Mason, he also belonged to the American Radio League, Kappa Sigma fraternity, Khiva Temple, and the First Methodist Church in Dumas.

His wife survives, as do a son, Lt. Emil Reed, Jr., serving in the US Army in Germany; two daughters, Mrs. Elizabeth Faulkner, Dumas; and Mrs. Virginia Allen, Great Yarmouth, England; a brother, Dr. K. A. Reed of Santa Rosa, Calif., and five grandchildren.

DR. REED

DR. W. L. BUSH

Dr. William Leslie Bush, 60, a Dallas urologist, died there on Sept. 8, 1966. He was born Aug. 8, 1906, in Greenville, Tex.

He attended Wesley College in Greenville and received his bachelor's degree from the University of Texas in 1927. He was graduated from the University of Texas Medical Branch in Galveston in 1931. He served internships at Bellevue General Hospital, New York City, and John Sealy Hospital in Galveston. After completing a one-year rotating residency at Shannon West Texas Memorial Hospital in San Angelo, he practiced there for eight years. He then entered the US Army Medical Corps during World War II. When he returned from the war, having attained the rank of colonel, Dr. Bush practiced in Greenville for six months. He then served a

DR. BUSH

residency in urology at John Sealy Hospital. He practiced urology in Dallas from 1949 until his retirement due to ill health in 1964.

Elected to inactive membership in the Texas Medical Association in 1965, Dr. Bush also belonged to the Dallas County Medical Society and the American Medical Association. A deacon in Northway Christian Church, he belonged to the Northwood Country Club.

His wife, Mrs. Nell Bush, survives, as do a son, Pvt. Thomas E. Bush, serving in the US Marine Corps in San Diego, Calif.; two daughters, Miss Katherine Bush of Austin, and Mrs. Devereux Dunlap of Albuquerque, N. Mex.; one sister, Mrs. R. L. Craven of Greenville; and two grandchildren.

DR. O. R. TAYLOR

Pioneer Linden physician, Dr. Orval R. Taylor, 82, died Aug. 30 in a hospital there after a long illness.

Dr. Taylor was born in Linden on Oct. 19, 1884, the son of W. C. and Fredonia Black Taylor. He attended school in Atlanta, Tex. He attended the University of the South, Sewanee, Tenn., and the Memphis Hospital Medical College. He received his medical degree from Texas Christian University Medical Department (combined with Baylor University College of Medicine in 1918) in 1917. He did postgraduate work at the then Tulane University of Louisiana School of Medicine in 1926-1930. He began his medical practice in Linden in 1917 and remained in practice there until his retirement in 1963. In 1957 Dr. Taylor was honored by the City of Linden with a testimonial dinner. The day was named "Dr. Taylor Day" in honor of the forty years he had practiced in Linden.

He was a member of the Cass-Marion Counties Medical Society, which he had served as secretary and president, the Texas Medical Association, the American Medical Association, and was a charter and life member of the American Academy of General Practice.

Dr. Taylor was active in many civic organizations in Linden. He served for 30 years as president of the Linden School Board. A 32nd degree Mason, he was a Shriner and Knight Templar. He served on the official board of the Linden Methodist Church for 15 years, and was active in the Men's Bible Class. He was a member of the Linden Lions Club, the Linden Chamber of Commerce, and served as financial chairman of the Boy Scouts of America Drive for Cass County.

Dr. Taylor and Annie L. Henderson were married Nov. 6, 1949, in Naples, Texas. She survives as do a daughter, Mrs. Kathryn Temple, Linden; two sisters, Mrs. Willie Spearman, Oklahoma City, Okla., and Mrs. Lucille Cochran, Conroe; and one granddaughter, Kay Temple, a student at the University of Arkansas.

DR. TAYLOR

DR. S. BUSCH

Dr. Sylvan Busch, 37, died Aug. 28, in a Dallas hospital following a long illness. He was born March 17, 1929, in Philadelphia, Pa., the son of Hyman and Anna Doyne Busch.

He attended the University of Pennsylvania and received his bachelor of science degree from the University of Kansas in 1950. After receiving his medical degree from the University of Kansas School of Medicine in 1955, he interned at Bellevue General Hospital in New York, N.Y. He served a residency in internal medicine at Bronx Municipal Hospital Center in New York, N.Y. After completing his residency, Dr. Busch served as a lieutenant in the U.S. Navy at the U.S. Naval Hospital in Annapolis, Md. He then moved to Dallas where he practiced until his death. He was secretary of the medical staff of St. Paul's Hospital.

A diplomate of the American Board of Internal Medicine, he was a member of Dallas County Medical Society, Texas Medical Association, American Medical Association, Alpha Omega Alpha honorary medical fraternity, and Phi Beta Pi medical social fraternity.

A member of Temple Emanu-El, he was a member of Alpha Epsilon Pi social fraternity and Alpha Chi Sigma chemistry fraternity.

He married Carolyn Sue Lipshy on April 13, 1952, in Lubbock, Tex. She survives as do a son Alan Busch, three daughters, Susan Gail, Saralynn, and Lauren Busch, all of Dallas; his mother, Mrs. Anna Busch of Miami, Fla.; brother, Leonard Busch of Morrisville, Pa., and a sister, Mrs. Ruth Seltzer of Miami, Fla.

DR. BUSCH

DR. C. H. HAGGARD

San Antonio physician, Charles Houston Haggard, 74, died July 23 in Houston of pulmonary edema and pneumonia due to acute myelogenous leukemia. He had practiced in San Antonio since 1927.

He was born June 26, 1892, in Georgia, the son of John C. and Nancy Henry Haggard. He received his medical degree from the University of Texas Medical Branch in 1917. He served an internship at the Temple Sanatorium. Before moving to San Antonio, Dr. Haggard was with the U.S. Public Health Service. He served for many years as surgeon for Missouri Pacific Railroad.

A member of the Texas Medical Association through Bexar County Medical Society, he was a member of the American Medical Association and the American Academy of General Practice. He was a member of the First Presbyterian Church and served as elder.

Dr. Haggard is survived by a daughter, Mrs. Walter Brenan of San Antonio; two brothers, Frank N. Haggard, San Antonio, and Horace Haggard, Fort Worth; and two sisters, Mrs. Mary Smallwood, Hillsboro, and Mrs. Bessie Marshall, San Francisco, Calif.

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*Diplomate of The American board of his specialty

**Required training for Certification by the American Board of his specialty has been completed.

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COMING MEETINGS

American Medical Association, Clinical Meeting, Las Vegas, Nov. 27-Dec. 1, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 60610, Exec. Vice Pres.

Texas Medical Association, Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

OCTOBER

American Academy of General Practice, Boston, Oct. 7-14, 1966. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 64112, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16-21, 1966. Dr. W. L. Benedict. 15 2nd St., S.W., Rochester, Minn. 55901, Sec.

American Academy of Pediatrics, Chicago, Oct. 22-27, 1966. Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Cancer Society, Inc., New York City, Oct. 18-20, 1966. Mr. Charles R. Ebersol, 219 E. 42nd St., New York 10017, Sec.

American College of Gastroenterology, Philadelphia, Oct. 23-26. 1966. Mr. D. Weiss, 33 W. 60th, New York 10023, Exec. Dir.

American College of Obstetricians and Gynecologists, Houston, Oct. 24-26, 1966. Mr. D. F. Richardson, 79 W. Monroe, Chicago 60603, Exec. Sec.

American College of Surgeons, Annual meeting, San Francisco, Oct. 9-14, 1966. Dr. Samuel P. Harbison, 55 E. Erie St., Chicago, Ill. 60611.

American Heart Association, New York City. Oct. 21-25, 1966. Mr. Rome A. Betts, 44 E. 23rd, New York 10010, Exec. Dir.

American Public Health Association, Inc., San Francisco, Oct. 31-Nov. 4, 1966. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

Association of American Physicians and Surgeons, Inc., Disneyland, Calif., Oct. 13-15, 1966. Mr. H. E. Northam, 185 N. Wabash, Chicago 60601, Exec. Dir.

Medical Society of United States & Mexico, Tucson, Ariz., Oct. 6-8, 1966. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz. 85716, Sec.

Southern Psychiatric Association, Memphis, Tenn., Oct., 1966. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Texas Association for Mental Health, Austin, Oct. 27-28, 1966. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin 78756, Exec. Sec.

Texas Dermatological Society, El Paso, Oct. 1-2, 1966. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio 78205, Sec.

Texas Surgical Society, Houston, Oct. 2-4, 1966. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco 76702, Sec.

Third District Society, Amarillo, Oct. 29, 1966. Dr. Louis Devanney, 2209 W. 7th. Amarillo 79106, Sec.

Oklahoma City Clinical Conference, Oklahoma City, Okla., Oct. 24-26, 1966. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 73102, Exec. Sec.

NOVEMBER

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American Medical Women's Association, Washington, D.C., Nov. 2-5, 1966. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

Gastrointestinal Research Group, Chicago, Nov. 4. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn. 55901, Sec.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb. 68104, Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. R. R. Kierland, Mayo Clinic, Rochester, Minn. 55901, Sec.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 7-9, 1967. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe. 715 Lake St., Oak Park, Ill. 60301, Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo. 80302, Treas.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, Jan. 31-Feb. 4, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American Congress of Physical Medicine and Rehabilitation, Miami Beach, Fla., Aug. 27-Sept. 1, 1967. Mr. Creston C. Herold, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn. 55901, Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C. 27103, Sec.

American Gynecological Society, Phoenix, May 4-6, 1967. Dr. G. W. Douglas, 550 First Ave., New York 10016, Sec.

American Hospital Association, Chicago, Aug. 21-24, 1967. Mr. James E. Hague, 840 N. Lake Shore Drive, Chicago 60611, Sec.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Society of Clinical Pathologists, Las Vegas, Feb. 26-March 4, 1967 (Interim). Dr. Lester H. Hoyt, Methodist Hospital, Indianapolis, Ind. 46207, Sec.

American Society of Anesthesiologists, Las Vegas, Sept. 30-Oct. 4, 1967. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill. 60608, Exec. Sec.

American Surgical Association, Colorado Springs, May 11-14, 1967. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis 46207, Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Gastrointestinal Research Forum. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa. 19104, Sec.

International College of Surgeons, April 30-May 5, 1967. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago 60610, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Place, San Antonio 78212, Exec. Sec.

Private Clinics and Hospitals, Austin, December, 1966, A. A. Hubbard, Alvarado Community Hospital, Alvarado 76009, Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La. 70806, Sec.

Southern Surgical Association. Boca Raton, Fla., Dec. 6-8, 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore 21201, Sec.

South Texas District, American College of Surgeons, Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio 78205, Sec.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston 77550, Pres.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine, Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla. 73103, Sec.

State

Texas Academy of General Practice, Austin, June 12-14, 1967. Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 10, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth 76104, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Dallas, May 4-7, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics, R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, Dallas, Sec.

Texas Division, American Cancer Society, Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Heart Association, Houston. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 U. S. Highway 290 E., Austin, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, April 4-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Houston, Sept. 28-30, 1967. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston, 77025, Sec.

Texas Proctologic Society, Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, San Antonio, Feb. 26-Mar. 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967, Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth, Sec.

Texas Rehabilitation Association, D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 509 West 26th, Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society on Aging, Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, Dr. W. Cone Johnson, Scott and White Clinic, Temple, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont, Sec.

Texas Society of Plastic Surgeons, Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences, Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 5-7, 1966. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 76102, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Traumatic Surgical Society, Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso, Sec.

Seventh District Society, Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society, Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston, Sec.

Tenth District Society, Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Tyler, Spring, 1966. Dr. Lucy Mathis, 107 Redland Lane, Palestine, Sec.

Twelfth District Society, Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Fort Worth, Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Clinics

Blackford Memorial Lectures, Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas, Dir.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 17-19, 1967. Dr. W. Tom Arnold, 6448 Fannin, Houston 77025, Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 Fifth Ave., Fort Worth, Sec.

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The "Socio-geographic" mystery

Why is one man's gastric ulcer another man's duodenal?

Geographic variation in the *incidence* of peptic ulcer is a familiar fact. But the proclivity of certain *kinds* of ulcer for certain geographic areas is a recently recognized phenomenon.[1,2]

For example, in one particular Norwegian fishing village there is a tendency for patients to develop a gastric ulcer; anywhere else in Norway, ulcers are usually duodenal. Peruvians high in the Andes have more gastric ulcers than their compatriots in the lowlands. Why? Nobody knows.

Social variations, too. Even in the same geographic areas there are interesting variations. An Englishman's ulcer depends on his social standing—professional men suffer with duodenal ulcers, while workingmen have more of the gastric variety. In southern India the pattern is reversed. Here, duodenal ulcers are common among laborers and agricultural workers and rare among the upper classes.

Investigators are exploring every possible theoretical avenue in their search for the cause of peptic ulcer. Of all the factors implicated in ulcerogenesis, the one that is generally acknowledged to be of primary importance is hypersecretion of gastric acid.[3-8] Or, as one author states it: "The medical management of peptic ulcer pharmacologically is, in the final analysis, concerned largely with the effective inhibition of peptic activity."[3]

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Precautions: Administer with caution in the presence of incipient glaucoma.
Adverse Reactions: Dryness of the mouth, blurred vision, urinary difficulties, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.
Dosage: Dosage should be adjusted according to individual patient response. Average and maximum recommended dose is 1 tablet 3 times a day: in the a.m., early p.m., and at bedtime. *See product literature for full prescribing information.*
Supply: Robinul (glycopyrrolate 1 mg.); Robinul Forte (glycopyrrolate 2 mg.); Robinul-PH (glycopyrrolate 1 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming); Robinul-PH Forte (glycopyrrolate 2 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming.) In bottles of 100 and 500 scored tablets.
References: **1.** Jones, F. A., and Gummer, J. W. P.: Clinical gastroenterology, Springfield, Ill., Charles C Thomas, 1960, pp. 322-3. **2.** Bockus, H. L.: Gastroenterology, 2nd ed., vol. I, Philadelphia, Saunders, 1963, p. 468. **3.** Sun, D. C. H.: Ann NY Acad Sci 99:153 (Feb. 28) 1962. **4.** Moore, V. A.: Postgrad Med 38:216 (Sept.) 1965. **5.** Dragstedt, L. R., Woodward, E. R., Storer, E. H., Oberhelman, H. A., Jr., and Smith, C. A.: Ann Surg 132:626 (Oct.) 1950. **6.** Posey, E. L., Jr., Smith, P., Turner, C., and Aldridge, J.: Amer J Dig Dis 10:399 (May) 1965. **7.** Lamphier, T. A., Siegel, L., and Goldberg, R. I.: Amer J Gastroent 37:551 (May) 1962. **8.** Kasich, A. M., and Fein, H. D.: Ibid 39:61 (Jan.) 1963. **9.** Epstein, J. H.: Ibid 37:295 (Mar.) 1962. **10.** Moeller, H. C.: Ann NY Acad Sci 99:158 (Feb. 28) 1962. **11.** Slanger, A.: J New Drugs 2:215 (Jul.-Aug.) 1962. **12.** Barman, M. L., and Larson, R. K.: Amer J Med Sci 246:325 (Sept.) 1963. **13.** Shutkin, M. W.: Amer J Gastroent 38:682 (Dec.) 1962. **14.** Fleshler, B.: J New Drugs 2:211 (Jul.-Aug.) 1962.

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SYMPOSIUM ON ADOLESCENCE

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Irvin Kraft, M.D., Professor of Child Psychiatry at Baylor Medical School, Houston, Tex.

John Schimel, M.D., Associate Director of William Alanson White Institute of Psychiatry, Psychoanalysis and Psychology, New York, N. Y.

George Tarjan, M.D., Professor of Psychiatry and Program Director of Mental Retardation Project at University of California in Los Angeles, Calif.

Carroll Witten, M.D., President-Elect of American Academy of General Practice, Louisville, Ky.

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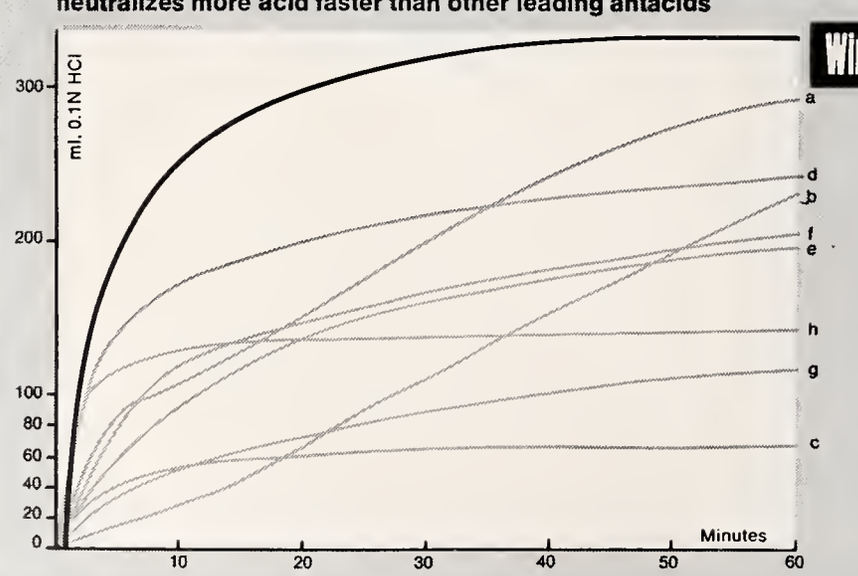

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Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

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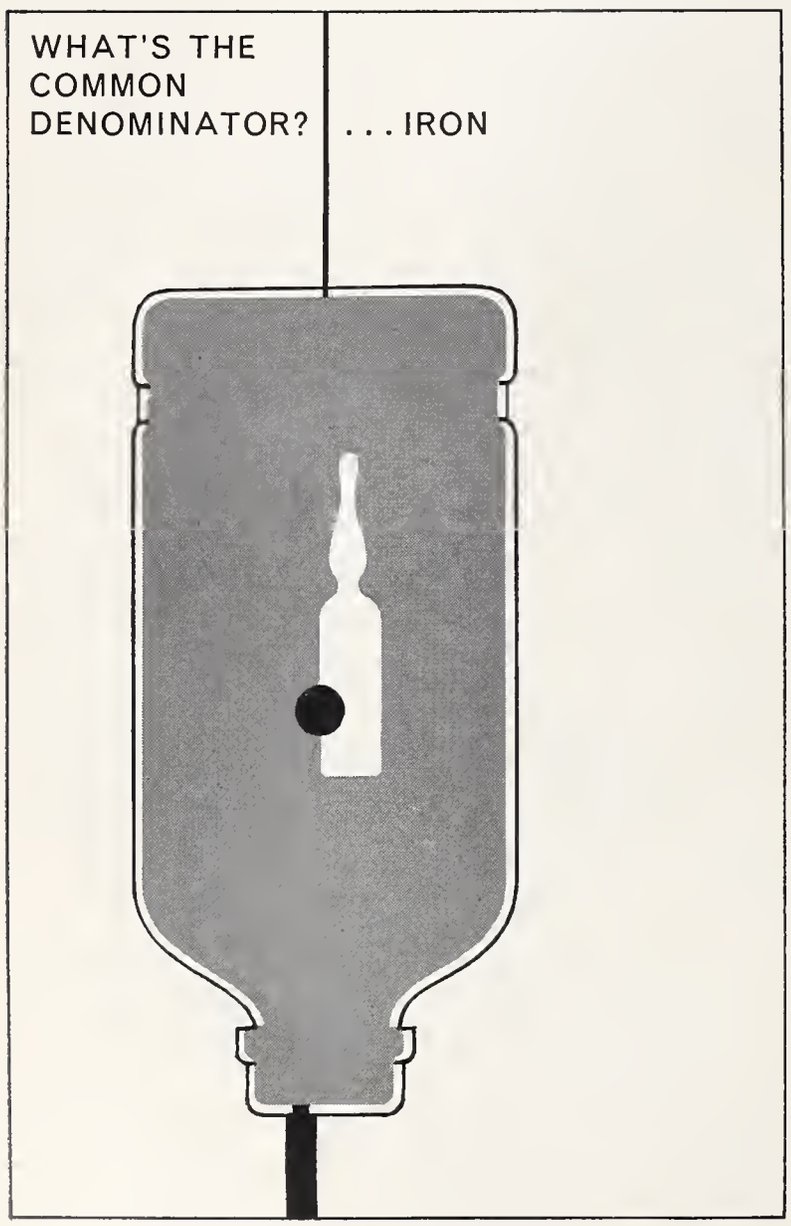

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SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylatoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

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Each chewable tablet contains: nicotinic acid (niacin) 75 mg. and aminoacetic acid (glycine) 750 mg.

Administration and Dosage: One or two chewable tablets 3 times a day before meals. If flushing is objectionable, dosage may be lowered. However, tolerance to flushing usually develops without loss of efficacy in regard to vasodilation. The recommended dosage should not be exceeded.

Side effects: Occasional lightheadedness or transient itching which may disappear with continued use. There are no known contraindications; however, caution is advised when there is a concomitant administration of a coronary vasodilator.

Supplied: Packages of 50 chewable tablets.

Also available in liquid form as Geriliquid®, in bottles of 8 and 16 ounces.

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Each tablet contains:
Dactil® (piperidolate hydrochloride), 50 mg.;
Standardized cellulolytic* enzyme, 2 mg.;
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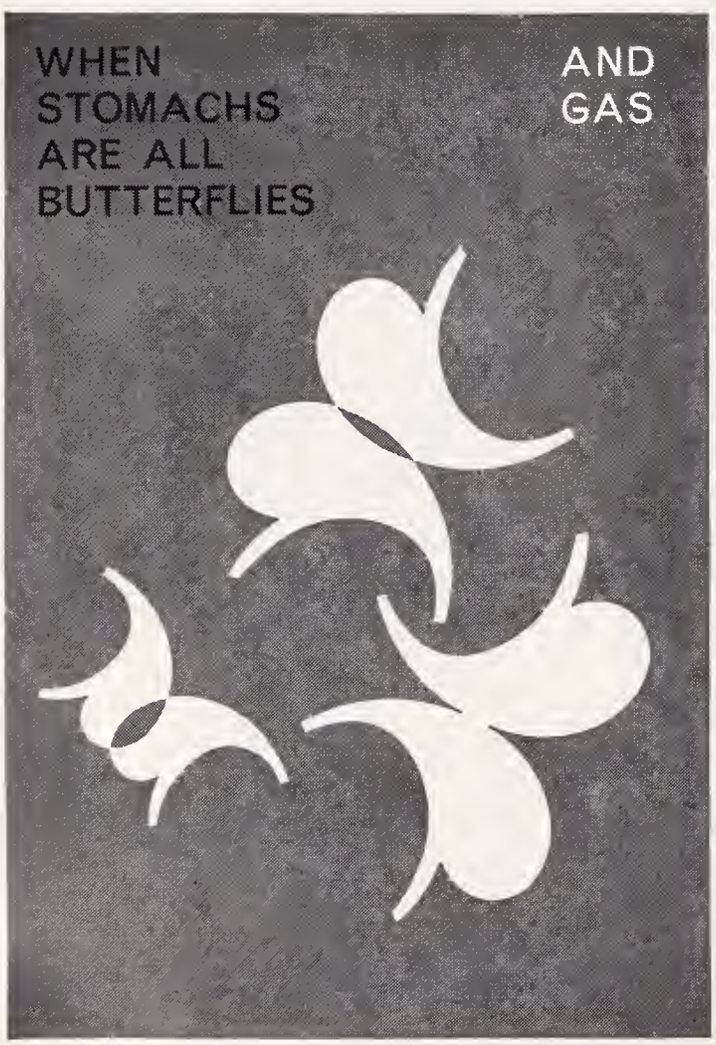
Pancreatin 3X** (source of lipolytic activity), 100 mg.; Taurocholic acid, 15 mg.

*Need in human nutrition not established.
**As acid resistant granules equivalent in activity to 300 mg. Pancreatin N.F.



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Side Effects and Contraindications: Dactilase is almost entirely free of side effects. However, it should be withheld in glaucoma and in jaundice due to complete biliary obstruction.

Administration and Dosage: One tablet with, or immediately following, each meal. Tablets should be swallowed whole.

Supplied: Bottles of 60 and 250.

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"In 40 of 44 cases of irritable or spastic colon, Cantil [mepenzolate bromide] or Cantil with Phenobarbital reduced or abolished abdominal pain, diarrhea and distention and promoted restoration of normal bowel function . . . Cantil [mepenzolate bromide] proved to be singularly free of anticholinergic side-effects . . . Urinary retention, noted in two cases was eliminated in one by reducing dosage."[1]

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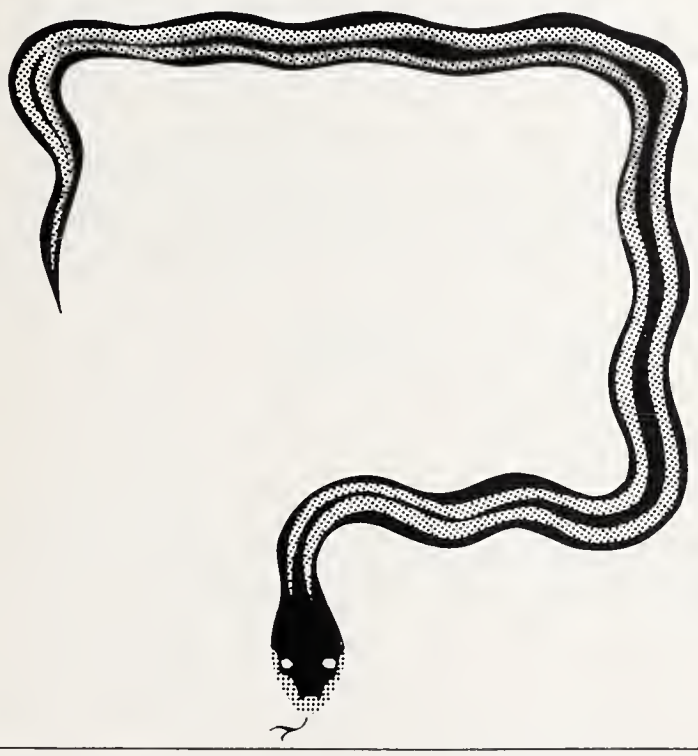

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One or two tablets three times a day and one or two at bedtime usually provide prompt relief. Cantil with Phenobarbital may be prescribed if sedation is required.

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1. Riese, J.A.: Amer. J. Gastroent. 28:541 (Nov.) 1957

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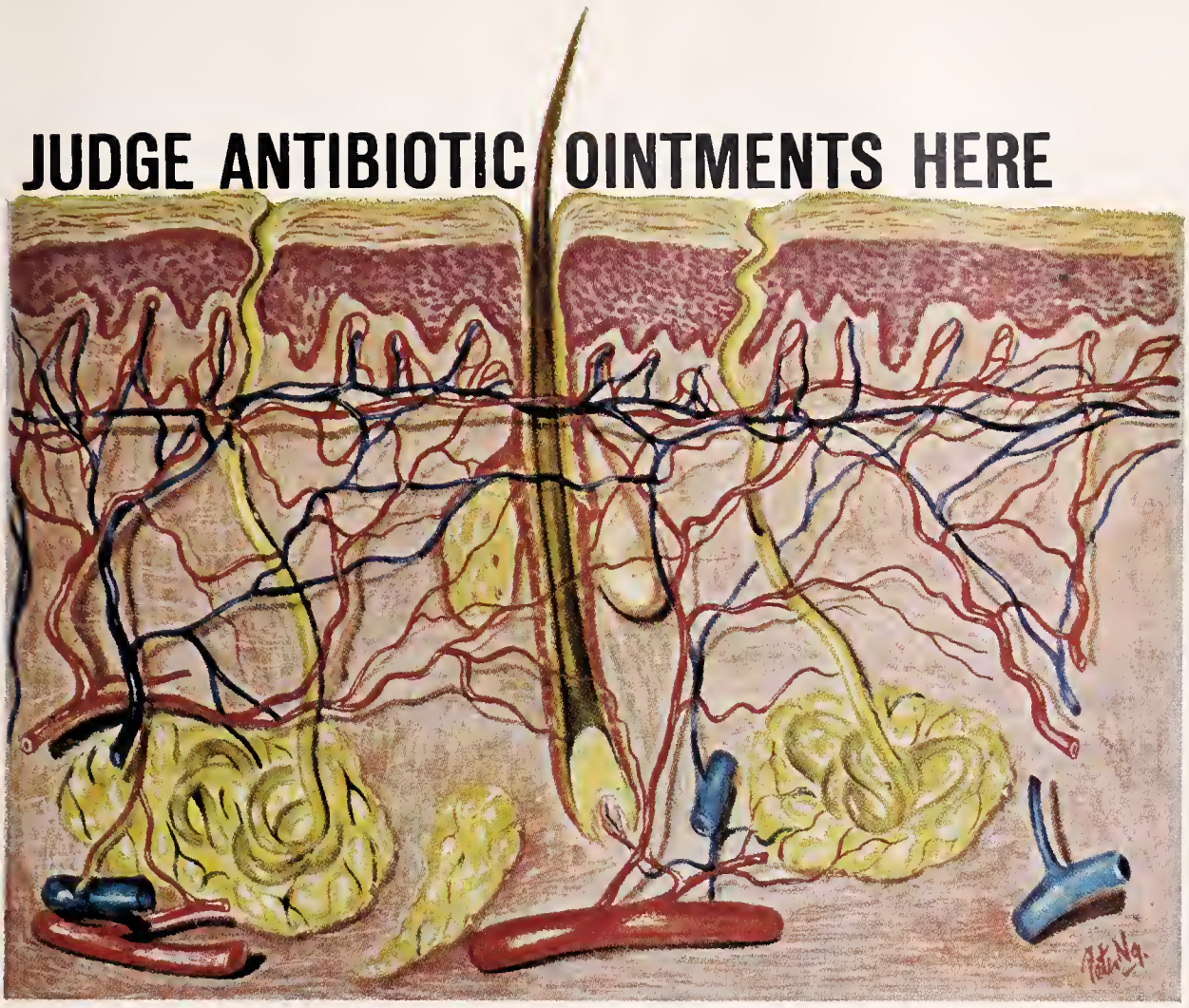

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Contraindication: Not recommended during pregnancy.

Caution: May be habit-forming. Do not use in patients sensitive to barbiturates or in those with moderate to severe hepatic disease.

Side Effects: Nausea, transitory dizziness, rash. Overdosage of allobarbital produces symptoms typical of acute barbiturate excess.

Dosage: *Adults:* 1 or 2 tablets every 4 hours. Not to exceed 8 tablets in 24 hours. *Children 6 to 12:* ½ to 1 tablet every 4 hours. Not to exceed 4 tablets in 24 hours.

Supplied: *Tablets* (white, scored), each containing 15 mg allobarbital and 300 mg acetaminophen; units of 3 bottles of 30.

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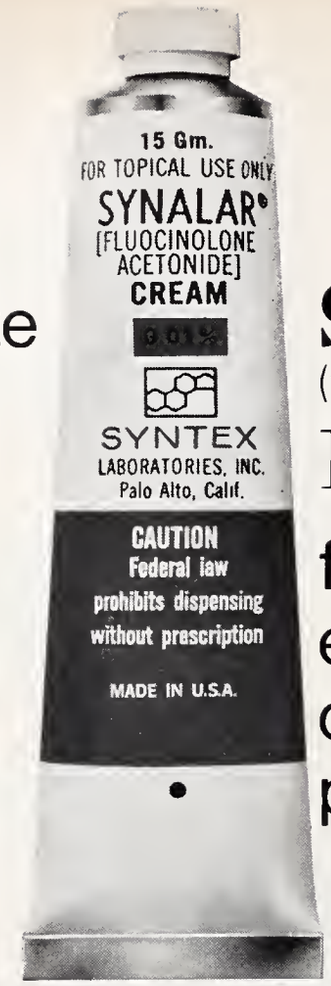

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Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of its components. **Precautions:** 1. *General*—Synalar Cream 0.01% is virtually nonsensitizing and nonirritating. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. 2. *Occlusive dressing method*—With occlusion of extensive areas, systemic absorption of the corticosteroid may occur, and suitable precautions should be taken. Occasional patients may show contact sensitivity to a particular dressing material or adhesive. Miliaria, folliculitis, or pyodermas have been seen infrequently with the use of this technique. The development of infection requires appropriate antibacterial therapy and discontinuation of the occlusive dressing method. Local atrophy and striae have been reported with protracted occlusive dressing therapy. While lesion relapses can be expected to occur in many psoriatic patients, remissions may persist for several weeks to several months in favorable cases. The patient whose psoriasis is in an active stage, with recent appearance of new lesions, may not be a good candidate and may show early relapse. Some plastic films may be flammable, and due care should be exercised in their use. Similarly, caution should be employed when such films are used on or left near children to avoid the possibility of accidental suffocation. **Side Effects:** Side effects are not ordinarily encountered with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. **References:** 1. Cahn, M. M., and Levy, E. J.: J New Drugs 1:262 (Nov.-Dec.) 1961. 2. Meenan, F. O.: J Irish Med Ass 52:75 (Mar.) 1963. 3. Robinson, H. M., Jr., Raskin, J., and Dunseath, W. J. R.: Southern Med J 56:797 (Jul.) 1963.

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MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.

"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

Reduce the blood pressure with Rautrax-N

Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

References: 1. Page, I. H., and Dustan, H. P.: The Usefulness of Drugs in the Treatment of Hypertension, in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 95. **2.** Hollander, W.: The Evaluation of Antihypertensive Therapy of Essential Hypertension in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 97. **3.** Nickerson, M.: Antihypertensive Agents and the Drug Therapy of Hypertension, in Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 3, New York, The Macmillan Co., 1965, p. 727. **4.** Berkson, D. M.: Indust. Med. & Surg. **32**:371, 1963. **5.** Cohen, B. M.: M. Times **91**:645, 1963. **6.** Lee, R. E., et al.: Am. J. Cardiol. **11**:738, 1963. **7.** Moyer, J. H.: Am. J. Cardiol. **9**:821, 1962. **8.** Moser, M.: New York J. Med. **62**:1177, 1962. **9.** Wood, J. E., and Battey, L. L.: Am. J. Cardiol. **9**:675, 1962. **10.** Moyer, J. H., and Heider, C.: Am. J. Cardiol. **9**:920, 1962. **11.** Moser, M., and Macaulay, A. I.: New York State J. Med. **60**:2679, 1960. **12.** Judson, W. E.: Nebraska M. J. **44**:305, 1959. **13.** Hodge, J. V.; McQueen, E. G., and Smirk, H.: Brit. M. J. **1**:5218, 1961. **14.** Moyer, J. H., and Brest, A. N.: Hypertension Recent Advances, Philadelphia, Lea & Febiger, 1961, p. 633. **15.** Berry, R. L., and Bray, H. P.: J. Am. Geriatrics Soc. **10**:516, 1962. **16.** Reid, W. J.: J. Am. Geriatrics Soc. **13**:365, 1965. **17.** Feldman, L. H.: North Carolina M. J. **23**:248, 1962.

Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

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Effective in controlling complicating tetracycline-sensitive bacterial infection and providing symptomatic relief in allergic diseases of the upper respiratory tract.

Contraindication—History of hypersensitivity to tetracycline.

Warning—If renal impairment exists, even usual doses may lead to liver toxicity. Under such conditions, lower than usual doses are indicated and if therapy is prolonged, tetracycline serum level determination may be advisable. Hypersensitive individuals may develop a photodynamic reaction to natural or artificial sunlight during use. Individuals with a history of photosensitivity reactions should avoid direct exposure while under treatment and treatment should be discontinued at first evidence of skin discomfort.

Precautions—Some individuals may experience drowsiness, anorexia, and slight gastric distress. If excessive drowsiness occurs, it may be necessary to increase the interval between doses. Persons on full dosage should not operate any vehicle. Use may result in overgrowth of nonsusceptible organisms. If infections appear during therapy, appropriate measures should be taken. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Infections caused by beta-hemolytic streptococci should be treated for at least 10 full days to help prevent rheumatic fever or acute glomerulonephritis. Use of tetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect has been observed in usual short treatment courses.

Average adult dosage: 2 tablets four times daily, given at least one hour before, or two hours after meals.

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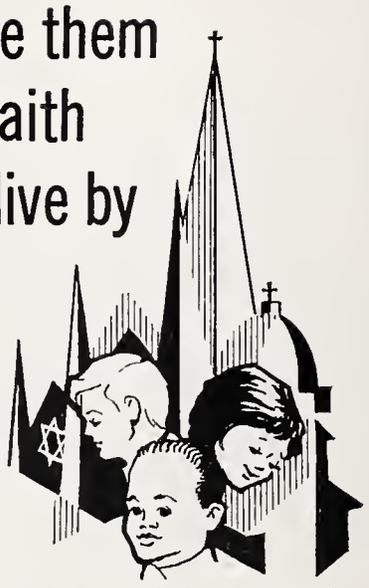

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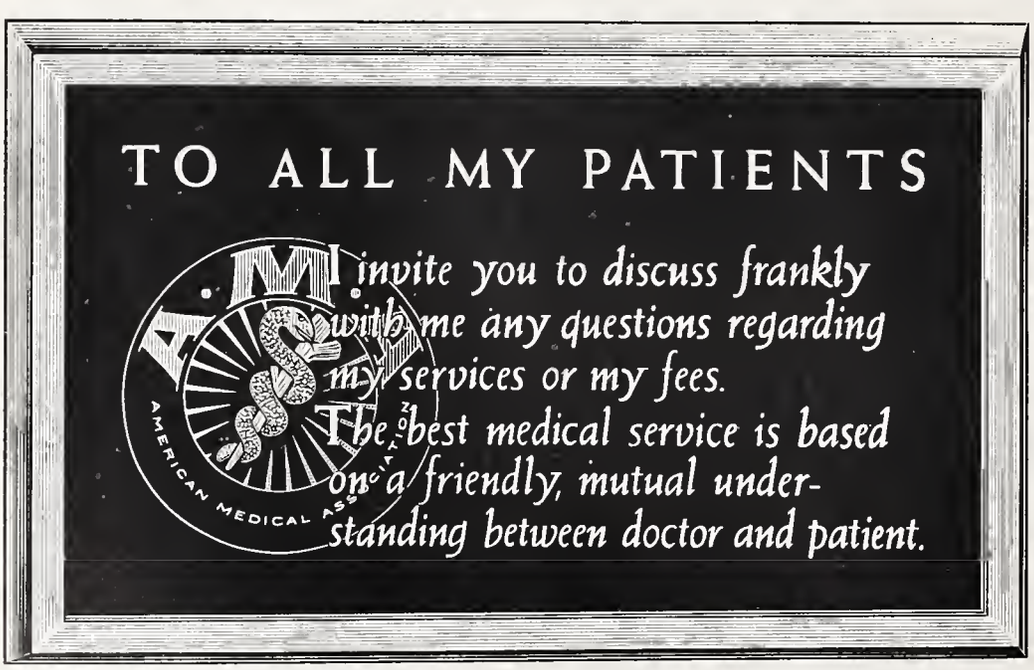
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GENERAL PRACTITIONER WANTED, 30-35 years old with some surgical experience for Texoma area. To be associated with an established 11 man, multi-specialty group which includes general practitioners. Guarantee of $1,000 per month for 1st year followed by full participation without investment. Contact: George F. Erzinger, Business Manager, Medical Arts Clinic, P.O. Box 1242, Sherman, Texas.

OPENING FOR FOURTH PHYSICIAN with group of three in clinic. Facilities of 100-bed hospital immediately available. Practice general surgery, obstetrics, pediatrics, and internal medicine. Salary open. Contact Robert E. Chapman, M.D., 5808 E. Belknap, Fort Worth, Texas.

WANTED: PHYSICIAN to join two man group in Whitney, Texas. Salary first year with privilege to join partnership. New modern clinic, 20-bed hospital existing with new 40-bed, fully equipped hospital ready to undergo construction. Practice includes all phases of medicine and surgery with working arrangements shared by all. Five miles from Lake Whitney, hour driving distance from Dallas, Fort Worth, and Waco. Contact James G. Barton, M.D. or William B. Emmett, M.D. Phone 694-2222 or P. O. Box 546, Whitney, Texas.

DOCTOR WANTED FOR MONAHANS, TEXAS. 12,000 population, 20,000 immediate area. 50-bed, modern Hill-Burton hospital, fully accredited. Stable oilfield maintenance economy. Per capita income high. Seven general practitioners all in solo practice. Two are of retirement age and two are in ill health. Specialists available from nearby town, radiologist on staff. Office space available or association with other doctor if desirable. Climate arid. Excellent deer hunting and mountain resort nearby. Contact Dr. C. S. Patterson, Chief of Staff, or Norman O. Loftin, Hospital Administrator, 406 South Gray, Monahans, Texas, phone WIlson 3-2511.

GENERAL PRACTITIONER NEEDED TO ASSOCIATE with two young GP's who have had general practice residencies. Hurst has a population of 15,000 with average age 24 years. Right between Fort Worth and Dallas in fast-growing "mid-cities" area. Excellent 100-bed hospital with quick consultation availble. New clinic facility with room for three busy doctors. Practice is general and industrial. Contact Aubrey E. Guthrie II, M.D., 752 Mary Drive, Hurst, Texas.

WANTED A GENERAL PRACTITIONER to join a two-man group in year-around vacation land of Rio Grande Valley of Texas. Salary and/or percentage with desire to associate fully in near future. Please reply to Box 105, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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WANTED TWO DOCTORS who are licensed in Texas, or eligible for licensure. Psychiatric experience not necessary, but desirable. Also openings for Board eligible or certified psychiatrists. Salary $16,000 to $19,000, vacation, sick leave, good retirement plan, forty hours per week. No floaters, please. Write James H. Kreimeyer, M.D., Acting Superintendent, Rusk State Hospital, Rusk, Texas.

TEXAS CITY—respected clinic group desires men qualified and interested in: 1) OB-GYN, 2) general practice, 3) industrial medicine 4) internal medicine. If interested in a stable practice with a growing clinic in an active area, contact Stan M. Sheppard, Manager, Beeler-Manske Clinic, 818 5th Street N. Texas City, 77590.

GENERAL PRACTITIONER WANTED. Join group of three; adequate clinic and hospital facilities. Must be young, top-notch man. If you can find a better opportunity—take it. Please reply to Box 110, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED: ENERGETIC GENERAL PRACTITIONER to join present physician in East Texas town. Salary $2,000.00 per month with fringe benefits. Please reply to Box 106, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

WANTED—A GENERAL PRACTITIONER, but would consider internist; to become salaried associate to a six man clinic group who operate an accredited hospital under same roof with clinic in a central Texas town of 10,000; would be on salaried basis for two years as associate and then become owner-partner. Please reply to Charles A. Garrett, M.D., 215 Craig Street, Hillsboro, Texas.

WANTED: GENERAL PRACTITIONER (well-oriented A.A. G.P. man), well qualified GENERAL SURGEON (Board qualified) or INTERNIST. Share building with established GP in individual practice. Lease or rent space. New well-equipped three man clinic—two doctors, one dentist. Plenty of parking. Near progressive shopping center. Convenient to hospitals. Reply to Box 107, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

OPPORTUNITY: Ambitious GP to associate, or otherwise with overworked GP in fully equipped clinic. Choicest surburban area of Houston, Texas, near hospitals, ideal living. Income limited only by ambitions. No money needed. For information write Box 108, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

PHYSICIANS WANTED FOR NEW GROUP—The Medical and Surgical Group of Baytown, Texas, need three general practitioners, one internist, one ophthalmologist, and one E.N.T. To begin January 1, 1967, or thereafter. Financial arrangement will be worked out on an individual basis. Reply to Box 109, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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Wanted Surgeon or General Practitioner to join three man group of General Practitioners in suburb near Dallas. Excellent salary, vacation time and partnership in near future. Contact George W. Apple, Jr., M.D., P. O. Box 158, Plano, Texas.

For Sale or For Rent

IMMEDIATELY AVAILABLE: Fully-equipped clinic of recently deceased physician and surgeon. Patients records and some personnel available. Doctor's widow will introduce. Reply to Lowry Clinic, P. O. Box 2005, Laredo, Texas.

FOR LEASE: A partially equipped upstairs clinic in midst of down-town Conroe with offices for three doctors. Good opportunity for a pediatrician or a dermatologist. If interested contact Mrs. W. M. Holland, 1303 N. Thompson, Conroe, Texas. Phone PL 6-2662.

NORTH EAST DALLAS. New Medical Building just completed, Ferndale and Northwest Hwy. For M.D.'s and D.S.'s only. 12,000 feet of space. Will decorate to suit. In area of 45,000 people with few physicians close. Leasing now. Call DI 8-2320 or RI 2-8265.

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FOR SALE: Fully equipped general practice in the Central Texas area. In 1965 grossed $40,000. Lease on building with or without equipment. Also home available. Write Box 981, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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FOR SALE: One Birtcher Megason Ultrasonic therapy unit, model 105-F, including roll around stand. This unit has underwater head and is in fine operating condition. Price: $275.00. Contact W. L. Lirette, M.D., 21 Heritage Place, Temple, Texas.

ACTIVE PRACTICE AVAILABLE. Due to unexpected death of 55 year old physician and surgeon, (Fellow American College of Surgeons, Fellow International College of Surgeons, Diplomate International Board of Surgery, Fellow International Academy of Proctology and Founders Group of American Board of Abdominal Surgery) active practice available in East Texas town of 15,000. Includes furnished and equipped clinic and services of R.N. with years of experience with long time patients. Town offers 125-bed modern county hospital, fully equipped x-ray and laboratory departments with radiologist and pathologist. Direct inquiries to P. O. Box B.S., College Station, Texas.

FOR LEASE—office for one or two practitioners. 1500 square feet of floor space. Located county seat, Hidalgo County, in Lower Rio Grande Valley, 20,100 population with nine general practitioners at the present time. New 50-bed hospital to be built in the near future. Contact B. R. Stewart, Attorney, Box 330, Edinburg, Texas.

MEDICAL SPACE AVAILABLE October 1, 1966, in modern clinic. 1050 square feet; four examining rooms, private office, lab, rest room. Reception room and business office furnished and shared with dentist. Ample parking. Please reply to 6629 East Bellfort Blvd., Houston, Texas. Call MI 5-1665 or MI 9-3119.

HOUSTON: Moved out of town, must lease 1,500 square feet of new space to specialist. Excluded are fields of general medicine and surgery, and OB-GYN. Located in suburban area one block from hospital. Will modify for occupant's needs. Please reply to F. T. Barfield, Jr., M.D., 1221 Bowie Street, Columbus, Texas 78934.

HOUSTON: Texas Medical Center. Office suite for lease. Thirty three cents square foot. Two thousand square feet or less. Free parking at door. For floor plan and picture of building, write John Hannah, P. O. Box 20032, Houston, Texas 77025 or call MO 8-0471.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.

Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

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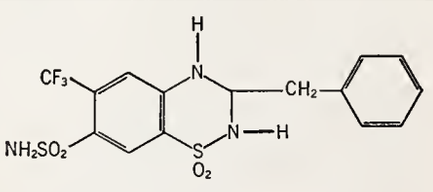

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References: (1) Based on 23 clinical papers, 1512 cases. Bibliography on request. (2) Bush, I. M., Orkin, L. A., and Winter, J. W., in Sylvester, J. C.: Antimicrobial Agents and Chemotherapy—1964, Ann Arbor, American Society for Microbiology, 1965, p. 722.

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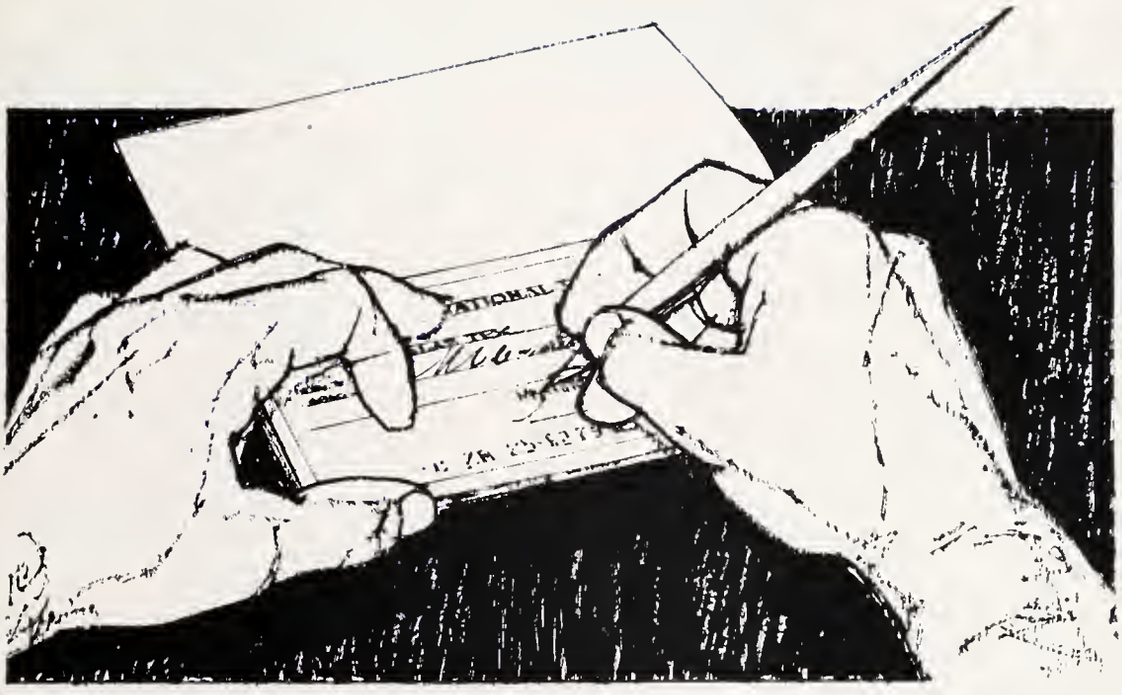

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"...quite apart from the problem of vascular damage, there arises a possibility of virtual 'cure' or remission of hypertension when treatment is early, i.e., before too many other secondary pressor systems have entered into the disequilibrium of pressor control, and when it is adequately suppressive."

Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

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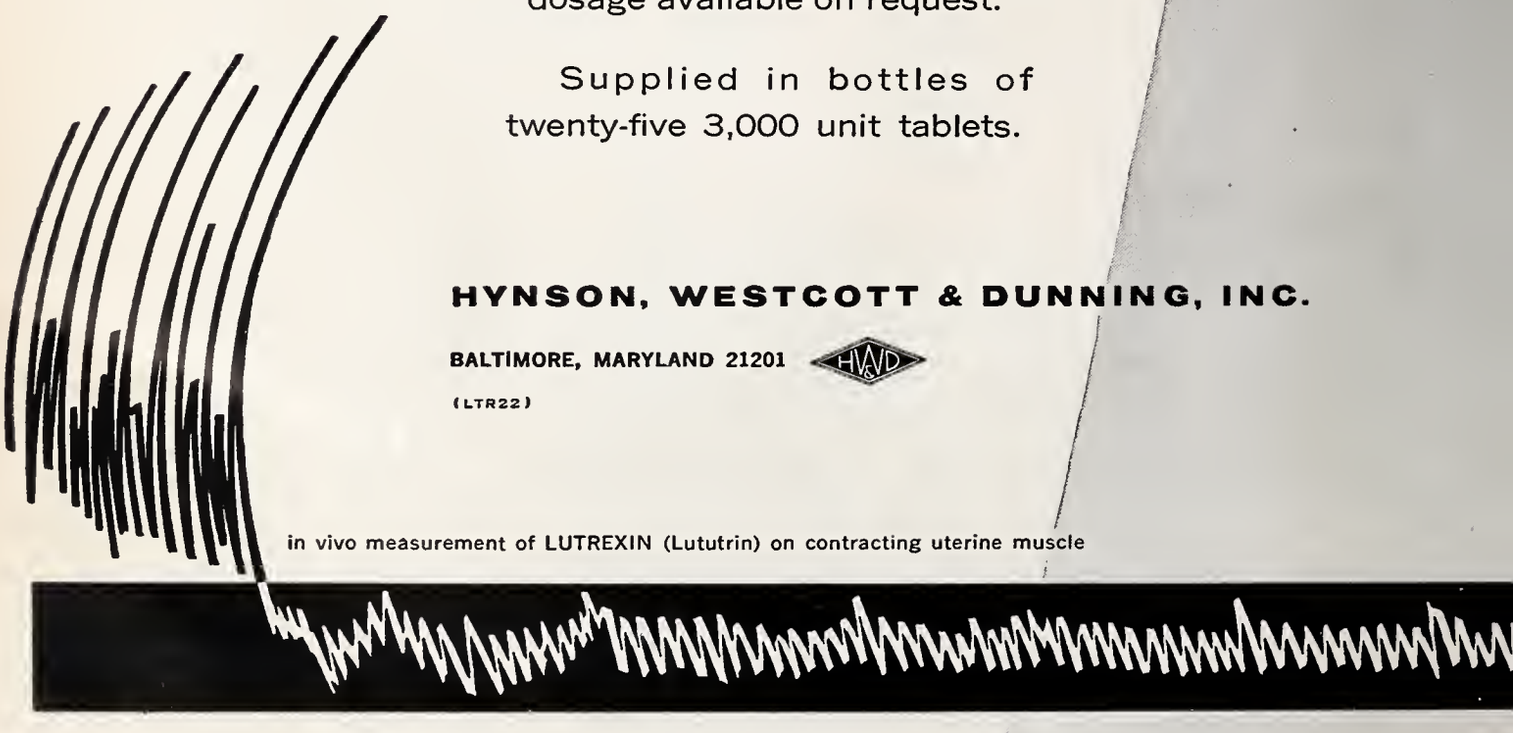

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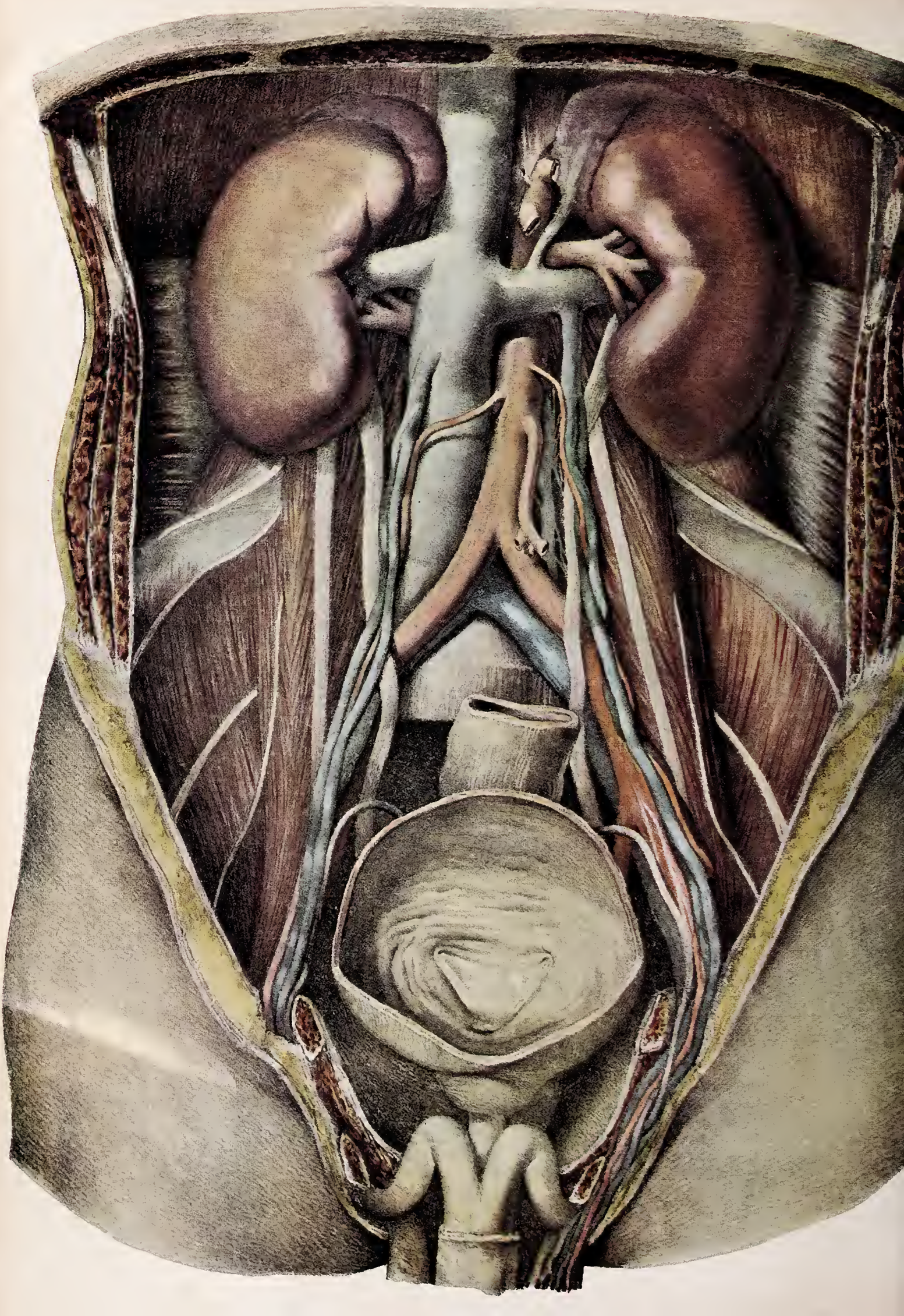

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Contraindications: Hypersensitivity to penicillin; infections due to penicillinase-producing staphylococci and other penicillinase-producing bacteria.

Precautions: If allergic reaction occurs, discontinue ampicillin and administer epinephrine, corticosteroids, antihistamines and/or pressor amines as indicated. Transient moderate elevation of SGOT values of undetermined significance was noted in a few infants. Liver and kidney function as well as hematopoietic tests are advisable during therapy, particularly in infants. As with other antibiotics, precautions should be taken against gastrointestinal superinfection. Safety for use in pregnancy has not been established.

Adverse Reactions: Occasional mild side effects as urticaria, skin rash, pruritus, diarrhea, nausea and vomiting. There have been no reports of blood dyscrasias, liver or kidney damage. Anaphylaxis has been reported.

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Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depressian may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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vignettes of angina pectoris—
no. 1 in a series:

angina and the surgeon

John Hunter—
British surgeon (1728-1793)

angina of anger

"My life is in the hands of any rascal who chooses to annoy and tease me."[1] So said the great British surgeon and anatomist, John Hunter, realizing that he could not control the anger which precipitated frequent and severe attacks of angina pectoris. According to Mettler: "His statement was no exaggeration. On October 16, 1793, he attended a meeting of the St. George's hospital staff, and, while defending the interests of several students, he was contradicted and thoroughly antagonized. The pains of angina commenced, he started toward another room, gained it, and fell dying into the arms of a physician."[2]

Why Edward Jenner withheld his paper on angina In 1777, at an earlier stage of the condition, Hunter's angina alarmed a favorite pupil, Edward Jenner, who wrote to Dr. Heberden that he feared his teacher was "affected with symptoms of the Angina Pectoris."[3] So concerned was Jenner about his former teacher's emotion-related condition that he deliberately cancelled publication of a paper on angina pectoris, fearing that Hunter would read it, and have "his fears excited by its truly formidable nature."[4]

Severity of angina described Hunter's brother-in-law, Dr. Everard Home, who witnessed his death and performed an autopsy, gave this account of the later stages of the condition:

"...the pain became excruciating at the apex of the heart; the throat was so sore as not to allow of an attempt to swallow anything and the left arm could not bear to be touched....

"The affections above described were, in the beginning, readily brought on by exercise... but they at last seized him when lying in bed, and in his sleep...."[5]

18th century ancestor of the modern coronary candidate Surgeon, anatomist, pathologist, physiologist, geologist, and teacher, Hunter had a passion for research which led him to disregard his practice, his health and even the

law. When the Irish giant O'Brien learned that Hunter desired his skeleton for a museum, he willed that his body be sunk at sea in a lead coffin. But Hunter was not to be denied. According to Major, he "... bribed the watchers and finally obtained the body at a cost of 500 pounds although he had to borrow the money to pay the men."[3]
In 1767, he experimentally inoculated himself with gonorrhea and syphilis, treated himself with mercury for three years, and was apparently cured.[5] Hunter had feelings of inadequacy about his education and speaking ability, but this did not prevent him from being hard driving and abrupt with his colleagues. His competitiveness with his physician older brother was also well known, and ended in complete estrangement between the two men.[2,3] Today, the personality traits seen in John Hunter are recognized to be important predisposing factors in the development of coronary artery disease—often manifested as angina pectoris. According to Friedman and Rosenman,[6] in a group of men whose behavior was characterized by intense ambition and competitive drive, a greater than average incidence of angina pectoris was among those abnormal conditions noted.

References: 1. Paget, S., cited by Mettler, C. A.: History of Medicine, Philadelphia, The Blakiston Company, 1947, p. 85. 2. Mettler, C. A.: *Op. cit.*, pp. 84-85. 3. Major, R. H.: A History of Medicine, Springfield, Ill., Charles C Thomas, 1954, vol. 2, pp. 601-607. 4. Baron, J., cited by Major, R. H.: *Op. cit.*, p. 607. 5. Major, R. H.: Classic Descriptions of Disease, ed. 3, Springfield, Ill., Charles C Thomas, 1955, p. 423. 6. Friedman, M., and Rosenman, R. H.: J.A.M.A. *169*:1286, 1959.

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(20 mg. in immediate release layer and 60 mg. in sustained release base)

Peritrate (pentaerythritol tetranitrate) is a nitric acid ester of a tetrahydric alcohol (pentaerythritol).

Actions: The exact cause of angina pectoris (that is, the pain associated with coronary artery disease) remains obscure, despite the numerous and often conflicting hypotheses concerning its pathophysiology. Therapy at the present time, therefore, remains essentially empiric. Customarily, clinical improvement has been measured by: reduction in (1) number, intensity and duration of angina pectoris attacks and (2) necessity for glyceryl trinitrate intake for prevention or relief of anginal attacks.

Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. has been reported in clinical usage to reduce in number and severity the incidence of angina pectoris attacks, with concomitant reduction in glyceryl trinitrate intake.

In the evaluation of Peritrate (pentaerythritol tetranitrate) and Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. in angina pectoris, clinical improvement has been customarily measured subjectively by reduction in number and severity of attacks and necessity for glyceryl trinitrate intake for prevention or abortion of anginal attacks. Individual patterns of angina pectoris differ widely as does the symptomatic response to antianginal agents such as pentaerythritol tetranitrate. The published literature contains both favorable and unfavorable clinical reports. In conjunction with total management of the patient with angina pectoris, Peritrate (pentaerythritol tetranitrate) and Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. have been accepted as safe for prolonged administration and widely regarded as useful.

Animal pharmacology: In a series of carefully designed studies in pigs, Peritrate (pentaerythritol tetranitrate) was administered for 48 hours before an artificially induced occlusion of a major artery and for seven days thereafter. The pigs were sacrificed at various intervals for periods up to six weeks. The result showed a significantly larger number of survivors in the drug-treated group. Damage to myocardial tissue in the drug-treated survivors was less extensive than in the untreated group. Pigs rather than dogs were used because their coronary artery distribution more closely resembles that of human beings. Studies in dogs subject to oligemic shock through progressive bleeding have demonstrated that Peritrate (pentaerythritol tetranitrate) is vasoactive at the postarteriolar level, producing increased blood flow and better tissue perfusion. These animal experiments cannot be translated to human behavior

Indications: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. is indicated for the relief of angina pectoris (pain associated with coronary artery disease). It is not intended to abort the acute anginal episode but is widely regarded as useful in the prophylactic treatment of angina pectoris.

Contraindications: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. is contraindicated in patients who have a history of sensitivity to the drug.

Warning: Data supporting the use of Peritrate (pentaerythritol tetranitrate) during the early days of the acute phase of myocardial infarction (the period during which clinical and laboratory findings are unstable) are insufficient to establish safety.

Precautions: Should be used with caution in patients who have glaucoma.

Adverse reactions: Side effects reported to date have been predominantly related to headache (which may require discontinuation of medication) and gastrointestinal distress which are usually transient with continuation of medication.

Dosage: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg., 1 tablet immediately on arising and 1 tablet 12 hours later (on an empty stomach).

Additional dosage forms
Peritrate (pentaerythritol tetranitrate)—10 mg. and 20 mg. tablets with or without phenobarbital.

Peritrate with Phenobarbital SA Sustained Action—80 mg. pentaerythritol tetranitrate and 45 mg. phenobarbital.

(**Warning:** Tablets containing phenobarbital may be habit forming.)

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"Unlike Aminophylline, this drug is tolerated in large doses and does not cause the gastro-intestinal symptoms so frequently seen with Aminophylline."—**PAUL, W. D., M.D.** State Univeristy of Iowa, Dept. of Internal Medicine.

INDICATIONS: **GLYNAZAN** is indicated in the symptomatic management of bronchial asthma, bronchitis, and pulmonary emphysema.

SIDE EFFECTS: Theophylline may cause central nervous system stimulation such as insomnia, restlessness, or irritability. Theophylline can cause gastric irritation with nausea and vomiting.

PRECAUTIONS: Should not be used more than every 4 to 6 hours. Should not be given within 12 hours following rectal administration of theophylline or aminophylline. Do not use when cough preparations containing theophylline or aminophylline are being administered. Because of the diuretic effect of Theophylline, children under four years of age should be watched for signs of dehydration. Caution is indicated in patients with severe renal and hepatic disease, myocardial damage, hyperthyroidism, and glaucoma.

DOSAGE: The size, frequency, and duration of dosage must be determined by the physician in each individual case. Clinical data lead to suggested doses as follows:

Glynazan Tablets, uncoated, 5 grains (equivalent to 2½ grains Theophylline N.F.): Adult dose—1 to 3 tablets every 4 to 6 hours; preferably administered with water after meals.

Glynazan Elixir (contains alcohol 15%): A palatable elixir containing Glynazan 5 grains (equivalent to 2½ grains Theophylline N.F.) per 5 cc. teaspoonful. **Children over 12 years:** ½ to 1½ teaspoonfuls every 4 to 6 hours. • **6 to 12 years:** ½ to 1 teaspoonful every 4 to 6 hours. • **3 to 6 years:** ½ teaspoonful every 4 to 6 hours. • **1 to 3 years:** ¼ to ½ teaspoonful every 4 to 6 hours. • **Adult dose:** 1 to 2 teaspoonfuls every 4 to 6 hours.

HOW SUPPLIED: **Tablets**—Bottles of 100 and 1000, Elixir—Pint and gallon bottles.

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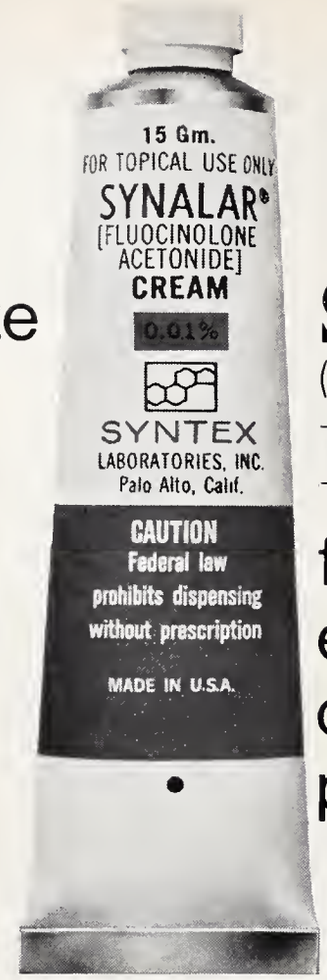

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Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of its components. **Precautions:** 1. *General*—Synalar Cream 0.01% is virtually nonsensitizing and nonirritating. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. 2. *Occlusive dressing method*—With occlusion of extensive areas, systemic absorption of the corticosteroid may occur, and suitable precautions should be taken. Occasional patients may show contact sensitivity to a particular dressing material or adhesive. Miliaria, folliculitis, or pyodermas have been seen infrequently with the use of this technique. The development of infection requires appropriate antibacterial therapy and discontinuation of the occlusive dressing method. Local atrophy and striae have been reported with protracted occlusive dressing therapy. While lesion relapses can be expected to occur in many psoriatic patients, remissions may persist for several weeks to several months in favorable cases. The patient whose psoriasis is in an active stage, with recent appearance of new lesions, may not be a good candidate and may show early relapse. Some plastic films may be flammable, and due care should be exercised in their use. Similarly, caution should be employed when such films are used on or left near children to avoid the possibility of accidental suffocation. **Side Effects:** Side effects are not ordinarily encountered with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. **References:** 1. Cahn, M. M., and Levy, E. J.: J New Drugs 1:262 (Nov.-Dec.) 1961. 2. Meenan, F. O.: J Irish Med Ass 52:75 (Mar.) 1963. 3. Robinson, H. M., Jr., Raskin, J., and Dunseath, W. J. R.: Southern Med J 56:797 (Jul.) 1963.

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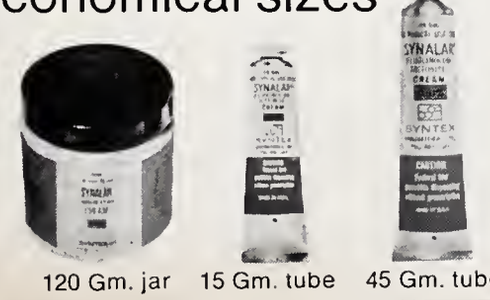

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(tetracycline 250 mg. triacetyloleandomycin 125 mg.)

Indications: Indicated in the therapy of acute severe infections caused by susceptible organisms and primarily by bacteria more sensitive to the combination than to either component alone. In any infection in which the patient can be expected to respond to a single antibiotic, the combination is not recommended. Signemycin should not be used where a bacteriologically more effective or less toxic agent is available. *Triacetyloleandomycin, a constituent of Signemycin, has been associated with deleterious changes in liver function.* See precautions and adverse reactions.

Contraindications: Contraindicated in individuals who have shown hypersensitivity to any of its components. Not recommended for prophylaxis or in the management of infectious processes which may require more than 10 days of continuous therapy. If clinical judgement dictates therapy for longer periods, serial monitoring of liver function is recommended. Not recommended for subjects who have shown abnormal liver function tests, or hepatotoxic reactions to triacetyloleandomycin.

Precautions and Adverse Reactions: Triacetyloleandomycin, administered to adults in daily oral doses of 1.0 gm. for 10 or more days, may produce hepatic dysfunction and jaundice. Adults requiring 3 gm. of Signemycin initially should have liver function followed carefully and the dosage should be reduced as promptly as possible to the usual recommended range of 1.0 to 2.0 gm. per day. Present clinical experience indicates that the observed changes in liver function are reversible after discontinuation of the drug.

Use with caution in lower than usual doses in cases with renal impairment to avoid accumulation of tetracycline and possible liver toxicity. If therapy is prolonged under such circumstances, tetracycline serum levels may be advisable. In long term therapy or with intensive treatment or in known or suspected renal dysfunction, periodic laboratory evaluation of the hematopoietic, renal and hepatic systems should be done. Formation of an apparently harmless calcium complex with tetracycline in any bone forming tissue may occur. Use of tetracycline during tooth development (3rd trimester of pregnancy, infancy and early childhood) may cause discoloration of the teeth. Reversible increased intracranial pressure due to an unknown mechanism has been observed occasionally in infants receiving tetracycline. Glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and definite allergic reactions occur rarely. Severe anaphylactoid reactions have been reported as due to triacetyloleandomycin. Photosensitivity and photoallergic reactions (due to the tetracycline) occur rarely. Medication should be discontinued when evidence of significant adverse side effects or reaction is present. Patients should be carefully observed for evidence of overgrowth of nonsusceptible organisms including fungi, which occurs occasionally, and which indicates this drug should be discontinued and appropriate therapy instituted. Steps should be taken to avoid masking syphilis when treating gonorrhea.

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Pleasant-tasting Alertonic is pipradrol hydrochloride—an effective cerebral stimulant whose gentle analeptic action helps counteract the apathy and inertia that can often delay convalescence—together with an excellent vitamin and mineral formula, in a satisfying 15% alcohol vehicle.

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Each 45 cc. (3 tablespoonfuls) contains: alcohol, 15%; pipradrol hydrochloride, 2 mg.; thiamine hydrochloride (vitamin B_1) (10 MDR*), 10 mg.; riboflavin (vitamin B_2) (4 MDR), 5 mg.; pyridoxine hydrochloride (vitamin B_6), 1 mg.; niacinamide (5 MDR), 50 mg.; choline,† 100 mg.; inositol,† 100 mg.; calcium glycerophosphate, 100 mg. (supplies 2% MDR for calcium and for phosphorus) and 1 mg. each of the following: cobalt (as chloride), manganese (as sulfate), magnesium (as acetate), zinc (as acetate), and molybdenum (as ammonium molybdate).

*Multiple of adult Minimum Daily Requirement supplied.
†The need for these substances in human nutrition has not been established.

Indications: 1. Functional fatigue such as that often associated with: a depressing life experience or stressful time of life; advancing years; convalescence; limited activity or confinement. 2. Poor appetite and vitamin-mineral deficiency as they occur in: patients having faulty eating habits; geriatric patients who are losing interest in food; patients convalescing from debilitating illness or surgery.

Dosage: Adults, 1 tablespoonful; children (over 15 years old), 1 to 2 teaspoonfuls; children (4 to 15 years old), 1 teaspoonful. To be taken three times daily 30 minutes before meals.

Contraindications: As with other drugs with CNS stimulating action, Alertonic is contraindicated in hyperactive, agitated or severely anxious patients and in chorea or obsessive compulsive states.

Side effects: Reports of overstimulation have been rare. Patients who are known to be unduly sensitive to the effects of stimulant drugs should be observed carefully in the initial stages of treatment.

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Effectiveness confirmed by another double blind study*

SUMMARY

1. Forty cases reported.
2. Excellent to good results, 75% with Android, 20% with Placebo.
3. Cites synergism between androgen and thyroid.
4. No side effects in patients treated.
5. Alleviation of fatigue noted.
6. Case histories on 4 patients.
7. Although psychotherapy still needed, role of chemotherapy cannot be disputed.

*"*Sexual impotence treatment with methyl testosterone - thyroid (ANDROID) a double blind study" - Montesano, Evangelista: Clinical Medicine, April 1966.*

CONTRAINDICATIONS — Methyl testosterone is not to be used in malignancy of reproductive organs in male, coronary heart disease, hyperthyroidism. Thyroid is not to be used in heart disease, hypertension unless the metabolic rate is low.

CAUTION: Federal law prohibits dispensing without prescription.

REFER TO PDR

ANDROID
Each yellow tablet contains:
Methyl Testosterone ..... 2.5 mg.
Thyroid Ext. (1/6 gr.) ..... 10 mg.
Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 tablet 3 times daily.
Available:
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ANDROID-HP
Each red tablet contains:
Methyl Testosterone ..... 5.0 mg.
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Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 tablet 3 times daily.
Available:
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ANDROID-X NEW
Each orange tablet contains:
Methyl Testosterone ..... 12.5 mg.
Thyroid Ext. (1 gr.) ..... 64 mg.
Glutamic Acid ..... 50 mg.
Thiamine HCL ..... 10 mg.
Dose: 1 or 2 tablets daily.
Available:
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ANDROID-PLUS
Each white tablet contains:
Methyl Testosterone ..... 2.5 mg.
Thyroid Ext. (¼ gr.) ..... 15 mg.
Thiamine HCL ..... 25 mg.
Ascorbic Acid (Vit. C) ..... 250 mg.
Glutamic Acid ..... 100 mg.
Pyridoxine HCL ..... 5 mg.
Niacinamide ..... 75 mg.
Calcium Pantothenate ..... 10 mg.
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Riboflavin ..... 5 mg.
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Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Warn against hazardous occupations requiring complete mental alertness. Use caution in administering to addiction-prone patients or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In elderly and debilitated and in children over five, limit dosage to smallest effective amount, increasing gradually as needed and tolerated. In general, concomitant use with other psychotropics is not recommended. Paradoxical reactions have been reported in psychiatric patients and hyperactive aggressive children. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Observe usual precautions in presence of impaired renal or hepatic function, impending depression and suicidal tendencies.

Adverse reactions: Drowsiness, ataxia and confusion may occur, especially in elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. Syncope occurs rarely. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis, jaundice and hepatic dysfunction) may develop occasionally, making periodic blood counts and liver-function tests advisable during protracted therapy. Individual maintenance dosages should be determined.

Dosage: *Oral*—Adults: Mild to moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

Contraindications: Patients hypersensitive to antihistamines. Not recommended for use during pregnancy.

Precautions: Until the patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. Administer with care to patients with cardiac or peripheral vascular diseases or hypertension.

Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability, or excitement may be encountered.

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Codeine phosphate.....¼ gr. (No. 2),
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Contraindications: Hypersensitivity to any ingredient.

Precautions: As with all phenacetin-containing products, avoid excessive or prolonged use.

Side Effects: Side effects are uncommon—nausea, constipation, and drowsiness have been reported.

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(tetracycline phosphate complex with analgesics and antihistamine)

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MEDICAL PRODUCTS DIVISION

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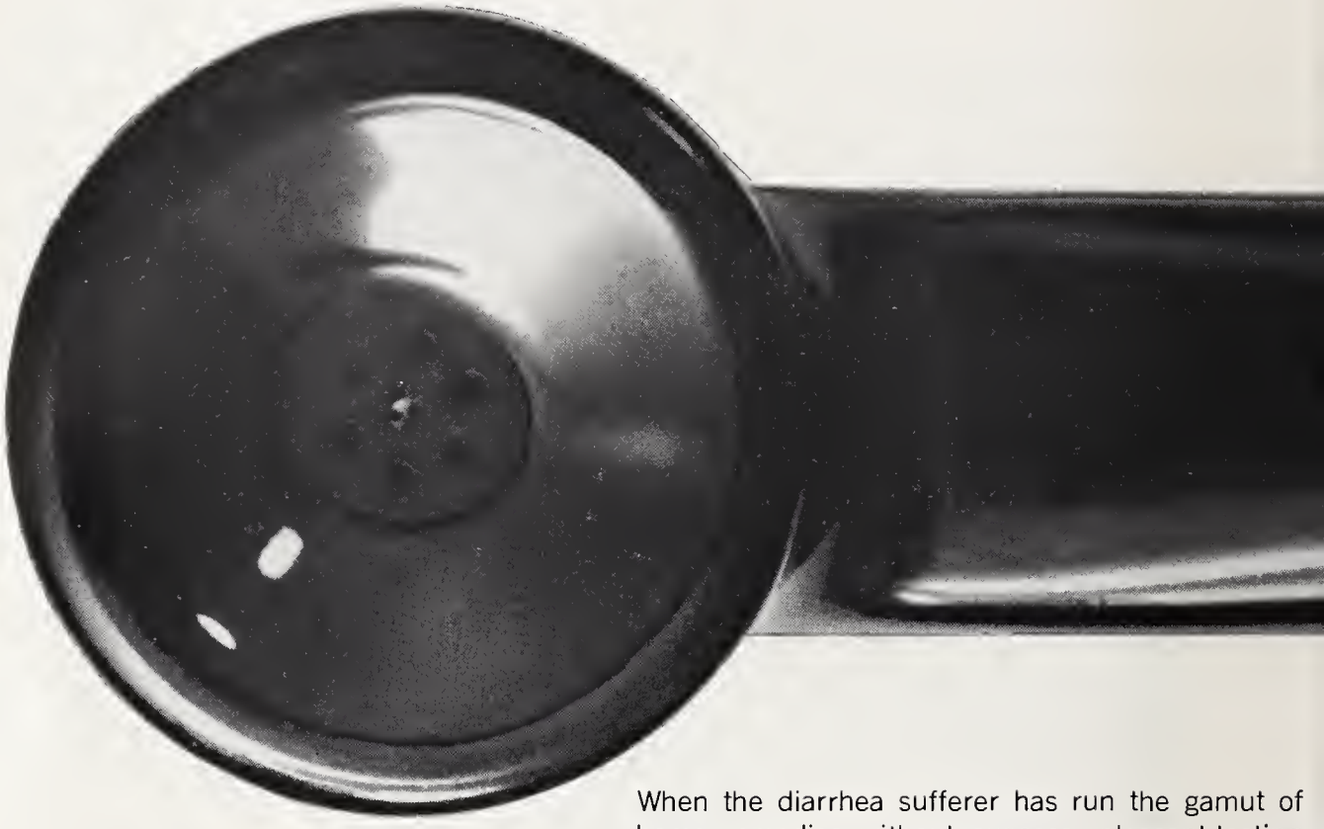
Mount Vernon, Ohio, U. S. A.

When uncontrolled diarrhea brings a call for help



When the diarrhea sufferer has run the gamut of home remedies without success, pleasant-tasting CREMOMYCIN can answer the call for help. It can be counted on to consolidate fluid stools, soothe intestinal inflammation, inhibit enteric pathogens, and detoxify putrefactive materials — usually within a few hours.

CREMOMYCIN combines the bacteriostatic agents, succinylsulfathiazole and neomycin, with the adsorbent and protective demulcents, kaolin and pectin, for comprehensive control of diarrhea.

INDICATIONS: Diarrhea.

CONTRAINDICATIONS: Do not use in intestinal obstruction, extensive ulceration of bowel, or diverticulosis; in hypersensitivity to sulfonamides or neomycin; in pregnancy at term, in premature infants, or during first week of life in the newborn.

WARNINGS: Use only after critical appraisal in patients with hepatic or renal damage, urinary obstruction, or blood dyscrasias. Fatal hypersensitivity reactions and blood dyscrasias reported with use of sulfonamides. Consider periodic blood counts, hepatic and renal function tests during intermittent or chronic use.

PRECAUTIONS: Succinylsulfathiazole: Use with caution if there is history of significant allergies and/or asthma. Continued use requires supplementary vitamins B_1 and K. *Neomycin:* Watch for

your ℞ for Cremomycin can provide relief

curare-like neuromuscular block during anesthesia if neomycin is used preoperatively in large doses when renal function is poor; watch for overgrowth of nonsusceptible organisms. Consider possibility of ototoxicity and nephrotoxicity with prolonged high dosage.

SIDE EFFECTS: As with all sulfonamides: Headache, malaise, anorexia, G.I. symptoms, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, rash, conjunctival and scleral injection, petechiae, purpura, hematuria, and crystalluria have been noted. Reduced fecal output of thiamine and decreased synthesis of vitamin K have been reported. *Neomycin:* Nausea, loose stools.

Before prescribing or administering, read package circular with product or available on request.

promptly relieves diarrheal distress

Cremomycin®

ANTIDIARRHEAL

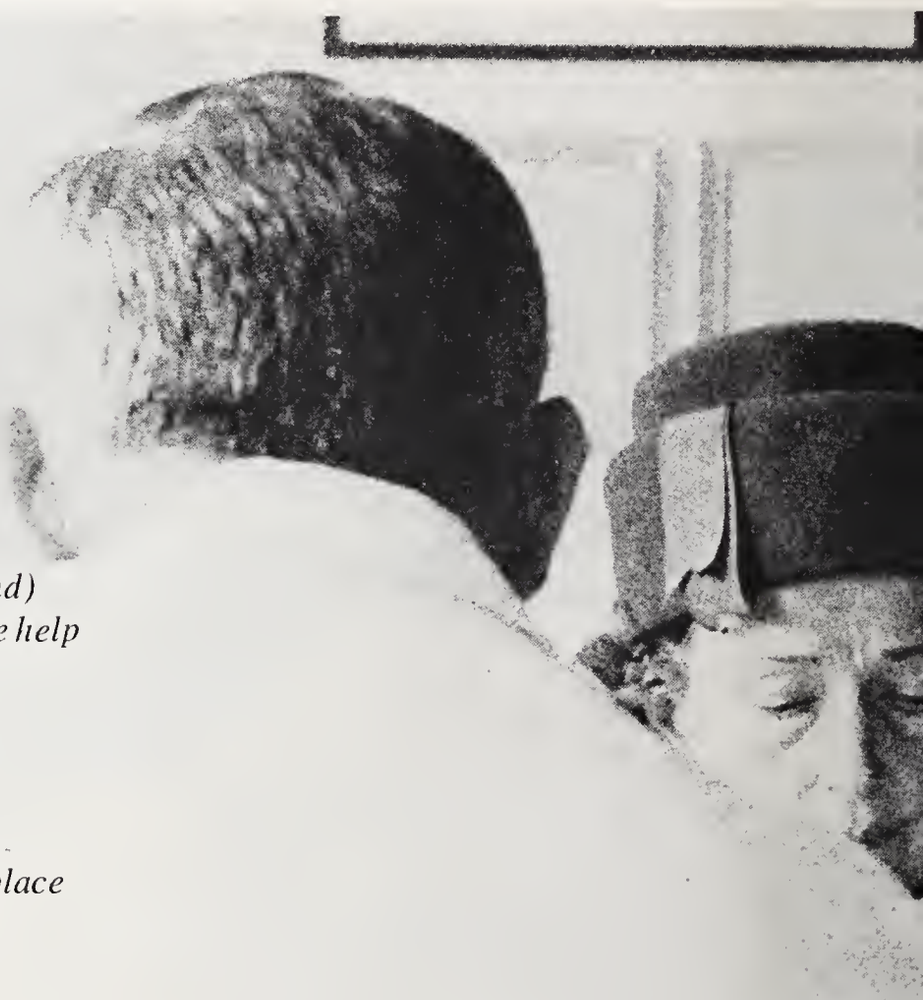
Composition: Each 30 cc. contains neomycin sulfate 300 mg. (equivalent to 210 mg. of neomycin base), succinylsulfathiazole 3.0 Gm., colloidal kaolin 3.0 Gm., pectin 0.27 Gm.

MSD **MERCK SHARP & DOHME** Division of Merck & Co., Inc., West Point, Pa.

where today's theory is tomorrow's therapy

The Mediatric Age:

There is a growing senescent body of people on their way to malignant inactivity, who sorely need your interest and direction to help them back to a more active and useful life. There are medicines too, designed to help. One such has proved useful in clinical practice.



"A steroid-nutritional compound (Mediatric) was used in 100 patients to relieve some of the symptoms caused by degenerative changes of aging . . . This therapy resulted in improvement of 75 per cent of the patients . . ."

McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

"Mediatric (steroid-nutritional compound) capsules, one a day, seem to give definite help to debilitated patients."

Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

"Nutritional and hormone bolstering of function in the aged may have a useful place in geriatrics."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"In diets which for any reason are restricted in calories, enough of these substances (B vitamins) may not be supplied . . . The use of B and C vitamin supplements may then be justified and indeed may be necessary."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"Intensive nutritional therapy is necessary, especially in elderly people, to correct dietary deficiencies created by large losses of protein, vitamins and other nutrients."

Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.

Mediatric®

Designed for the "metabolically spent"

Nutritional reinforcement for those who can't – or won't – eat properly...balanced amounts of estrogen and androgen to counteract declining gonadal hormone secretion and its sequelae of premature degenerative changes...mild antidepressant for a gentle "mood" uplift...

The estrogen component in MEDIATRIC is PREMARIN® (conjugated estrogens—equine), the natural estrogen most widely prescribed for its superior physiologic and metabolic benefits.

MEDIATRIC also provides *nutritional reinforcement—blood-building factors and vitamin supplementation.* It contributes a *gentle "mood" uplift* through methamphetamine HCl.

Three different dosage forms—Liquid, Tablets, and Capsules—offer convenience and variety.

MEDIATRIC Liquid

Each 15 cc. (3 teaspoonfuls) contains:

*Conjugated estrogens—equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Thiamine HCl	5.0 mg.
Cyanocobalamin	1.5 mcg.
Methamphetamine HCl	1.0 mg.

Contains 15% alcohol

MEDIATRIC Tablets and Capsules

Each MEDIATRIC Tablet or Capsule contains:

*Conjugated estrogens—equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Ascorbic acid	100.0 mg.
Cyanocobalamin	2.5 mcg.
Intrinsic factor concentrate	8.0 mg.
Thiamine mononitrate	10.0 mg.
Riboflavin	5.0 mg.
Niacinamide	50.0 mg.
Pyridoxine HCl	3.0 mg.
Calc. pantothenate	20.0 mg.
Ferrous sulfate exsic.	30.0 mg.
Methamphetamine HCl	1.0 mg.

*Orally active, water-soluble conjugated estrogens derived from pregnant mares' urine and standardized in terms of the weight of active, water-soluble estrogen content.

MEDIATRIC helps keep the older patient alert and active; helps relieve general malaise, easy fatigability, vague pains in the bones and joints, loss of appetite, and lack of interest usually associated with declining gonadal hormone secretion.

CONTRAINDICATION: Carcinoma of the prostate, due to methyltestosterone component.

WARNING: Some patients with pernicious anemia may not respond to treatment with the Tablets or Capsules, nor is cessation of response predictable. Periodic examinations and laboratory studies of pernicious anemia patients are essential and recommended.

SIDE EFFECTS: In addition to withdrawal bleeding, breast tenderness or hirsutism may occur.

SUGGESTED DOSAGES: *Male and female:* 3 teaspoonfuls of Liquid, 1 Tablet, or 1 Capsule, daily or as required.

In the female: To avoid continuous stimulation of breast and uterus, cyclic therapy is recommended (3 week regimen with 1 week rest period—Withdrawal bleeding may occur during this 1 week rest period).

In the male: A careful check should be made on the status of the prostate gland when therapy is given for protracted intervals.

SUPPLIED: No. 910 – MEDIATRIC Liquid, in bottles of 16 fluidounces and 1 gallon. No. 752 – MEDIATRIC Tablets, in bottles of 100 and 1,000. No. 252 – MEDIATRIC Capsules, in bottles of 30, 100, and 1,000.

Mediatric®

steroid-nutritional compound

AYERST LABORATORIES, NEW YORK, N.Y. 10017 • Montreal, Canada

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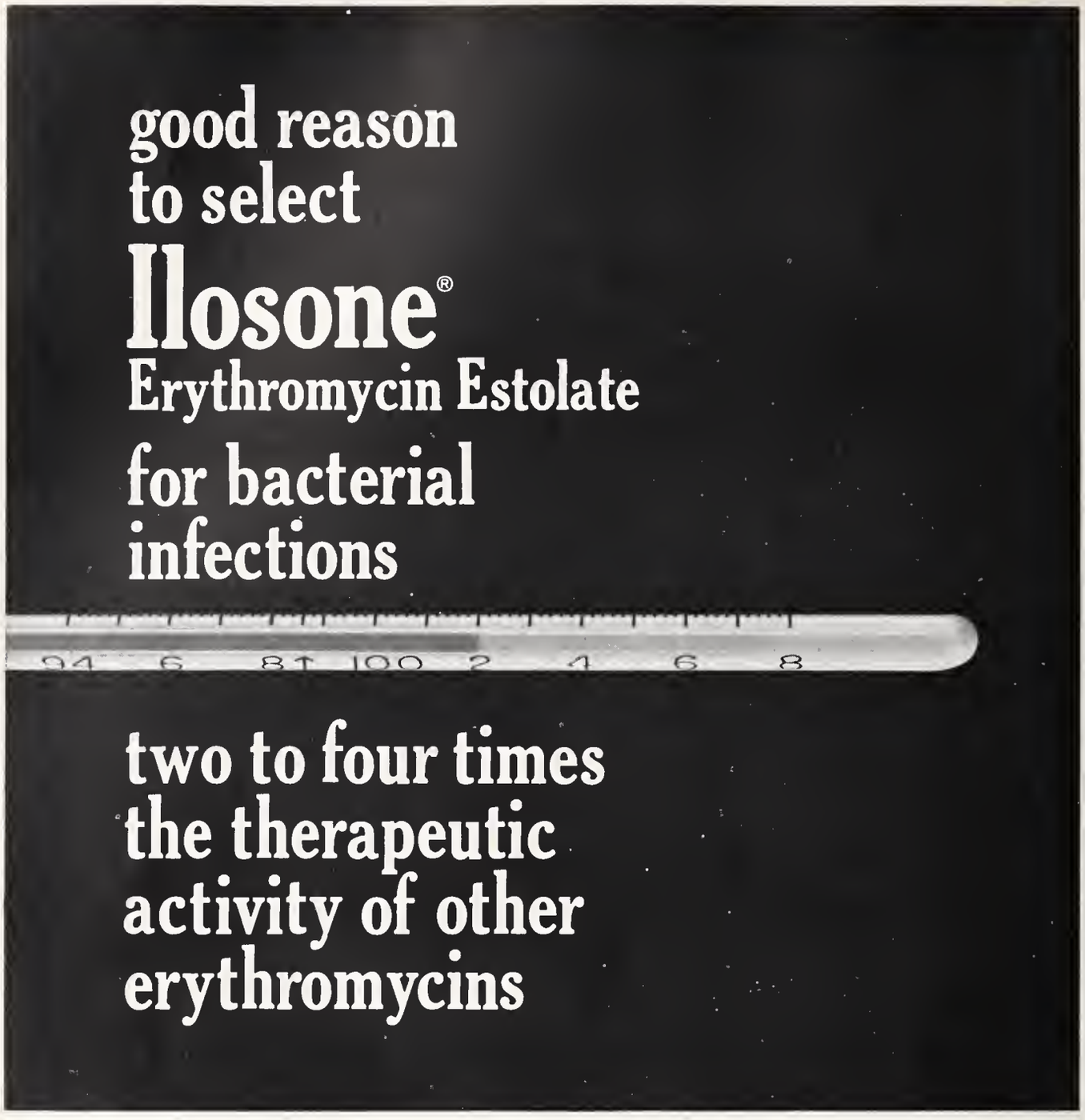

CONTRAINDICATIONS: Ilosone is contraindicated in patients with a known history of sensitivity to this drug and in those with preexisting liver disease or dysfunction.

SIDE-EFFECTS: Even though Ilosone is the most active oral form of erythromycin, the incidence of side-effects is low. Infrequent cases of drug idiosyncrasy, manifested by a form of intrahepatic cholestatic jaundice, have been reported. There have been no known fatal or definite residual effects. Gastro-intestinal disturbances not associated with hepatic effects are observed in a small proportion of patients as a result of a local stimulating action of Ilosone on the alimentary tract. Although allergic manifestations are uncommon with the use of erythromycin, there have been occasional reports of urticaria, skin eruptions, and, on rare occasions, anaphylaxis.

DOSAGE: *Children under 25 pounds*—5 mg. per pound of body weight every six hours. *Children 25 to 50 pounds*—125 mg. every six hours. *Adults and children over 50 pounds*—250 mg. every six hours. For severe infections, these dosages may be doubled.

Available in Pulvules®, suspension, drops, and chewable tablets. Ilosone Chewable tablets should be chewed or crushed and swallowed with water.

Additional information available to physicians upon request.
Eli Lilly and Company, Indianapolis, Indiana 46206.

Lilly

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TEXAS MEDICINE

VOLUME 62 NUMBER 11 NOVEMBER 1966

Devoted to the interests of the medical profession and public health in Texas

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Various Treatments Used For Malignant Exophthalmos

OCULAR PROTRUSION was noted in 1825 by Perry, who first described thyrotoxicosis. Since then, exophthalmos has been listed as a cardinal finding in Graves' disease. Even though the term "exophthalmic goiter" is used synonymously for Graves' disease, true exophthalmos occurs in only 25 percent of the patients. It can occur also in the absence of thyroid disease. The term "malignant exophthalmos" should be reserved for progressive symptomatic exophthalmos rather than cosmetically obvious exophthalmos.

Several theories have been advanced to explain the occurrence of exophthalmos. Its association with thyroid disease has made it tempting to equate the thyroid gland with this problem. However, exophthalmos is not produced by thyroid feeding, so this theory was discarded years ago. In guinea pigs, exophthalmos can be produced by injecting extracts of the anterior pituitary gland or thyroid stimulating hormone (TSH). From pituitary extracts, Dobyns isolated a non-TSH exophthalmos producing substance (EPS).[1] Thus, abnormal pituitary secretion was theorized as the cause of malignant exophthalmos. Recently a plasma protein, long acting thyroid stimulator (LATS), has been suggested as the cause of malignant exophthalmos. In support of this was the finding that LATS levels parallel exophthalmos activity more closely than does TSH or EPS.[2] Thus in effect, our inquiry into the cause of exophthalmos has led us in a circle beginning and ending with the thyroid gland, since LATS is a gamma globulin produced as a result of abnormal thyroid activity.

Various types of treatment have been used in malignant exophthalmos. When the disease is symptomatically mild, which is the case in the vast majority of patients, conservative therapy and thyroid replacement are advised. If palpebral edema becomes

a problem, diuretic therapy and salt restriction may be instituted. Symptomatic improvement may be obtained with prednisone administration. During the past ten years, irradiation has been used as a therapy for this condition. The radiation fields chosen were such that retrobulbar irradiation occurred simultaneously with pituitary irradiation, although the dose was insufficient to produce hypopituitarism.[3] Others have used hypophysectomy as treatment of this condition. Each of these therapies has been tried with modest success. Generally, the only certain method of reducing bulbar protrusion is by one of the various surgical procedures that has been designed for this purpose.

Since LATS has been found to decrease after thyroidectomy and cortisone therapy, total thyroid ablation with radioactive iodine has been suggested as a new treatment for malignant exophthalmos.[4] As with all previous therapies, early reports suggest a beneficial effect, but it is still too soon to determine whether this form of therapy will be permanently beneficial. Medical experience dictates a conservative attitude to this new treatment of exophthalmos. To date, no nonsurgical therapy has produced anything more than a slight decrease in bulbar protrusion, and exophthalmos has increased during and following all types of treatment. The disadvantages of iatrogenic lifelong hypothyroidism should not be traded for slight changes in bulbar protrusion. Since the course of exophthalmos is difficult to determine without prolonged observation and frequent exophthalmic measurements, any new therapy should be confined to individuals in whom the exophthalmos progresses during the observation period and is associated with muscle palsy and or papilledema. The presence of an edematous conjunctiva is an insufficient reason to perform a procedure of this magnitude, which completely destroys the thyroid gland by ^{131}I.

—PHILIP C. JOHNSON, M.D., Houston.

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3. Blahut, R. J., et al.: Exophthalmos Response During Roentgen Therapy, Amer. J. Roentgenol. 90:261 (Aug.) 1963.
4. Catz, B., et al.: Ablation of the Thyroid Gland as Treatment for Malignant Exophthalmos, Clin. Res. 14:176 (Jan.) 1966.

Direct Billing Preserves Physician-Patient Relationship

THE AMERICAN Medical Association, from the time of passage of Part B of Title 18, PL 89-97, has urged physicians to adopt the direct billing option authorized under the law. The Texas Medical Association is vigorously in accord and has worked hard at home and at the AMA level to assure that each physician understands his legal right to bill his Medicare patients directly if he chooses.

In the Oct. 3 issue, the *AMA News* published an editorial on the importance of direct billing. The Board of Councilors urges every physician to read thoughtfully these significant quotations from the editorial:

> In the long run, the willingness to deal directly with the fiscal intermediaries of government will prove to be a serious blunder. It is one matter to accept payments based on determinations of "reasonable" fees by Blue Shield plans and health insurance companies that operate as free agents, usually with close medical society liaison. It is another to accept such direct payments from carriers that must operate as fiscal agents of the federal government.
>
> The interposition between government and the physician or carrier "intermediaries" does not alter the basic fact that the federal government is responsible under Part B, Title 18, PL 89-97, for financing most of the benefits. Under the law the responsible governmental operating agency must assume authority that it cannot delegate to any private group. Inevitably, governmental medical care programs encounter cost control problems. Fee schedules, mandatory generic drug prescribing, elimination of "unnecessary" diagnostic tests and therapeutic agents, and other actions that impinge on the physician's professional freedom, are common developments, usually promoted under the guise of quality control.
>
> Direct billing of the patient makes these developments less likely and preserves a physician-patient relationship relatively free from government control. It carries the risk of non-payment of some

bills, but this loss is more than offset by the gain in professional control of the rendition of medical care.

—Walter Walthall, M.D., San Antonio, Chairman, Board of Councilors.

Physicians Aid Colleagues Whose Needs Are Great

THROUGHOUT HISTORY the physician has been known for his willingness to share his time, his service and his worldly goods with his fellow man. In Texas, he has the Physicians Benevolent Fund, through which he brings aid and comfort to those physicians and their widows whom tragedy seems to follow.

Though only four or five years old, the fund yields income to provide monthly checks to each and every destitute physician, or his widow, who has been brought to the attention of the active and dedicated Committee on Physicians Benevolent Fund. Two actual cases will give an idea of the need:

The first was that of a physician in his ninth decade of life who, together with his wife, subsisted on $68 per month.

The second case is that of a physician who graduated from medical school with high honors. During the first decade of active practice, he was stricken with narcolepsy, which rendered him unable to drive a car. He then obtained employment in an institution where driving was not necessary. But ere long, Parkinson's disease made it impossible for him to work at all. For many years he was bedridden, and required constant care. During the last several months of his life, he received checks from the Physicians Benevolent Fund. But tragedy continued to stalk the family, and within a few days after the doctor's death, his only son was one of the many victims of a sniper's bullet. The physician's widow will continue to receive aid if funds are available and her needs continue.

Thanks to the wonderful cooperation of state and local TMA offices and officers, the state and local Woman's Auxiliaries, and the rapidly growing, ever active Texas Medical Assistants Association, we have a few funds with which to meet the constantly increasing requests. Many county societies are obtaining donations from 100 percent of their members. In fact, most physicians of Texas who are familiar with the functions of the Physicians Benevolent Fund make regular donations.

How much should you give? You are the sole judge. So far, the donations have varied from small amounts to $2,500.

All contributions have the blessings of the Internal Revenue Service, and investment of, and disbursements of proceeds from, the funds are under the watchful supervision of your excellent Board of Trustees of Texas Medical Association.

Checks payable to the Physicians Benevolent Fund may be mailed to: Physicians Benevolent Fund, Texas Medical Association, 1801 North Lamar Blvd., Austin, Texas 78701.

—Denton Kerr, M.D., Houston.

Medical Self-Help Course Teaches Laymen To Survive

SURVIVAL of individual Americans—that is the aim of the Medical Self-Help Training Program—survival of as many as possible in the event of disaster, whether it be hurricane, tornado, or nuclear attack.

Survival demands that people be able to live on their own for an indefinite time, during which they may be isolated from outside assistance by physicians and hospitals. Medical Self-Help training is designed to help people meet elemental health needs in times of disaster.

The program was conceived and developed by physicians, who recognize the physical impossibility of properly caring for patients under disaster conditions. It is advocated throughout the nation by the American Medical Association and in our state by the Texas Medical Association and local medical societies. Initiated by the American Medical Association through its Commission on National Emergency Medical Care, the program was developed by the US Public Health Service with the help and endorsement of medical, hospital, and paramedical groups.

The approval of this training by members

of the medical profession, and their participation in it, have long been recognized as being essential to the success of this effort to prepare citizens for caring for themselves and others under emergency conditions in which no physician is available. The greatest success in bringing this program to fruition is in localities which have had the greatest interest and cooperation from the physicians.

The training is provided without charge to local communities and organizations under sponsorship of county medical societies, local civil defense offices, local Red Cross chapters, and health departments. It consists of 11 lessons given in 16 hours of instruction, namely: radioactive fallout and shelter, healthful living in emergencies, shock, bleeding and bandaging, artificial respiration, fractures and splinting, transportation of the injured, burns, nursing care of sick and injured, infant and child care, and emergency childbirth.

Medical Self-Help goes far beyond first aid principles of "help until the doctor comes." Medical Self-Help assumes that the doctor will not come—that medical help and hospitalization may not be available for days or weeks.

The mission of Medical Self-Help training is to teach people that they can survive disaster, even when professional medical help is not available. The goal is to train at least one person in every family.

Recently a new approach has been taken by the Texas Department of Health and the Texas Medical Association. It was decided to make the course available to junior and senior high school students throughout the state. In this way, a large number of men and women from all parts of the state will eventually possess the desired training. It is encouraging to note that as a result Texas has become the leading state in numbers of persons being trained.

The Texas State Department of Health, in cooperation with the Texas Medical Association, the American Red Cross, and the State Office of Defense and Disaster Relief, has assumed administration of the Medical Self-Help training program on the state level. Training kits, consultation, and educational materials will be dispensed from the State Health Department headquarters in Austin. Any inquiry may be addressed to the Department or to the local medical society.

—Ben A. Merrick, M.D., Dallas,
Chairman, Committee on
Disaster Medical Care.

Sportsmen: Beware of Allergy-Causing Lichens

Hunters and fishermen with a tendency toward skin allergies, or who have a personal or family history of asthma and hay fever, should be forewarned that lichens can cause allergic contact dermatitis and possibly respiratory allergy. The *Archives of Dermatology* reported that there are some 17,000 known species of lichens, about 500 occurring in British Columbia. Usnic acid has been identified in many species of lichens with wide geographical distribution in Canada and the United States.

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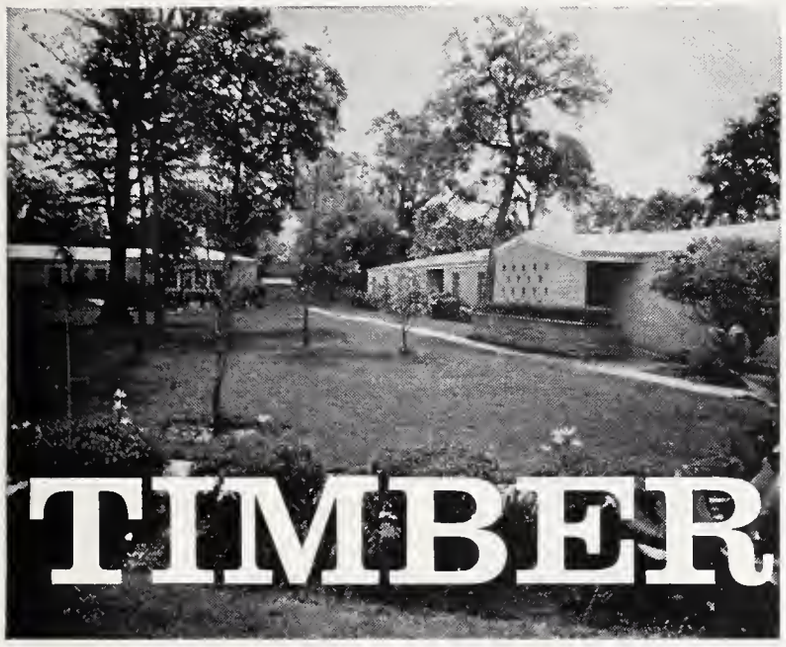

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Vasomist

(ANTIHISTAMINIC)

VASOCONSTRICTOR, NASAL DECONGESTANT AND LOCAL ANTIHISTAMINIC

Active Ingredients:
Phenylephrine Hcl 0.25%
Phenylpropanolamine Hcl . . . 0.50%
Chlorobutanol 0.15%
Tripelennamine Hcl 0.50%
In Isotonic Solution

USE: Temporary relief of nasal congestion due to colds and hay fever.

DIRECTIONS: Adults and Children: Instill two or three drops into each nostril, using no more than needed to provide relief. Repeat every three to four hours as needed but not oftener.

AVAILABILITY

In plastic squeeze bottles containing ¾ fluid oz. and in dropper bottles containing 1 fluid oz.

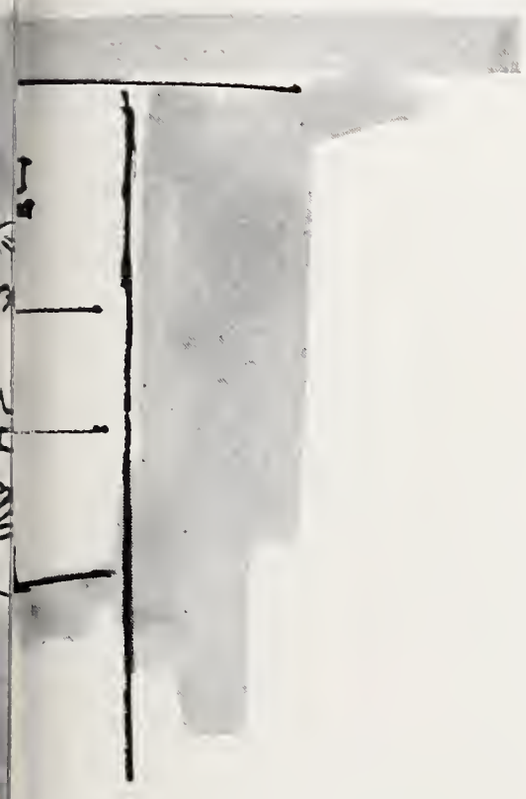
HUMCO LABORATORY
TEXARKANA, ARK.-TEX.

Vasomist

(PLAIN)

VASOCONSTRICTOR AND NASAL DECONGESTANT

Active Ingredients:
Phenylephrine Hcl 0.25%
Phenylpropanolamine Hcl . . . 0.50%
Chlorobutanol 0.15%
In Isotonic Solution



Wait, Annabelle. Mommy hasn't told the doctor yet that you *need* a shot.

Maybe she does, maybe she doesn't. But if her problem is nasal congestion due to cold or allergy, consider the medication that combines convenience and effectiveness—Novahistine Melet.

Melet is especially suitable in the treatment of children. It's a pleasant tasting, chewable tablet that leaves no aftertaste or gritty, undissolved particles in the mouth. It provides decongestant action almost as quickly as a liquid, but assures the accurate dosage of a tablet.

Because Melets are so convenient to carry and so easy to take at any time, adults, too, will appreciate this modern dosage form.

Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution adults who operate machinery or motor vehicles that drowsiness may occur. Each Melet contains phenylephrine hydrochloride 10 mg. and chlorpheniramine maleate 2 mg.

NOVAHISTINE®
MELET

DOW **PITMAN-MOORE** Division of The Dow Chemical Company, Indianapolis

Usual and Customary Fees

The usual and customary fee is the guiding principle for payment under Medicare in Texas. The Texas Medical Association will ask the Texas Legislature to establish this criterion when it implements Medicaid (Title XIX) next year.

Some states use the relative value scale, others have dual fee schedules, and still others determine usual and customary fees as long as the funds are available and when funds are decreased, a reduced fee schedule is used.

The Social Security Administration officials have stated repeatedly that recipients of these two health care programs should receive the same care at the same price as any health consumer.

Blue Cross has a great deal of usual fee information compiled on most physicians in Texas. The physician's usual charges have been averaged, disregarding Old Age Assistance charges, and this average is the usual and reasonable fee for a procedure for that physician. If your charges fall in line with this figure, you will automatically be paid. If the charges are above your average or above that of your colleagues in the community, the fee is questioned and the form is returned for an explanation. If your explanation of charges satisfies Blue Cross reviewers, it will be paid. If it does not, it will be reviewed by an advisory committee of Texas Medical Association representatives appointed to arbitrate questionable fees.

Sincerely,

James D. Murphy M.D.

J. D. Murphy, M.D., President

ANNOUNCING a potent combination in truly delicious orange-flavored forms:

ERYTHROCIN®-SULFAS

ERYTHROMYCIN ETHYL SUCCINATE–TRISULFAPYRIMIDINES

When combination antibiotic therapy is indicated...

CONSIDER: an exceptionally high cure rate in susceptible infections

The rationale: When combined, Erythrocin and the trisulfapyrimidines (triple sulfas) are indicated in infections that are more susceptible to the combination than to either agent alone. Such conditions are usually found in urinary, lower respiratory tract and chronic ear conditions.

The results: Clinical studies involving 142 young patients showed *an overall cure rate of 96.5%*. Side effects were experienced by only four of the patients.

The acceptance: The majority of the 142 patients studied expressed a definite liking for the products. *There were only two refusals.* An independent taste-test with 50 healthy children further substantiated the excellent acceptability of the orange-flavored forms.

602061

ERYTHROCIN®-SULFAS

ERYTHROMYCIN ETHYL SUCCINATE–TRISULFAPYRIMIDINES

In Chewable Tablets
In Granules for Oral Suspension

ERYTHROCIN®-SULFAS
Brief Summary

Indications: Use Erythrocin-Sulfas in infections more susceptible to the combination than to either agent alone. These are usually found in urinary, lower respiratory tract, and chronic ear infections.

Contraindications: Known sensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, premature or new born infants.

Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

If a reaction or overgrowth of nonsusceptible organisms occurs, withdraw the drug.

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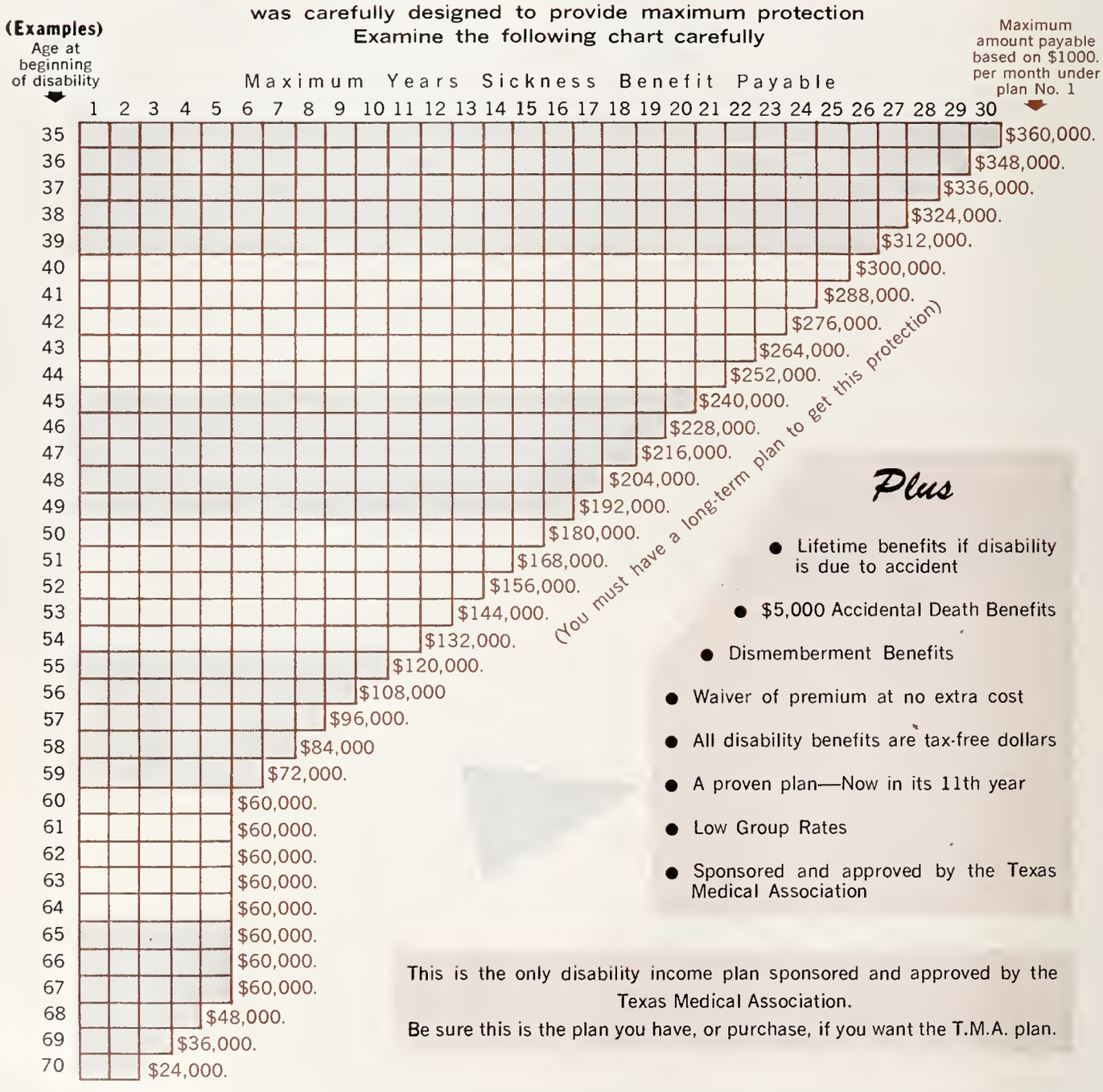

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Gout and the Urologist

REVIEW ARTICLES

THE ASSOCIATION between gout and urinary calculi is woven into the history of our earliest antiquities. The oldest renal calculus extant, recovered from an Egyptian mummy at least 7,000 years old, consists of a phosphatic shell with a uric acid nucleus.[1] Predicated upon reasonable statistics, the chances are within the range of 10 or 15 to 1 that this person suffered from the gout. The famous Ebers papyrus (which dates back to 1,500 B.C.) suggests that gout was a common medical problem during that era, because there are a number of prescriptions in this work which include crocus and saffron herbs, the natural sources of colchicum.[2] The latter is still the only specific therapeutic agent against the acute manifestations of gouty arthritis. The biblical era too, is not without representation in the early history of the gout. Talbott[2] quotes Stukeley, who in 1734 wrote a treatise on the gout. In this work, Stukeley submits that the following quotation from II Chronicles, 16:12, is without doubt a description of the gout: "And Asa in the thirtieth and ninth year of his reign was diseased in his feet, until his disease was exceeding great: yet in his disease he sought not to the Lord, but to the physicians. And Asa slept with his fathers and died in the first and fortieth year of his reign." At this day we can also conjecture that if Asa had gout and was 41 when he died, his death was caused by renal complications of that disease.

The first definite association between gout and stones in the kidney or bladder was proposed by Rufus of Ephesus, (A.D. 98-138). Soon after this, Galenus (A.D. 131-201) pointed out the causal relationship between gout and urinary stones, and expressed the belief that both elements had a common source. He further broadened the etiologic concept of stone disease by attaching importance to the hereditary, climatic, and nutritional factors, and even to racial characteristics. The association between gout and stones in the kidney or bladder was further developed by Paul of Aegina (A.D. 625-690).[2-5]

In more recent history, Paracelsus (1493-1541) was the first of the ancient practitioners to substitute a chemical for a "humoral" basis of both "the gout" and the associated renal calculi: He postulated that both gout and urinary stones were caused by retention of "acrid substances" within the body, not unlike the salts of tartar which were commonly deposited in the wine barrels of his day. He theorized that normal people excreted these substances through their kidneys, whereas the gouty people or those with the "gouty diathesis" accumulated them. He called this concept his theory of the "tartarous disease." [2,4,6]

The history of stones and gout in the 17th century is the story of the "English Hippocrates," Sir Thomas Sydenham (1624-1689). Sydenham developed hematuria seven years after his first attack of acute gout. He graphically described the highly colored urine which he passed, and which precipitated a red, gravelly sediment after standing. Sydenham suffered from gout for 34 years, and his is the first comprehensive treatise devoted to this disease and related urinary pathology.[2,4,7]

The first clinicopathologic findings relating to the metabolic disorder underlying gout were reported by Carl Wilhelm Scheele, a Swedish apothecary, who in 1776 discovered uric acid in bladder stones in the kitchen of his herb shop. He called the new substance "lithic acid." This discovery monopolized his attention for the ensuing ten years, and in 1786 he demonstrated that a small quantity of "lithic acid" was a normal urinary constituent. Working independently, Torben Bergman (1735-1784), also in Sweden, simultaneously discovered that uric acid was the chief component of bladder stones. W. H. Wollaston, in 1797, and Pearson, in 1798, showed that the crystalline ma-

Gout has been gnawing at man's kidneys for all of recorded history. If we look, we may even find its pebbles dropped along the way.

WILLIAM W. HOFFMAN, M.D.

terial in gouty tophi was the same substance as that found by Scheele in bladder stones, some 20 years before.[2, 4, 7]

The next significant contribution to the pathogenesis of gout and its relation to renal lithiasis was made in 1848, when Sir Alfred Garrod demonstrated through the murexide reaction an increased concentration of uric acid in the serum of gouty patients and described it as the "morbid circulating matter in the blood in cases of gout." In 1856, Garrod described his "thread test," in which uric acid was precipitated out of the serum of gouty patients onto a thread suspended in the acidified serum. He recommended that the test be performed at the bedside of the patient. The procedure is, without doubt, one of clinical medicine's first specific diagnostic procedures. Garrod was also the first to clearly document the basic renal pathology of the disease, and described the anatomical studies he made in support of his conclusions.[2, 4, 7]

The final chapter which unlocked the secrets surrounding the chemical pathology of gout was written by Emil Fischer, who published his "Investigations of the Purine Substances" at the turn of the century. In this epochal work, he described the entire process of purine metabolism and first described the synthesis of uric acid. For this work, he received the Nobel Prize in chemistry in 1902.[4]

By following the recorded history of the search for the answer to the gout, we have also traced the history of uric acid lithiasis, which is indeed the early history of urinary stone disease.

Introduction

Gout was originally called *podagra,* from the Greek words for foot and seizure; it was a descriptive term indeed. Talbott traces the derivation of the current term "gout" to the Latin noun "gutta" meaning a drop, or a defluxion of the humors, the visible tophus being a large accretion of the salts of uric acid.[2, 7]

According to our current terminology, the whole spectrum of clinical manifestations of gout is divided in two broad categories:[2, 7-9] primary gout, which describes the clinical state resulting from an intrinsic disorder in purine metabolism, and secondary gout, which describes the clinical syndrome resulting from the overproduction of uric acid in the body caused by increased nucleic acid production and breakdown, usually associated with myeloproliferative disorders and diseases of the hemopoietic or lymphatic systems or the therapy thereof.

Primary gout is generally broken down into four clinical types:

1. Asymptomatic gout (irregular, larvate, or suppressed gout, or gouty diathesis), in which the only demonstrable abnormality may be the passage of renal calculi or an elevated level of serum uric acid, an increased 24-hour uric acid excretion, and an increase in the amount of the so-called miscible metabolic pool of uric acid.
2. Acute gout, characterized by swollen, red, tender, acutely painful joints.
3. Intercritical gout, or the relatively asymptomatic periods between acute attacks, characterized by no symptoms or recurrent low-grade arthralgias.
4. Chronic tophaceous or deforming gout, characterized by the appearance of tophi in soft tissues, or irreparable and deforming periarticular changes.

Urinary symptoms due to stone formation can manifest themselves at any time, or repeatedly, in any of the clinical types of gout. Furthermore, clinically inapparent destructive processes are going on within the renal parenchyma almost continuously throughout the natural history of the disease in any patient. It is important to remember that gout is not a disease limited to the second joint of the great toe. It is a complex metabolic disorder that is frequently associated with an increased incidence of serious degenera-

tive renal disease, and often with hypertension.

Incidence and Pathophysiology

Gout is considered to be a rare disease because it is so frequently misdiagnosed or not diagnosed at all. It is conservatively estimated that there are currently some 800,000 people in the United States afflicted with this malady. In the arthritis clinics of both the Mayo Clinic and the University of Michigan, studies revealed that 5 percent of patients seen because of arthritis were in reality suffering from latent manifestations of gout.[10, 11]

Gout is predominantly a disease of men. In a review of several large series reported, Kaegi[12] found an incidence of 95.5 percent in men and only 4.5 percent in women. These figures recur consistently.[2, 4, 8]

The hereditary nature of the metabolic defect responsible for gout has been recognized throughout the ages. Recent studies, however, have not clarified the exact mode of transmission. Smyth[11] postulates that the hyperuricemia is due to an autosomal recessive gene in some families, but to an autosomal dominant gene in others. Considering the difficulty encountered by those concerned with tracing the heredity of gout,[13] Talbott[2] was probably nearest the truth when he postulated "Taking into consideration the already recognized multiple etiologic agents responsible for hyperuricemia in the gouty patient, one may conclude that several genetic factors may be operating."

The diagnosis of gout is based on three characteristics of the disease entity which all of us have returned by rote during any number of quizzes.

1. A familial history.
2. An increased concentration of uric acid in the serum (above 6.0 mg. per 100 ml. in men or 5.5 mg. per 100 ml. in women).
3. Increased excretion of uric acid in the urine, even when the patient is on a controlled low purine diet. This is generally accepted to be more than 500 mg. for 24 hours.

The latter two characteristics can be explained physiologically by postulating an increase in the miscible metabolic pool of uric acid in patients with gout. That this is an important factor is attested to by the fact that in normal individuals the miscible pool of uric acid is extremely constant, depending chiefly on the individual's physiognomy. The daily turnover of this uric acid is also constant in the normal person, ranging from 0.59 to 0.96 pools per day. In gouty patients, however, the miscible pool of uric acid is at least twice as large as in normal controls, and the daily turnover is distinctively smaller.[2, 3, 14] Recently, isotopes have aided investigations of the uric acid pool and its turnover rate. These sophisticated studies have fairly well proved that formation of uric acid from its metabolic precursors is increased in the gouty patient, but the reason is unknown. However, the answer to the entire problem is not yet at hand, and there is still reasonable argument that incompletely understood metabolic and physiologic disturbances are responsible for the clinical abnormalities associated with gout.[2, 5, 7] Herein, we shall not concern ourselves with these problems.

The Kidney in Gout

Within the kidney itself, the concentration of urate in the glomerular filtrate is the same as it would be in a protein-free filtrate of plasma. The concentration of urate in the urine, however, is up to 15 times greater; indeed, at times a supersaturated solution. Gutman[15] believes that all of the filtered urate is actually reabsorbed by the proximal tubule, and that the urinary urate is the product of distal tubular excretion. The renal clearance of urate in both gouty and nongouty patients is essentially the same, in the range of 10 ml. per minute, and tends to remain constant despite impaired glomerular filtration and urea nitrogen elevation in the blood.[16]

The gouty state is classically characterized by the deposition of crystalline urate in the articular and periarticular tissues. For centuries, since the earliest description of the pathologic changes in gout by Aretaeus in the second century, the deposition of uric acid crystals in the kidney has been repeatedly noted. Virchow described the small, shrunken kidneys resulting from interstitial nephritis, with thickened intrarenal vessels and uric acid crystals in the tubules. Garrod, in his classical treatise on the gouty state, noted the urate precipitate in the pyramidal area as well as in the cortical tissue.[2, 7, 9]

In 1960, Talbott and Terplan[17] reviewed the tissue sections and postmortem protocols of 300 cases in which gout was included in the final diagnoses. The clinical histories of these patients varied from clinical gout or

gouty diathesis to severe tophaceous gout, including cases of secondary gout due to blood dyscrasias. Only four kidneys in the whole series could be called pathologically normal. Most showed either predominantly pyelonephritic or vascular changes. Often it was impossible to determine which process was the primary one. Also, the renal changes tended to parallel the clinical course quite closely. In 11 cases, the process was quite obviously the cause of the patients' recorded hypertensive state. In approximately 20 percent of the patients, the renal changes were the cause of terminal renal insufficiency. The one and only distinctive pathologic feature of the gouty kidney that remained constant throughout the series was the widespread deposition of urate crystals. The collecting tubules in the renal medulla were dilated and filled with urate crystals or small concretions and casts, or were surrounded by small focal areas of inflammation and organized fibrosis. The glomeruli were usually thickened or even hyalinized. In addition, both the cortex and medulla often contained cysts or abscesses filled with pus. Other areas showed pathologic changes of pyelonephritis in various stages of healing. The pathogenesis of the gouty kidney is not yet fully developed. However, there can be little doubt that the high urate level in the glomerular filtrate and renal tubules predisposes to precipitation of uric acid in the renal tubules. Subsequently, the urate crystals invade the interstitial tissues, and the combined result is infection, tubular dilation, and small cyst formation.[18] That such processes are occurring insidiously in patients with gout is evidenced by the physical characteristics of the urine of patients with even subclinical gout, in whom slight albuminuria, pyuria and cylindruria may precede overt symptoms of gout by many years.[19] Indeed, renal degenerative changes, with possible uremia and renal calculi, may well represent the most critical complications of gout. Every patient suspected of having any of the forms of gout should have frequent urinalyses with attention directed particularly to the specific gravity and the presence or absence of albumin, cells, tubular casts, or uric acid crystals in the specimen. The kidneys also should be evaluated at intervals by intravenous pyelography in order to detect gross architectural changes within the hollow structures or direct or presumptive evidence of calculi.

Problem of Gout and Renal Calculi

Urinary calculi occur in 16 to 17 percent of patients who have symptoms or display clinical signs indicating any of the stages of gout.[2, 19, 20] In a review of 10,000 urinary calculi analyzed by means of crystallographic and x-ray diffraction techniques, Herring[21] reported that 944 specimens, or 9.44 percent, were composed wholly or partially of uric acid or uric acid salts. (This is similar to Boyce's experience in eliciting a history of gout in 10 percent of a large series of patients with stones.[2]) Furthermore, 740, or 86 percent of the 944 calculi were composed of more than 50 percent uric acid. The most frequent admixtures were hydroxyapatite and calcium oxylate monohydrate, either as thin laminae between layers of uric acid or as amorphous material filling the interspaces in crystalline or finer granular calculi. Most uric acid calculi range in color from shades of brown to red, and tend to have the smooth, round or ovoid shape, though some are flat and plaque-like. However, because of the processes occurring in the renal parenchyma described previously, which predispose to calculus formation, the gouty patient can form calcium stones without uric acid in them.

There is no characteristic pattern in a gouty patient's history that might suggest a stone-forming stage of the disease. Stones can form in any of the clinical stages of gout and may be the only symptoms of gouty diathesis over and above hyperuricemia and increased 24-hour total uric acid excretion. Talbott[2] reports a patient in whom the first attack of gout occurred 12 years after the first attack of renal colic, in which interval the patient passed several more calculi. An investigation to determine whether gout is present in patients who tend to form calculi should not, however, be limited only to those who repeatedly pass small stones. Gout can obviously be associated with larger calculi and even with so-called staghorn stones. The tendency to form calculi due to the increased excretion of uric acid can be aggravated in the susceptible individual by the uricosuric agents commonly used in the treatment of gout and even by relatively innocuous and commonly used drug substances and compounds such as penicillin, Butazolidin, and certain anticoagulants containing ethyl biscoumacetate and the thiazide diuretics.

An interesting association, discussed by

Talbott,[2] is that of gout and hyperparathyroidism. Though the true simultaneous occurrence of these two entities having renal calculi as one of their clinical manifestations is unknown, there does appear to be a relationship that is more than just chance. This could be a significant consideration in a patient with hyperparathyroidism whose calculi contain an admixture of calcium salts and uric acid or urates.

The problem of calculi in secondary gout, that due to the increased production and breakdown of nucleic acid seen in polycythemia, the blood dyscrasias, and myeloproliferative diseases, particularly during their aggressive treatment with cytotoxic agents or irradiation, is distinctive. These diseases and their therapy can serve as the occasion for the massive formation and passage of stones and so-called gravel.[2, 3, 7, 9, 20] I can recall two instances occurring during the active therapy of leukemia, when the precipitation of uric acid crystals was so severe that the ureters became occluded and the patient became anuric, requiring endoscopic manipulations for relief.

Treatment

In this article, therapy will be considered under several aspects, the first one only briefly.

A pressing problem with which the urologist is often confronted is that of the patient who may have a proven diagnosis of gout or will subsequently be found to be gouty, and who presents with acute symptoms of stone, be it in the renal pelvis, ureter, or bladder. Under these conditions, appropriate urological procedures must be employed to afford the patient relief from his symptoms. The uric acid calculus, under urgent or emergency symptomatic conditions, must often be removed and obstruction relieved. Every practicing urologist is well versed in the management of urinary calculi, and would be expected to apply those methods of relief which would seem indicated in the individual case, and which have been most effective in his hands. In only one case do we make exception to the dictum that "stones don't dissolve," and perhaps some isolated gouty patient was, indeed, the source of the inevitable reference to a far-removed uncle or cousin whose stone a now-forgotten doctor actually did dissolve. Though we have no comparable cases in our files, Atsmon[8, 22] has reported the dissolution and disappearance of renal pelvic and calyceal stones, presumably largely composed of uric acid, in a known gouty patient by oral alkalinization of the urine and a copious urinary output. When a silent pelvic or calyceal stone is discovered during routine evaluation of a patient with gout, one might well institute a rigorous course of therapy which includes alkalinization of the urine to pH 7.4 and concomitantly, barring any medical contraindications to voluminous fluid intake, a daily urine output of 2,000 ml. by actual measure. The usual gout therapy on which the patient is maintained should be continued.[2, 6, 8, 22] Citing a considerable experience gleaned from many years of observation, Talbott[2] argues convincingly that therapy with colchicine and Benemid does not increase the incidence of stones in a gouty patient. The new compound, allopurinol, because of its ability to lower the levels of both serum uric acid and urinary uric acid, may prove effective in the dissolution of renal uric acid calculi when combined with increased fluid intake and alkalinization.[23] At the time of this writing, no such cases using allopurinol in the therapeutic regimen have been reported.

As in any discussion of therapy, one must remember that the treatment should be adapted to each individual case. A patient may have few of the primary or classical manifestations of clinical gout, require no suppressive therapy on this account, and present only with a history of repeated episodes of renal colic with frequent recurrent formation of renal calculi. There are reports of successful repression of stone formation by continuous suppression of hyperuricemia by therapy with a uricosuric agent and high volume (2,000 cc. per day) urine output.[24] Unless the patient's urine is constantly acid, the prophylactic use of alkalizers such as bicarbonate of soda, citric acid, or mixtures of citrate salts is not routinely recommended. Furthermore, Talbott[2] presents valid arguments against stringent dietary prohibitions in the long-term treatment of gout and associated stone problems. He recommends that the only dietary restrictions should be elimination of foodstuffs high in purine substances, such as liver, kidney, sweetbread, and anchovies. Coffee, tea, and cocoa are not restricted nor are alcoholic beverages, taken in moderation, prohibited. The harmful effect of alcohol in gout is related to the quantity consumed, since the

metabolism of alcohol produces a rise in lactic acid in the body fluids which secondarily elevates the serum uric acid.

The role of allopurinol in the standard therapy in gout has not yet been completely worked out, and at the present time it seems probable that allopurinol will not be recommended for the patient with only an occasional episode of acute gout. Yet, our entire concept of the therapy of gout and gouty urinary calculus disease may, in the future, be significantly altered by allopurinol. In a presentation to the American Urological Association in June, 1966, Anderson and associates[25] reported a remarkable series of 12 patients who had formed numerous urate stones before being treated with allopurinol. In every one of these patients, stone formation completely ceased following initiation of allopurinol therapy. Observation of these patients continued for as long as three years.

The prevention of obstructive uric acid uropathy due to the hyperuricemia of secondary gout has often presented a distinctive problem. Frequently the patient is too sick to force fluids, or electrolyte problems preclude the administration of alkalinizing agents. There is every indication that allopurinol will become the agent of choice in the prevention of these complications, especially *during* aggressive chemotherapy or irradiation in the control or attempted cure of the underlying hematologic diseases.[23, 26]

The final therapeutic consideration is the treatment of that insidious complication of gout which is directly responsible for 20 to 25 percent of the deaths in gouty patients—gouty nephritis.[2, 7, 9, 23] In the early case manifested only by proteinuria, and sporadic showers of cellular elements and/or casts with mild impairment of tubular function of slight hypertension, suppressive therapy with colchicine and Benemid has proven its value over the years. Patients on a daily dose of colchicine and Benemid evidence no progression of renal dysfunction and there is evidence that renal lesions already present may reverse themselves when treatment mobilizes uric acid deposits within the organs.[2, 24] The patients with severe renal parenchymal disease (due to gout) who are azotemic should continue their antigout therapy. In addition, the classical supportive measures ordinarily used in the uremic patient should be instituted. Within the last year or two, however, it has become more apparent that allopurinol may actually be the agent of choice in the treatment of advanced stages of gout with severe renal disease. Significantly, early institution of allopurinol therapy, when renal damage is just becoming manifest, may well prevent the development of progressive renal disease. The total role of allopurinol in the therapy of gout is gradually being worked out. The present status of this drug is best summarized by Rundles,[23] who has done a great deal of the pioneer work on allopurinol. "The role of allopurinol in the standard therapy of gout, aiming to achieve the best long-term results with a minimum of risk, expense and effort, can be approximated at this time. A plethora of analgesic and anti-inflammatory agents are in popular use and relatively effective in the temporary control of symptoms. However, agents are now available which can be used on a sounder physiologic basis with better long-term results. For patients who have only an occasional episode of acute gout the symptomatic or prophylactic use of colchicine is probably sufficient. Uricosuric agents are effective in controlling hyperuricemia in a great number of patients who tend to deposit urate in their tissues, but allopurinol may eventually prove to be equally or more effective. In patients with severe disease poorly controlled by standard means, and in those with impaired renal function, urate stones, and nephropathy, allopurinol is the agent of prime importance, best used in many instances in conjunction with colchicine. Determining the full extent to which allopurinol can be used to prevent renal involvement, joint destruction, and progressive increase in disease severity over the course of decades will require careful long-term investigation."

Allopurinol (Zyloprim, Burroughs Wellcome) inhibits the action of xanthine oxidase, thus reducing the amount of uric acid that can be formed from its immediate precursors which are hypoxanthine and xanthine. It also reduces the formation, through the same mechanism, of the normal end product of purine metabolism, thus lowering serum uric acid levels and the daily urinary urate excretion (Fig. 1). In using allopurinol in the treatment of uric acid lithiasis, blockage of the conversion of xanthine and hypoxanthine to uric acid results in the appearance of all three products in the urine instead of uric acid alone. Xanthine is somewhat more soluble than urate in the urine, while hypoxanthine has even greater solubility. The excretion of the three compounds

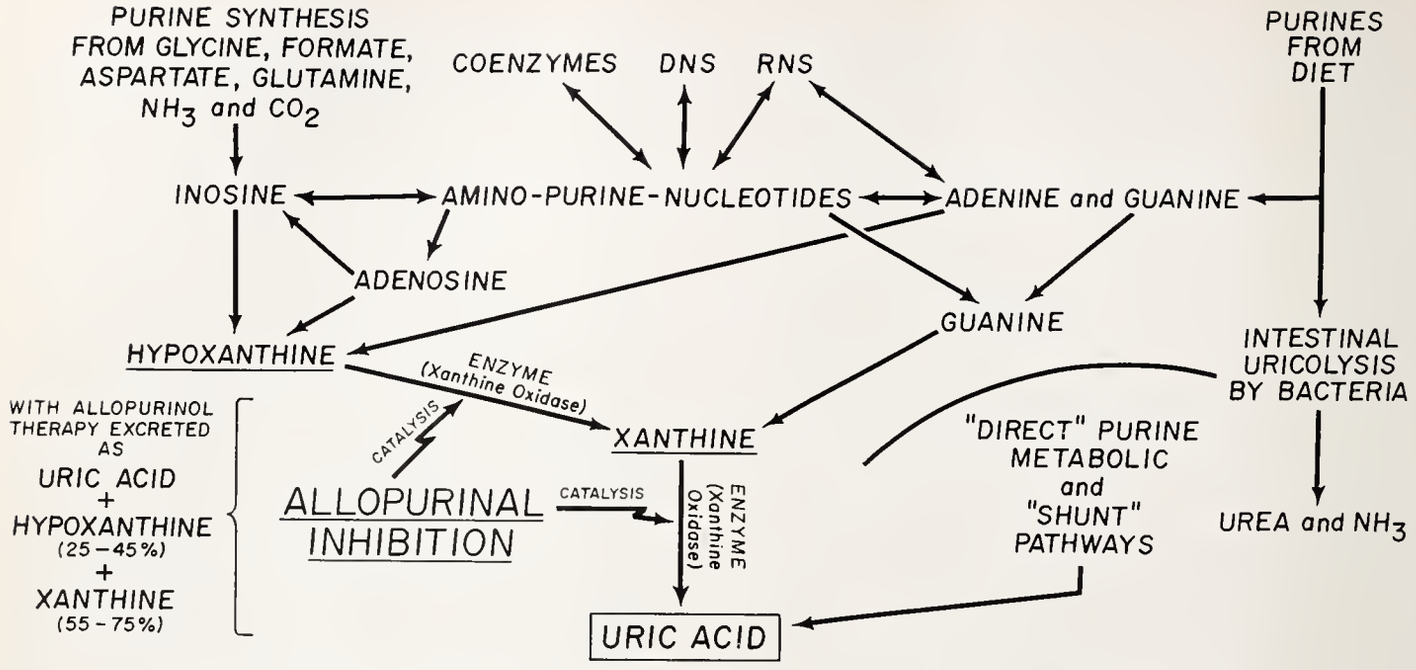

Fig. 1. Intermediate metabolism of uric acid and sites of inhibition by allopurinol.[7, 9, 23, 25, 26]

as end products of purine metabolism obviously decreases the possibility of uric acid stone formation.[23] Xanthine stones do occur but are extremely rare; Herring reports only one in his series of 10,000 calculi.[21] Allopurinol was recently released for general therapeutic use.

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Effects of Acute Ethanol Intoxication On Hepatic Protein Synthesis

Charles F. Johnson, III, M.D.

A chemical and morphologic study of the effects of acute ethanol intoxication on hepatic protein synthesis undertaken by Dr. Charles F. Johnson, III, while he was a student at the University of Texas Medical Branch resulted in a $300 award, the first annual *Texas Medicine* award for scientific writing by a Texas medical student. The Journal Advisory Committee for *Texas Medicine* proudly presents the winning paper in this issue.*

RESEARCH & INVESTIGATION

FATTY INFILTRATION is a well-known occurrence in the liver of the chronic alcoholic. It is generally attributed to a nutritional deficiency of the lipotropic substances choline and methionine. A deficit of these substances impairs phospholipid synthesis which is required for the transport of triglyceride from the liver to the blood as beta-lipoprotein.

Several investigators have recently found that *acute* ethanol intoxication will also result in increased triglyceride in hepatic parenchymal cells.[1-4] The hepatic steatosis is accentuated if a fatty meal is given with the ethanol. The biochemical mechanism responsible for this is unknown.

Physiologically, the amount of fat in the liver is dependent on three factors: (1) the rate with which fat is brought to the liver; (2) the rate of fat synthesis within the liver, and (3) the rate with which fat leaves the liver. It has previously been established that the hepatic lipid accumulation is not due to mobilization of peripheral fat stores.[5, 6] Therefore, the steatosis must be due to increased hepatic synthesis, or decreased secretion, or both, from the liver cell. It is presently believed that one of the derangements responsible for fat accumulation is failure of protein synthesis which results in inadequate formation of lipoprotein.

In accordance with the previous information, this experiment was designed to study alterations of hepatic protein synthesis and morphologic evidence of altered protein synthesis during acute ethanol intoxication.

Methods

Twenty female Holtzman rats, weighing about 150 gm., were fasted for 18 hours and then given an intoxicating dose of alcohol by stomach tube under light ether anesthesia. A 1:1 mixture of 33 percent ethanol and commercial corn oil was given in a dose of 2.0 ml. per 100 gm. body weight. Twenty control rats were given a mixture of glucose and corn oil which was isocaloric with the ethanol and corn oil.

Five hours later, each animal received a 2 microcurie dose of C-14-1-leucine by tail vein. Light ether anesthesia was used here also. The animals were then sacrificed at intervals of 30, 90, and 150 minutes after the leucine injection. Blood samples for protein and radiochemical analysis were obtained by aortic puncture.

The liver was weighed and samples were taken for lipid, protein, and radiochemical

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*Dr. Johnson is now serving as an intern at John Peter Smith hospital in Fort Worth. He extends his gratitude to Dr. F. L. Jennings, chairman of the Department of Pathology at the Medical Branch, who provided the grant and laid the groundwork for the study, which was completed in Dallas under the direction of Dr. Charles T. Ashworth, chairman of the Department of Pathology of the University of Texas Southwestern Medical School, Dallas.

The research project was sponsored jointly by the Departments of Pathology of the two schools. One purpose of the project was to initiate a program which would encourage an exchange of students and ideas between the two institutions for the advancement of medicine and medical education in Texas.

analysis. Histologic blocks were placed in 10 percent neutral formalin. Electron microscopic sections were placed in 2 percent Veronal-buffered (pH 7.4) osmium tetroxide.

Hepatic and serum total lipid and triglyceride were determined by methods previously reported.[7] Total serum proteins were measured by a biuret method,[8] and serum albumin and beta globulins were separated by paper electrophoresis using a Spinco-Durrum cell and an Analytrol. Hepatic total protein and total RNA-protein were obtained in fractions from homogenates by perchloric acid precipitation.[9]

Radiochemical analysis for carbon 14 was carried out on diluted serum, on paper electrophoretic strips containing albumin and globulin, and on total hepatic protein and RNA protein. Liver homogenates were prepared with a Branson sonifier. A Nuclear-Chicago Liquid Scintillation system was used for measurements of radioactivity. Counts were done in duplicate. The quantitative data were subjected to statistical analysis, using the "t" test. Confidence limits were placed at 95 percent.

Tissue for light microscopy was embedded in paraffin and Carbowax. Paraffin sections were stained with hematoxylin and eosin, azure B, and by periodic acid-Schiff methods. Carbowax sections were stained with Sudan black B for fat. Tissue for electron microscopy was embedded in Maraglas.[10] Sections were examined on 300 mesh grids with and without lead hydroxide staining with an RCA EMU-3 electron microscope.

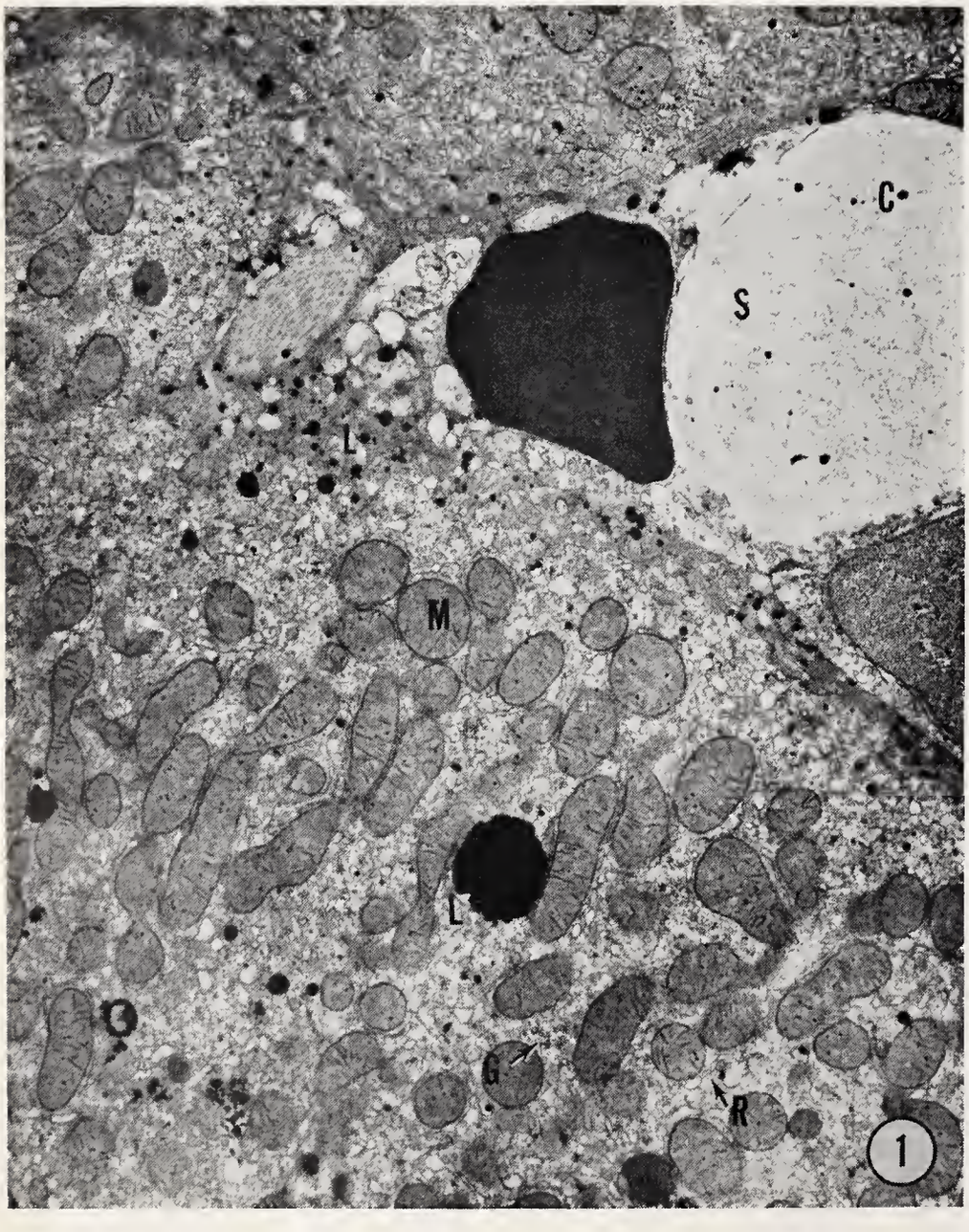

Fig. 1. Liver cells of an alcohol-treated animal, sacrificed seven hours after intoxication. There are some red cells and chylomicrons (C) in the sinusoid (S). The cytoplasm contains large osmophilic lipid vesicles (L), surrounded by a single membrane. They are larger, more numerous, and present deeper within the cytoplasm than in the controls. Glycogen rosettes (G) are virtually absent. The mitochondria (M) and endoplasmic reticulum (R) appear normal. (X 8,360)

Results

All animals receiving ethanol and corn oil showed evidence of intoxication. They were sluggish, ataxic, and developed diuresis. At sacrifice a small amount of oily fluid was present in the stomach and small intestine. There was no gross change in the liver. There was a slight increase (2 percent) in the hematocrit of the intoxicated group.

Histologic Changes in the Liver: In the hematoxylin and eosin sections, the size, granularity, and basophilia of the liver cells of the control animals were normal. There was some cytoplasmic vacuolization in the intoxicated group. The PAS stain revealed decreased glycogen in the control group, and a virtual absence in the experimental group. Sudan black B sections of control animals contained sparse and irregularly distributed cytoplasmic lipid droplets in the centrilobular area; cells of the experimental group had a definite increase in number and size of lipid droplets. These were concentrated at the periphery of the cell. Azure B stain for RNA revealed a normal amount of particulate azurophilic substance in the control animals; the azurophilia was also abundant and granular in the intoxicated animals.

Fat Content of the Liver: Analysis for lipid revealed a significant increase in triglyceride (Table 1). Average triglyceride content of control liver was 0.4 gm. per 100 ml.; average content of intoxicated liver was 1.54 gm. per 100 ml. There was no significant difference in liver weight between the two groups of animals.

Electron Microscopic Changes in the Liver Cell: The major ultrastructural changes were related to cytoplasmic droplets of lipid.

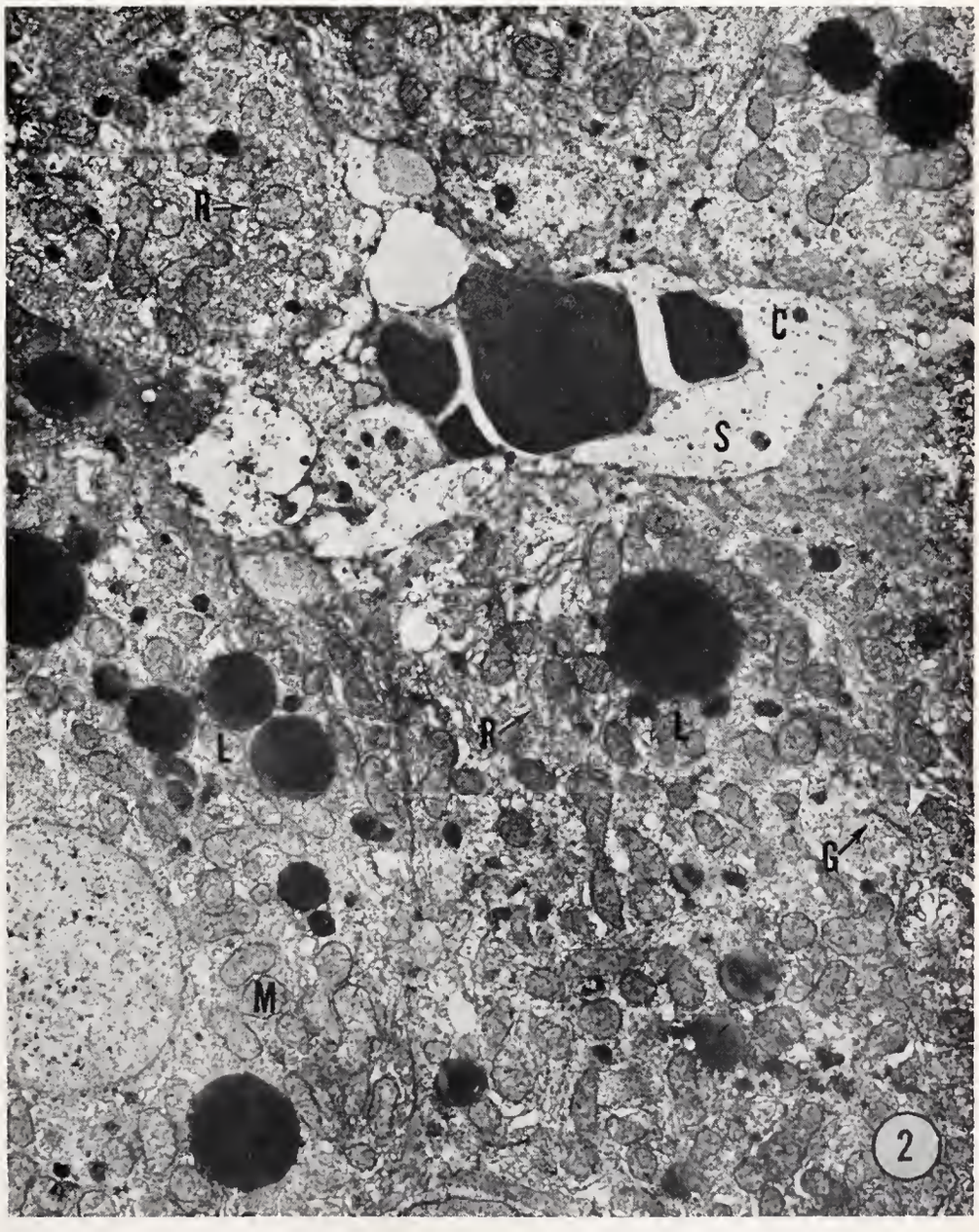

Fig. 2. Liver cells of a glucose and corn-oil fed control animal sacrificed seven hours after feeding. The sinusoid (S) in the upper right hand corner contains a red blood cell and some osmophilic particles which are chylomicrons (C). The lipid (L) is present in the cytoplasm in single-walled vesicles which decrease in size and number as they approach the nucleus. The very small clusters of osmophilic particles are glycogen rosettes (G). The mitochondria (M) and endoplasmic reticulum (R) appear normal. (X 9,500)

For the control animals which received glucose and corn oil, the sinusoidal blood contained an increased number of chylomicrons. The space of Disse also contained chylomicrons which were smaller. Just within the cytoplasmic membrane of the parenchymal cell there were many lipid droplets. These were located in small vesicles surrounded by a single membrane approximately 100 angstroms thick. A few were present in vesicles deeper in the cytoplasm. They decreased progressively in size and number toward the nucleus. Some of the deeper vesicles contained small osmophilic fat particles, but others appeared empty. A few very large lipid droplets were located randomly in the cytoplasm, and there was a membrane surrounding them. Glycogen particles were decreased. No changes were seen in the nucleus, nucleolus, nuclear membrane, Golgi apparatus, or mitochondria. The cisternae of the endoplasmic reticulum were prominent. In most areas they were empty, but occasionally there were scattered aggregates of fine, filamentous structures. Here and there a small lipid particle was present in a cisterna. The ribosomes and polyribosomes were normal.

In the intoxicated animals, which received ethanol and corn oil, the cytoplasmic lipid droplets were more abundant and larger than in controls. They were also found deeper within the cytoplasm. Several vesicles of varying sizes were often found in a localized area suggesting that coalescence was occurring. The margins of the larger lipid droplets were irregular because of indentation by surrounding cytoplasmic organelles. Ribosomal number and organization were unaltered. There were no changes seen in any of the other cell organelles.

Analysis of Hepatic Proteins: Liver protein fractions are recorded in Table 1 and Fig. 3. Hepatic RNA was relatively increased in the intoxicated group 30 minutes after C-14 leucine administration. From that point, there was a decrease in concentration of RNA in both groups, but there was a slightly greater quantity at 150 minutes in the ethanol group. Radiochemical analysis of hepatic RNA showed that there was no significant difference of specific activity in the intoxicated animals and the comparable controls. Total protein analysis revealed significant increases in concentration and in specific activity in the intoxicated animals.

Analysis of Serum Protein: Serum protein fractions are recorded in Table 2. The concentration and specific activity of total serum protein in the intoxicated and control animals did not differ significantly 30 minutes after C-14 leucine administration. However, at 90 and 150 minutes after administration of C-14 leucine, there was an increase in concentration and specific activity of albumin and beta-lipoprotein in the intoxicated animals as compared to the controls. These data are plotted in Figs. 3 and 4.

Discussion

The data demonstrate that acute ethanol intoxication is accompanied by moderate fatty infiltration of the liver. This is in agreement with other previous studies.[1, 2] The accumulation is not striking grossly, but is easily demonstrated on histologic sections and chemical analysis. The mechanism of this change is unknown. Since there is considerable evidence against increased mobilization of fat as the cause of acute ethanol-induced steatosis,[5, 6] this experiment was directed toward evaluating the protein-synthesizing activity of liver cells during the acute phase of intoxication. This was for the purpose of accumulating evidence for or

TABLE 1.—*RNA-Protein and Total Protein Specific Activity; Liver Lipid and Liver Weight.*

Animals	Minutes After Given C-14 Leucine	Liver RNA Protein gm.%	Liver RNA Protein Sp.Ac. (10^5)	Liver Total Protein gm.%	Liver Total Protein Sp.Ac. (10^5)	Liver Lipid gm.%	Weight in gm.
Control	30	2.6	5.0	18.8	2.7	.4	5.6
Ethanol		3.1	4.3	18.6	3.8	1.3	5.0
Control	90	1.6	8.8	18.3	2.5	.4	5.4
Ethanol		1.6	10.3	18.9	3.2	1.3	5.4
Control	150	1.4	9.2	16.8	2.8	.4	5.6
Ethanol		1.7	8.5	17.5	3.4	2.0	5.6

TABLE 2.—*Serum Protein Values and Specific Activity of Serum Albumin and Beta Globulin.*

Animals	Minutes After Given C-14 Leucine	Serum Albumin gm.%	Serum Albumin Sp.Ac. (10^5)	Serum Beta Globulin gm.%	Serum Beta Globulin Sp.Ac. (10^5)	Serum Total Protein gm.%	Serum Total Protein Sp.Ac. (10^5)
Control	30	2.6	2.4	1.2	3.8	6.3	3.8
Ethanol		2.8	2.6	1.0	3.2	5.5	4.4
Control	90	3.1	2.0	.99	4.4	5.9	3.7
Ethanol		3.3	3.3	.96	5.8	6.1	5.3
Control	150	3.1	2.3	.96	6.2	5.9	4.5
Ethanol		3.3	3.3	.90	8.0	5.9	6.2

against the ability of hepatic cells to synthesize lipoprotein.

During ethanol intoxication, the liver cell is not seriously injured structurally. RNA does not decrease and electron microscopy shows no evidence of alteration of mitochondria or other organelles except for a slight increase in number and size of intravesicular lipid particles. Therefore, the derangement responsible for hepatic steatosis is not dependent on structurally recognizable alterations.

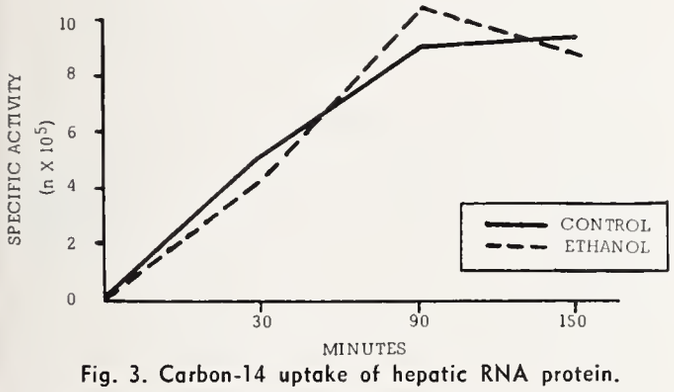

Fig. 3. Carbon-14 uptake of hepatic RNA protein.

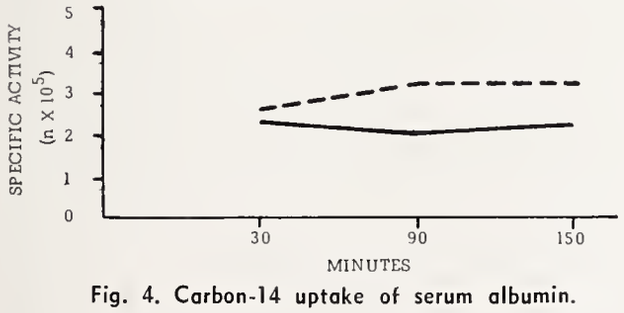

Fig. 4. Carbon-14 uptake of serum albumin.

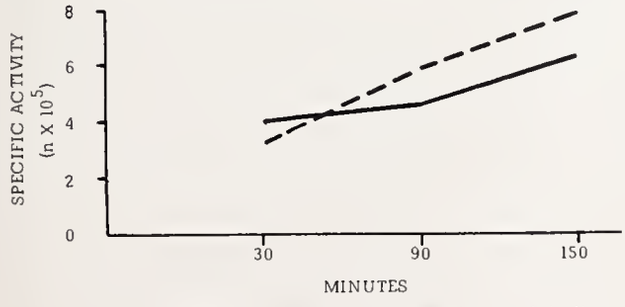

Fig. 5. Carbon-14 uptake of serum beta globulin.

The radiochemical data relating to protein synthesis in ethanol intoxication can be correlated with the morphologic factors. The rate of incorporation of radioactivity of C-14 leucine into total hepatic protein and RNA-protein is increased in the alcohol group in comparison with controls. These observations suggest that in acute ethanol intoxication there is normal or increased activity of the mechanism for the uptake of amino acid and its incorporation into RNA and other cytoplasmic proteins.

It would be possible for increased labeling of hepatic cell protein to occur with normal or reduced rate of amino acid uptake if the final steps in hepatic protein synthesis and the secretion of albumin and globulins were not being completed. Measurement of carbon-14 in serum albumin and beta lipoproteins is an indication of completion of protein synthesis and secretion by the hepatic cells.

Albumin and beta globulin synthesis, as measured by incorporated radioactivity, occurred more rapidly in the intoxicated animals than in the controls. If compared to the normal relative concentrations of albumin and beta globulin, the concentration of beta lipoprotein in the intoxicated animals was increased. However, these animals were absorbing large amounts of triglyceride and the synthesis of beta lipoprotein would be relatively increased under these conditions. These data illustrate that during acute ethanol intoxication the cellular uptake and incorporation of amino acid occur at a normal or possibly increased rate, and albumin and beta globulin synthesis and secretion by the liver cells is slightly increased.

Correlation of the biochemical data of hepatic protein synthesis and the ultrastructure of the liver cells in acute ethanol intoxi-

cation, is met with two major limitations. First, changes in the rate of protein synthesis might not necessarily be accompanied by recognizable cytoplasmic fine structural changes. Second, the ultrastructural location of step-by-step protein synthesis in liver cells is incomplete.

Nuclear and nucleolar size is generally slightly increased in cells undergoing increased protein synthesis, due to increased concentrations of RNA. The absence of any change in the nuclear size and RNA content of the liver cells of the intoxicated group of animals can be explained by the fact that this was an acute experiment; the longest time period between injection of C-14 leucine and sample collection was 150 minutes, and the longest period of intoxication was 7 1/2 hours. Nucleolar enlargement is seen only after several days of increased protein synthesis.

Ribosomes on the endoplasmic reticulum (ER) and those free in the cytoplasm are as abundant in intoxicated animals as in control animals. This is in contrast to the steatosis produced by carbon tetrachloride which causes ribosomes to become detached and disappear from the ER and cytoplasm.[11] The persistence of normal ribosomes and polyribosomes suggests that this parameter of protein synthesis is unimpaired in acute ethanol intoxication.

The synthesis of protein is thought to occur upon the polyribosomes which line the cisternae of the ER. The completed proteins are then "stored" within the cisternae and released as needed into the circulation. Within the ER cisternae of the intoxicated animals there is frequently found finely granular, filamentous structures which probably represent synthesized plasma proteins. This, again, suggests the continuation of protein synthesis and secretion during acute ethanol intoxication.

Summary

Acute ethanol intoxication in the rat results in an increased amount of triglyceride in hepatic cells. The increase in liver lipid is accentuated if a fatty meal is given with the ethanol.

The biochemical mechanism responsible for the increased hepatic lipid is unknown. It is not due to increased mobilization of fatty acid from body fat stores. It appears to result either from increased hepatic fat synthesis or decreased delivery of lipoprotein from the liver cell.

Fine structural alterations and the rate of protein synthesis, using radiochemical methods, were followed in a series of animals intoxicated for 5 1/2 hours. Samples of liver and serum were collected at 30, 90, and 150 minutes after C-14 leucine injection.

It was found that acute ethanol intoxication did not greatly disturb the fine structure of the cell. The primary alteration was a greater abundance of small cytoplasmic lipid droplets occurring in single-walled vesicles. The most surprising observation was that there was a slight but significant increase in the rate of protein synthesis as measured by a greater uptake of C-14 leucine in the serum proteins of the intoxicated animals.

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Frequent or Severe Respiratory Infections in Children

W. J. Oliver, M.D.; R. C. Kelsch, M.D.; and J. V. Baublis, M.D.

As children grow older and acquire more antibodies, they have less frequent bouts with respiratory disease. A systematic approach to evaluation of severe or complicated illnesses is described, as is clinical diagnosis of defective gamma globulin synthesis.

REVIEW ARTICLES

FREQUENTLY PARENTS complain that a child is subject to respiratory infections which are numerous or severe or both. Investigation of this complaint is a challenge, but it may become frustrating to both parents and physician unless care is used in the selection of patients for study. This process of selection is simpler if the usual frequency and characteristics of respiratory infections in the pediatric age group are considered. In a long-term study, Dingle[1] observed a yearly frequency of three to four respiratory illnesses for normal preschool children, and four to five for normal school age children. It is not surprising that a "normal" child can have repeated respiratory infections over a number of years since more than 130 viral agents may cause respiratory disease.[2] The development of antibodies to these agents with increasing age[3-6] suggests that the high incidence of respiratory infections represents a lack of prior immunologic experience. Furthermore, the extent of exposure influences frequency. The toddler who has three school age siblings, each of whom may bring home different agents, may have many more infections than a child without siblings. Likewise, the oldest or "only" child entering school may contract more infections at this age than his peers who have older siblings.

The child with abnormal resistance can be distinguished from normal children by the severity of his illnesses and the etiologic agents. Pneumonia, sinusitis, adenitis, and sepsis are frequent, and pyogenic organisms such as staphylococci, pneumococci, *Haemophilus influenzae*, and *Pseudomonas* are commonly detected on culture. Improvement is slow, recurrence common, and progression to a chronic illness frequent. Although great interest has been focused on infections associated with defects in circulating antibodies, these defects are rare. More common are local factors, congenital or acquired, which predispose to respiratory infection. A useful classification of these is presented in Table 1.

Bronchial Disease

The rare "H" type tracheo-esophageal fistula may produce the picture of recurrent respiratory tract infection in infants. Diagnosis is difficult but a Lipiodol swallow with the patient in the prone position is helpful.

Cystic disease of the lung may result in recurrent infections in children who live beyond the newborn period. The diagnosis may be apparent on roentgenograms and is often suggested by repeated infection in one area of the lung.

Presented before the TMA Section on Pediatrics during the 1965 annual session in San Antonio.

Cystic fibrosis of the pancreas is the underlying etiology in a large group of patients with recurrent, severe respiratory tract infections. The diagnosis can be made by demonstrating an abnormally high concentration of sodium or chloride in the sweat.

Bronchiectasis is an important acquired disease associated with recurrent pulmonary infection. A persistent cough, which never entirely clears, is characteristic of the disease. The copious sputum production observed in adults may not be apparent in children, because they usually swallow their secretions. In most cases the etiology is unknown. Most studies report sinusitis in 45 to 80 percent of patients with bronchiectasis. The association of sinusitis with lower respiratory tract disease should suggest the possibility of bronchiectasis. The diagnosis is confirmed by bronchography. The presence of dextrocardia in a patient with sinusitis and bronchiectasis is diagnostic of Kartagener's triad.

Primary tuberculosis has been implicated as a cause of bronchiectasis.[7] Recurrent pyogenic pneumonia results from external compression of the bronchus by mediastinal nodes or from internal narrowing of the bronchus by endobronchial lesions. The process once initiated may result in bronchiectasis years after the primary complex has healed. A skin test is therefore valuable in most patients who have chronic cough despite the absence of persistent pulmonary lesions on x-ray.

TABLE 1.—*Diseases Associated with Recurrent Respiratory Infection.*

I.	Bronchial Disease
	A. Congenital
	1. "H" type tracheoesophageal fistula
	2. Cystic disease (congenital bronchietasis)
	3. Cystic fibrosis of the pancreas (mucoviscidosis)
	B. Acquired
	1. Bronchiectasis
	2. Foreign body aspiration
	3. Allergy
II.	Pulmonary Disease
	A. Idiopathic pulmonary hemosiderosis
	B. Progressive pulmonary fibrosis
III.	Cardiac Disease
	A. Congenital anomalies associated with pulmonary congestion
IV.	Neurogenic Disease
	A. Aspiration pneumonitis
	B. Familial dysautonomia
V.	Defective Immunoglobulin Synthesis
	A. Hypogammaglobulinemia
	B. Dysgammaglobulinemia

Foreign body aspiration may lead to recurrent episodes of pneumonia and is important in the pathogenesis of bronchiectasis.[8] A careful history is essential since the onset of the pulmonary disease is characteristically explosive and the infections are persistent. Physical examination shows signs of partial bronchial obstruction. This may be confirmed by x-rays obtained in full inspiration and expiration. The presence of bilateral disease does not exclude this diagnosis. Bronchoscopy is diagnostic and therapeutic.

A large group of infants and young children have a history of bronchitis and pneumonitis with each respiratory infection. These attacks may be a manifestation of allergy since with increasing age, some of these children develop classical asthmatic episodes unassociated with infections. This allergic bronchitis is distinguished by the diffuse wheezing usually present. Early in an attack, the relief of dyspnea and wheezing by epinephrine is diagnostically helpful. Later in the illness, when bronchial edema and exudate develop, there is usually no improvement with epinephrine. A strong family history of allergy and the finding of eosinophils in the nasal secretions early in an attack support this diagnosis. Negative skin tests do not exclude the diagnosis.

Pulmonary Disease

Idiopathic pulmonary hemosiderosis and a similar syndrome described by Heiner and associates[9] may masquerade as recurrent episodes of pneumonia. The complex of cough, fever, dyspnea, and pulmonary infiltrates is compatible with infection. The true diagnosis is suggested by anemia, reticulocytosis and right heart failure. The demonstration of iron in the macrophages of the sputum during symptomatic episodes or in the histiocytes found on lung biopsy specimens is required to make the diagnosis. Since some of these patients have milk precipitins in their sera and have improved on a milk-free diet, such a diet has been recommended.[9] Our patients have not responded to this regimen but some have improved with steroid therapy.

Idiopathic progressive pulmonary fibrosis is a chronic disease with frequent exacerbation of respiratory symptoms (cough, dyspnea, and cyanosis).[10] It is rarely confused with recurrent infection since the roentgenogram does not show the lungs to be clear between symptomatic attacks.

TABLE 2.—*Immunoglobulins in Man.*

Class	Synonyms	Adult Levels (mg./100 ml.)	Cord Levels (mg./100 ml.)	Age When Adult Level Reached
IgG	γ2, γss, 7s	600-1,200	Slightly higher than maternal levels	3-4 years
IgA	γ_{1a}, B_{2a}	175-600	0.1-1.0	12-13 years
IgM	γ_{1m}, B_{2m}	60-480	3-24	2 years

Cardiac Anomalies

Cardiac anomalies which produce a significant increase in pulmonary blood flow are associated with an increased incidence of lung infections. The presence of a heart murmur simplifies the diagnosis. The absence of a striking murmur in patients who have an anomalous venous return, congenital mitral obstruction, atypical ductus arteriosus, or atrial septal defect may require cardiac catheterization and other studies to make a diagnosis. The presence of increased vascular markings on the roentgenogram between episodes of infection should alert the physician to these cardiac anomalies as the cause of recurrent respiratory infection.

Neurogenic Disorders

Repeated aspiration may be the cause of recurrent respiratory infection. It is most common in children who have severe brain damage, but does occur in children who have minimal damage and nocturnal seizures.

Familial dysautonomia is associated with frequent respiratory infection. These infections have been attributed to the lack of a coordinated swallowing mechanism or to increased bronchial secretions. The diagnosis should be suspected in children of Jewish parentage who cry without tears and have no fungiform papillae on their tongues.[11] It may be supported by the histamine flare test,[12] or by measurement of catecholamine metabolites in the urine.[13]

Defective Immunoglobulin Synthesis

Serum antibodies, arising as a normal response to infection, are associated with the gamma globulins. There are three immunochemically distinct components of this group which have been well studied: IgG, IgA, and IgM. These three, and IgD, a newly described globulin,[14] are referred to as the immunoglobulins. On paper or cellulose electrophoresis, the majority of the gamma globulin peak is IgG. The other components are more readily demonstrated by immunochemical methods. The characteristics of the immunoglobulins have been reviewed recently by Fahey[14-16] and are summarized in Table 2. Children with deficient immunoglobulin synthesis may have frequent and severe bacterial infections of the respiratory tract.[17] A variety of syndromes associated with these defects has been studied by West and associates;[18] they are listed in Table 3. Most of these children will have reduced levels of gamma globulin as determined by paper electrophoresis, but some diagnoses require the use of immunochemical methods.

Interpretation of immunoglobulin levels must be made cautiously, since these concentrations are age dependent. Gamma globulin levels in the newborn infant will be equal to or slightly higher than those of his mother. By three months of age most of the gamma globulin acquired from the mother will be catabolized. At this time normal infants may have gamma globulin concentrations as low as 200 mg. per 100 ml. Lower levels are encountered in infants with delayed synthesis. This situation may persist beyond the first year of life. During this time these infants may have frequent or severe infections. These children differ from those with

TABLE 3.—*Classification of Defects in Immunoglobulin Synthesis.*

I. Hypogammaglobulinemia
   - A. Congenital
     1. Bruton type
     2. Swiss type
   - B. Acquired
     1. Physiologic, of infancy
     2. Associated with granuloma or neoplasm
     3. Idiopathic

II. Dysgammaglobulinemias
   - A. Deficiency of IgG and IgA with increased IgM
   - B. Deficiency in IgA and IgM with normal IgG
   - C. Deficiency in IgA (Ataxia telangiectasia)
   - D. Deficiency in isoagglutinin with increased IgA (Aldrich's syndrome)

congenital hypogammaglobulinemia in that they have gamma globulin concentrations greater than 100 mg. per 100 ml. and may show some antibody response on challenge. If these children can be supported through the transient phase, the prognosis is excellent.

Children with congenital hypogammaglobulinemia of the Bruton type (Table 3) are distinguished from those with acquired varieties by the earlier age of onset and lower concentrations of IgG (<25 mg. per 100 ml.). They are also deficient in IgA, IgM, and lymphoid tissue but may have a normal white blood cell count and differential. These children can develop delayed sensitivity and will reject homografts. The defect is inherited as a x-linked recessive trait.

Children with the Swiss type of congenital hypogammaglobulinemia have thymic agenesis and lymphopenia in addition to the immunoglobulin defects characteristic of the Bruton type. They also lack the ability to develop delayed sensitivity and will tolerate homografts. Disastrous reactions to smallpox vaccination have occurred in these children.[19, 20] The Swiss type appears to be inherited as an autosomal recessive trait. Children with this defect usually die during the first year of life despite therapy.

The acquired hypogammaglobulinemias may affect both sexes and all ages, and are frequently associated with collagen disease and leukemia. They may occur alone or in association with other disease states. In general, these patients will have levels of IgG higher than 100 mg. per 100 ml.

A group of defects in synthesis, the dysgammaglobulinemias, have been described in which one immunoglobulin is normal or increased and others are decreased or absent. They are diagnosed on the basis of a quantitative determination of the individual immunoglobulins. The significance of IgA deficiency is difficult to appraise since it has been described in normal individuals as well as in patients with ataxia-telangiectasia.[21] Paradoxically, patients with Aldrich's syndrome (eczema, thrombocytopenia, recurrent infection) have elevated levels of IgA.[18] Despite an absence of isohemoagglutinins, children with Aldrich's syndrome may have normal levels of IgG and IgM and show a normal response to vaccines.[22] LoGrippo and associates[23] have described patients with recurrent infections who have an apparent inability to form antibodies to enteroviruses, but are normal by other immunologic criteria. The frequency and significance of this phenomenon has not been fully investigated.

In most general hospitals, it is possible to investigate the immunologic status of a child.[24] The essentials of such an investigation are as follows.

Detailed History.—It should be stressed that children with disorders of the immune system have frequent and/or severe bacterial infections, such as pyoderma, pneumonia, and otitis media. In contrast, the course of acute viral respiratory infections and childhood exanthems is usually uncomplicated. The familial nature of hypogammaglobulinemia and its association with other collagen diseases (rheumatoid arthritis, lupus) makes a careful family history essential.

Physical Examination.—These children may show the effects of chronic and recurrent infections by impaired growth. The absence of tonsillar or adenoidal tissue without a history of tonsillectomy suggests congenital hypogammaglobulinemia. Older children with this disease may also have rheumatoid disease.

Routine Laboratory Studies.—The presence of a persistent lymphopenia suggests the Swiss type of hypogammaglobulinemia. The absence of adequate platelets is an integral feature of Aldrich's syndrome. Episodic neutropenia has occurred in some children with hypogammaglobulinemia.

Isohemagglutinins.—The natural isohemagglutinins to blood group antigens are usually absent in patients with congenital or acquired hypogammaglobulinemia. They may be absent in those with Aldrich's syndrome and in some patients with dysgammaglobulinemia.

Vaccine Response.—The response to a bacterial vaccine is usually absent in both congenital and acquired hypogammaglobulinemia. If the child has received routine immunizations, measurements of serum levels of antibodies to pertussis or diphtheria can be sought. The Schick test performed after a booster immunization serves as a readily available screening test. Since most laboratories routinely perform measurements of typhoid-paratyphoid serum antibodies, these determinations are frequently

TABLE 4.—*Response to Typhoid-Paratyphoid Immunization in a Normal Child and in a Child with Hypoimmunoglobulinemia.*

	Normal Child				Hypoimmunoglobulinemic Child			
	Typhoid		Paratyphoid		Typhoid		Paratyphoid	
	"O"	"H"	'A'	'B'	"O"	"H"	'A'	'B'
Pre-Immunization	—*	—	—	—	—	—	—	—
2 Weeks	80†	20	100	160	—	40	—	—
3 Weeks	160	80	160	160	—	20	—	—
4 Weeks	160	160	160	160	—	20	—	—

*Undetectable at 1/1 dilution.
†Reciprocal of greatest dilution giving positive test.

the easiest to obtain. The response of the hypoimmunoglobulinemic patient to typhoid-paratyphoid vaccine is either absent or poor when compared with the normal response (Table 4).

Measurement of Immune Globulins.— Methods for measuring total serum protein concentrations and performing serum protein electrophoresis upon paper or similar supporting media are available in most hospitals. If the total serum gamma globulin is greater than 400 mg. per 100 ml., as calculated from these determinations, hypoimmunoglobulinemia is unlikely. A comparison of serum protein patterns, determined by paper electrophoresis, from a normal child and a child with hypoimmunoglobulinemia is shown in Fig. 1. Levels of total gamma globulin below 400 mg. per 100 ml. require that quantitative determination of the various immunoglobulins be made. Immunoelectrophoresis can be used for this purpose, but requires considerable equipment, experience, and time to develop a standardized technique. A simpler and more efficient method employs prepared immunodiffusion plates which can be obtained commercially.

Management of patients with hypoimmunoglobulinemia involves replacement therapy with gamma globulin. The initial dose is 0.7 ml. per lb. of body weight, and the maintenance dose is 0.3 ml. per lb. every three to four weeks. Gamma globulin concentration of at least 200 mg. per 100 ml. should be maintained. Specific antibiotic treatment based upon culture and sensitivity studies should be employed for bacterial infections.

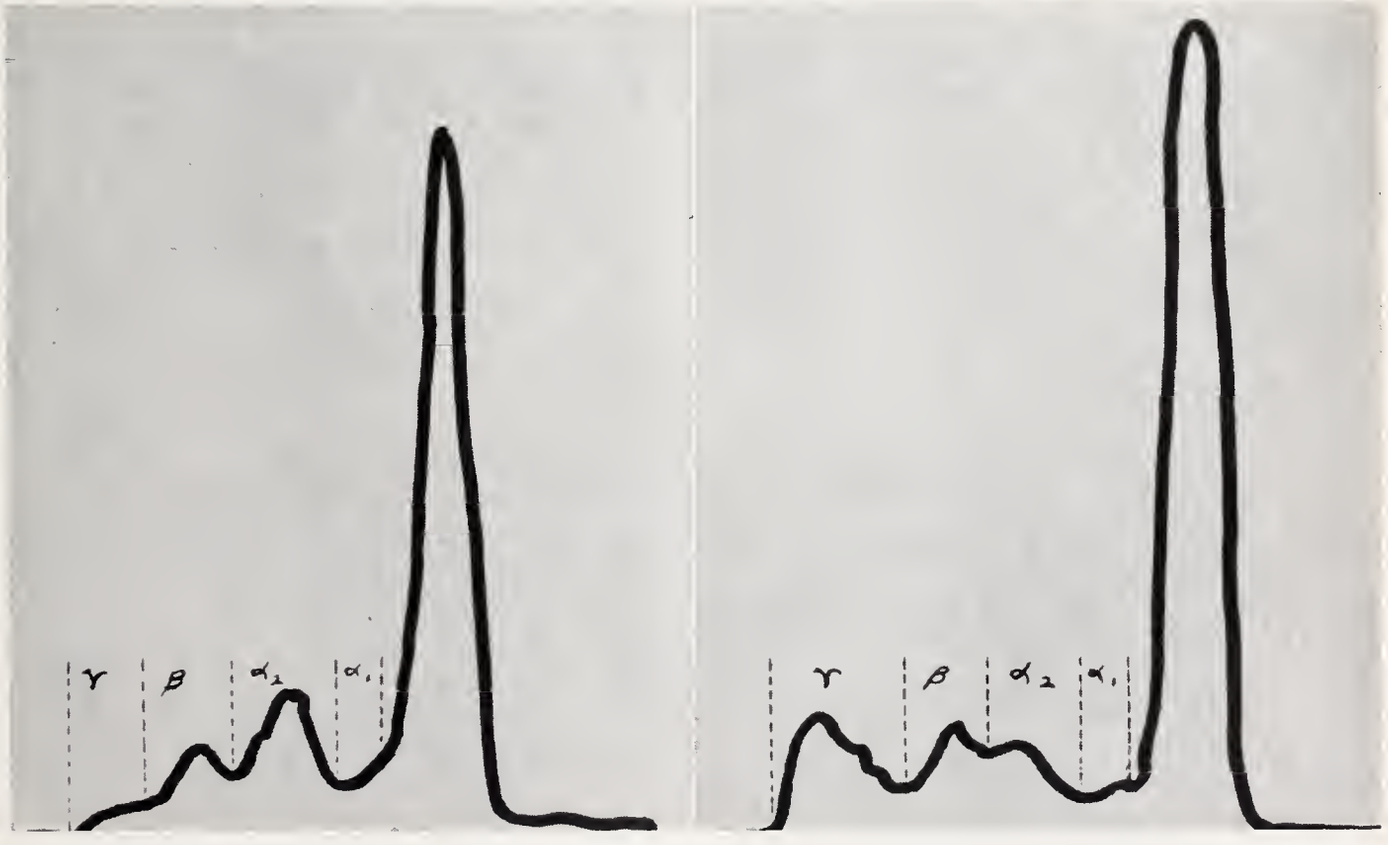

Fig. 1. Comparison of serum protein patterns as determined by paper electrophoresis. The pattern on the left is from a child with hypogammaglobulinemia; on the right is the serum protein pattern of a normal child.

Penicillin prophylaxis may be useful. *Live* vaccines are not advised for children with immunologic disorders.[25] Routine immunization with *killed* vaccines may be given since a response is seen in some patients. The use of disposable needles and syringes will eliminate the danger of serum hepatitis. Some of these patients develop sensitivities to gamma globulin, hence, it is well to keep the patient under observation at least 20 minutes following the administration of pooled gamma globulin.

Summary

The majority of children with a history of recurrent respiratory illnesses are experiencing infections with numerous, antigenically different infectious agents. With increasing age and progressive acquisition of protective antibodies, these illnesses will become less frequent. For those children whose illnesses are severe or complicated, a systematic approach to evaluation is described. Local and generalized disturbances producing recurrent respiratory tract infections are considered. The clinical approach to the diagnosis of defective synthesis of gamma globulin is discussed.

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72-Ton Room Shields Radiation Detection System

A steel room weighing 72 tons has been installed in a new wing of The University of Texas M. D. Anderson Hospital and Tumor Institute. Researchers in the Section of Nuclear Medicine and in the Environmental Health Unit will use the room to measure the natural radioactivity of human subjects.

The room has walls eight inches thick. The door alone weighs 6,500 lb., but is so perfectly balanced that only 15 lb. of pressure is required to open or close it.

A special crane weighing 162 tons lowered the room into the receiving area two stories below ground.

Accident Proneness in Aviation

Capt. Roger F. Reinhardt, MC, USN

Accident-prone pilots often can be spotted and grounded until they have made satisfactory adjustments which render them less dangerous to themselves and to those whose lives are in their keeping.

CASE REPORTS

MOST PEOPLE involved in accidents contribute in a major way to their own injury or death. The National Safety Council estimates that, of the 45,000 traffic accident deaths in this country in 1964, 85 percent were the result of driver error.[1] The boat operator is also his own worst enemy, for it is the operator who causes over 55 percent of all boating accidents. Varying with the type of flying, from 32 to 65 percent of aircraft accidents are caused by pilot error. In the most recent fiscal year for naval aviation, pilot error was the cause of over 45 percent of all aircraft accidents, or about four accidents each week. The average cost per accident was three-quarters of a million dollars; and, in every fourth accident, a naval aviator was killed. An aviation safety expert recently stated that unless we make a concerted attack on the human error accident, 60 percent of our future accidents will be caused by the human factor.

In this article, the historical development of the "accident proneness" concept will be reviewed and a hypothetical profile of the accident-prone aviator will be drawn. Nine cases in which we have accumulated some observations on behavior and emotional states during periods previous to accidents will be described and compared with the profile. The mistakes which people make and which result in aircraft accidents will be categorized and illustrative examples will be given for each category. Ways of coping with accident proneness in those who fly will be suggested.

Development of Accident Proneness Concept

The literature on psychological factors in accidents has developed in several phases. The first landmark was Freud's observation in 1904 that accidents are usually accompanied by unconscious intention.[2] He discovered that many accidents are not really *accidents* at all. The familiar accusation, "You did it accidentally on purpose," expresses this basic truth and paradox. In the 1940's, Dunbar,[3] Alexander,[4] and others, described the *accident-prone personality* as a person who is quick, decisive, active, impulsive, independent, and adventurous. He had a strict and religious upbringing, harbors a deeply ingrained rebellion against persons in authority, but has a strict conscience which makes him feel guilty for this rebellion.

In 1949, Tillman and Hobbs studied the personal lives of accident-repeater taxi drivers and found that a man drives as he lives.[5] In 1963, Hirschfeld and Behan studied hundreds of industrial accidents and found that mishap and injury provide a solution to the

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Opinions or conclusions contained in this article are those of the author and do not necessarily reflect the views or endorsement of the Navy Department.

patient's life problems.[6] In 1966, Tabachnik and other authors reported "psychological autopsies" in a series of accidental death cases.[7] Two-thirds of the subjects had recently moved into situations of greater responsibility (marriage, new job, change in environment). The most frequent cause of upset in their lives was criticism by others, to which they reacted with increased motor activity. They were more concerned with appearing attractive to others than with developing great emotional closeness.

Correlating this previous work with our clinical observations, we have drawn a hypothetical "profile" of the accident-prone aviator. (1) He had a strict moral upbringing resulting in a strong drive for independence matched by an equally strong conscience. Pressure from any source causes great anger and guilt. (2) He is action oriented and works off his frustration by action, not words. (3) His appearance in the eyes of others is very important; personal slight causes a sharp drop in self-esteem. (4) He has recently assumed major new responsibilities. (5) The "accident process," when set in motion, is manifested by errors in all areas of living—personal, social, and professional. (6) He senses that something unusual is going on and frequently sends out a disguised plea for help.

Case Reports

In the past year, nine cases have been studied because of apparent high accident potential.

Case 1.—A 28-year-old Navy lieutenant grew up in a home marked by strong religious beliefs and practices. He seldom dated until his junior year of college; he seldom drank until his designation as a naval aviator. During his first squadron tour, he began to "make up" for all he had missed, and after two years, married a girl he had impregnated. After four years of continued promiscuity by him, his wife left, taking their three children. Three weeks later, he had his first aircraft accident, crumpling the nose gear of his jet trainer. Six weeks later, he went to sleep at the wheel of his sports car, suffering multiple but nonfatal injuries. Moral wrongdoing and a strong sense of guilt immediately preceded the apparent punishment-by-accident.

Case 2.—A 33-year-old lieutenant commander gave an unusual accident history. His father had nine major accidents; his younger brother had six. His two sisters, though young, had eight major surgical procedures between them. The patient himself had 12 significant civilian accidents, six military aircraft accidents, and six aircraft incidents. He was in replacement pilot training to learn to fly the A-4 aircraft. His wife had returned to her parents because of impatience at his inability to quickly find a suitable house to rent. He was considered dangerous by his instructors because of a panic reaction to even a suggestion of aircraft malfunction. His hyperalertness led to several inflight incidents. During the interviews, he preferred pacing the room to sitting, even when showing no other signs of anxiety. This man was not only action oriented to a high degree, but he came from an accident-prone family. Dunbar's fracture cases gave a family accident-frequency three times higher than that of patients hospitalized with other illnesses. Whether this family tendency toward accidents is a matter of physiology and constitution or a matter of behavior patterns, or both, is a question that has received little attention.

Case 3.—A 24-year-old ensign flight student had his first five flights with a friendly, complimentary instructor, and did well. He was then assigned a Marine instructor who sharply criticized his flying. On his second dual flight in the primary precision stage, he slowed to 60 knots on final, sank rapidly, and even prompt corrective measures by the instructor could not prevent strike damage to the aircraft. There were no injuries. This student showed an extreme intolerance of personal criticism.

Case 4.—A 25-year-old lieutenant (jg) had worked himself up from a very low class family. His early life was marked by his father's alcoholism, his mother's loose morals, and frequent police raids of his house. Even so, he was an outstanding Naval Academy graduate and an outstanding flight student. While in replacement training as an F-8 pilot, he reported radio failure when he had the volume turned all the way down, broke to the right in a left-hand pattern, and ground-looped the aircraft by premature engagement of nosewheel steering. Over the next two weeks, several such incidents occurred; he even got between the number one aircraft and the target on a missile run. During a two-week period of interviews, he said he liked to fly because, "There people can watch you, see how good you are." He saw himself as on the threshold of success. He became aware of feelings that by failing he could punish his family, now so proud of him, for the many ways in which they had failed him.

Case 5.—A 22-year-old ensign was in replacement pilot training as an F-8 pilot. He was one of twin boys born to famous professional parents who were seldom at home. He grew up in various boarding schools and was materially well endowed, but affectively deprived. On completing flight training, he requested a West Coast assignment; he got the East Coast. He requested the A-6 aircraft; he got the F-8. Prior to his first F-8 familiarization flight, he did not feel adequately briefed, but was shamed when he mentioned it. The off-coast haze hid the horizon, and he did not feel his first flight should be made in such weather. Practicing landing pattern procedures, he pulled back on the stick too sharply, throwing the aircraft into a stall and spin, from which he successfully ejected.

Case 6.—A 32-year-old lieutenant commander was a quiet, shy, soft-spoken son of a minister. He was qualifying in an F-4 Phantom Two prior to his second operational squadron assignment. On starting his training, his progress was slow, and he appeared

before a pilot's disposition board. The board decided that the fault was not the student's and that, after flying props for four years, he should have been given a short jet refresher course. He had the refresher, but felt a deep stigma because of having had the disposition board. With every instructional hour, his flying got worse. He made 20 to 30 degree errors in heading and errors in altitude of as much as 5,000 feet; he took two or three minutes to make corrections. In tears, he told the examiner that ever since his disposition board, he had been depressed and embarrassed. He avoided old friends. This man's exquisite sensitivity to any personal blemish resulted in a neurotic depression and dangerous airmanship.

Case 7.—A 25-year-old Marine lieutenant, student naval aviator, was run over by a truck at age 15, had a knee operation following a football injury at age 16, injured the other knee playing lacrosse in college two years later, but still played all sports. He divorced his wife after one year, the marriage having been "hasty and impulsive." While still in primary flight training, he had an automobile accident. In advanced multi-engine training, he had a student pilot's disposition board because of excessively hard landings.

Case 8.—A 24-year-old lieutenant (jg) student naval aviator was the son of a prominent hotel owner and Navy Leaguer. The son, less successful in every way, concealed severe chronic airsickness. Three weeks before completion of jet training, he was vomiting into his mask while in inverted flight and while trying to follow complex instructions from the Air Traffic Control Center. He said that to be successful—a jet pilot—he would be willing to die.

Case 9.—A 24-year-old naval aviator was a "perfectionist" all his life, was in the upper 3 percent of his Naval Academy class, and later was an outstanding flight student. In July, he had a minor accident, not entirely his fault, for which he received a reprimand. In September, he accidentally dumped 1,500 pounds of fuel while on a long cross-country flight. In December, he was unable to drop his bombs on his first combat mission because of mechanical malfunction. Returning to the ship chagrined, he was unable to get aboard after six attempts, and had to eject when out of fuel. He was ordered back to his base for further training, and sprained his ankle in a skiing accident. In February, he landed 45 feet short of a runway during night practice; he got airborne again and was able to eject. Two weeks later, while on his way to have an eye examination, he collided with a Navy jeep. This pilot went to great lengths all of his life to avoid criticism, and his strong conscience demanded perfection. When he was not able for any reason to turn in his usual blemish-free performance, he went into a downward spiral of self-destruction.

The Last Step in the Accident Process

High *potential* for an accident is transformed into the *reality* of an accident through a mistake of some kind. Since feelings don't watch instruments or move controls, there has to be some act of commission or omission to complete the accident process. These last-step acts can be considered under such headings as: inattention to reality, poor judgment, "get-there-itis," misperception, and slips-of-the-hand. Knowledge of the most frequent last steps may offer some protection against them and will certainly increase our understanding.

Inattention to reality is perhaps best demonstrated in case 9, where the F-4 pilot, after two accidents and one incident, on probation and concentrating on perfect performance, landed 45 feet short of a beautifully lighted runway on a clear night. Poor judgment runs through the two nightmarish weeks of flying in case 4, especially when he wandered between the number one aircraft and the target on a missile run. The missile in use was a heat seeking device which would have flown right up the tail pipe of his jet.

A third common last step is referred to in the language of aircraft accident reports as "get-there-itis." By this, we mean impulsive decision making in the interest of a rapid termination of the trip. A young husband and father persuaded his reluctant wife to fly with him to his parents' home for the Christmas holidays. They were forced down short of their destination by bad weather and spent a miserable night at a poor motel. In the morning, the wife was still nagging that, "I told you we should drive." The pilot was not instrument trained, the ceiling was 1,000 feet, and the little valley airport was surrounded by mountains; it was Christmas Eve. The pilot took them off at ten o'clock the next morning. Apparently without benefit of close scrutiny of his chart, he tried to fly over a 3,800 foot mountain ridge at 3,500 feet. All major components of the aircraft were found heaped in an area approximately 15 feet in diameter.

Misperception is often used as a last step in the accident process. As an example, the number two man in a four-man F-8 Crusader flight noted in the aircraft's yellow maintenance sheet that the same discrepancies had been repeatedly reported by previous pilots, even though on each occasion the indicated repairs had ostensibly been made. Although the pilot was uneasy about the aircraft, the pilot's flight leader was in a hurry to get airborne. About halfway down the 8,000 foot runway, the pilot heard a transmission over the UHF in which he identified only the word "abort," and something about

hydraulic failure. Actually, the transmission was from another airplane taxiing out, but the pilot taking off immediately closed the throttle and tried to abort the take-off. The tailhook missed the arresting gear, and the aircraft rolled off the runway, shearing off the nose wheel as the nose buried in the soft ground. This came about because the pilot misperceived or mis*re*ceived the transmission, coming through loudly and clearly only with one word—abort—to which he was emotionally sensitized.

Slips of the hand occur frequently, and often have the same significance as slips of the tongue. Because flying is so often unforgiving, airborne slips of the hand are frequently disastrous. The pilot in case 9 was flying the F-4 with his commanding officer in the rear seat as passenger. The pilot used after-burner on take-off, as he had been taught, but was criticized by the commander for the technique because of the excess fuel consumption. The meaning in the pilot's slip of the hand a few minutes later, which resulted in the dumping of 1,500 pounds of fuel rather than transfer to another tank, is quite transparent.

Comments

Whether as pilots, flight surgeons, or aviation medical examiners, we all have a responsibility to understand and prevent aircraft accidents. It would seem that some of our knowledge about accidents is ready for practical application. The man who requires nothing short of perfection of himself should be looked on as having a head start in the accumulation of accident points (using the model that the accumulation of so many points, regardless of source, produces an accident). The man who acts out his problems picks up a few more points. He can be encouraged to talk them out, instead. Those who are exquisitely sensitive to criticism accumulate some points; they can be grounded, or ground themselves, when they feel severely slighted. The person who has moved sharply upward in life has acquired accident points. He may feel figuratively that he is "flying too high," or be angry at the demands or responsibilities placed on him. If someone is experiencing an abnormal burst of errors, or accident-activity, even in his social or nonflying activities, we know that his point accumulation is past the accident threshold, and that he should not fly. Lastly, we should be perceptive of uneasiness, even though the source may not be known. The uneasiness often expressed through somatic complaints may be an unrecognized signal of accident activity, an alerting survival mechanism.

A business executive or a physician can, in an office, make a slip of the hand and knock over an ash tray, break a pencil, or spill a cup of coffee. If he's flying an aircraft and makes a slip of the same psychomotor magnitude, he can cause death and destruction. Therefore, once a person is identified as accident prone, he should be removed from flying and other high risk situations until he is obviously less so, or until the causative edge has been taken off by an effective change in his life situation. Nowhere is it more true that the life you save may be your own.

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Management of Obesity

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Most obese people cope with environmental stress by overeating. Far more than further emphasis on food and diet, they need help in learning other ways to fulfill their psychological needs. Although diet, medication, and other measures are useful, lasting weight reduction is achieved by a psychotherapeutic approach.

CLINICAL MEDICINE

OBESITY is unquestionably one of the most troublesome problems of our times. According to Shipman and Plesset,[1] some 15 million people in the United States exceed their ideal weight by 20 percent or more. The role of obesity in cardiovascular disease, digestive difficulties, joint pains, and other disorders, as well as the cosmetic effects of the overweight condition, have been widely discussed. Omitting the pros and cons of weight reduction in these more serious situations, we shall consider the management of other patients who, for any reason, wish to lose weight as long as it is compatible with their medical situation and in accordance with general health practices. Any physician who has been in practice for long has been impressed with the person who "simply cannot stay on a diet," who says that everything he eats turns to fat, and who insists that the dietary intake has not been sufficient to produce the excess adipose tissue which makes him uncomfortable in clothes and a laughing stock on beaches and streets.

Obviously, under these circumstances, such people use food for psychologic reasons rather than for the sheer caloric needs. (A few organic lesions such as those involving the hypothalamus, may lead to obesity, but they are extremely rare.)

Motivation Is Basic to Therapy

Standard textbooks in medicine[2] point out that the basic problem in the treatment of obesity is motivation of the patient. As Harrison says, "In no disorder is the prescription so simple and the possibility of cure so high as in the treatment of obesity. Why, then, is there so much discussion concerning the 'best diets,' such concerted effort to seek the 'magic pill,' such quackery and chicanery surrounding attempts to restore weight to normal?"

Motivation usually arises from one or more of the following factors: (1) pride in personal appearance; (2) symptoms of disordered physical function; (3) fear of future disease; and (4) an attempt to avoid the antagonistic reaction or "pressure" manifested by friends, relatives, or business associates.[2] Actually, like the two-edged sword, these are sometimes precisely the elements which work against, rather than toward, the diet reduction which is essential if there is to be weight reduction.

When a psychotherapeutic approach is used, many patients will admit that the fat itself is a weapon which is used against the family in an attempt either to make them accept the obese patient in spite of his repulsive physical characteristic, or to show them that they cannot control the patient's food intake. As some of our patients say, "This is one thing I can do and no one can stop me."

Familiar traditions surround the fat person, and food has tremendous importance in many of our personal activities and our interpersonal reactions. Many patients come from families in which preoccupation with diets, body contours, and the like has been important in establishing eating habits which may persist through life.

In some instances, eating is associated not only with a sense of inner security, but also with the sense of acquiring inner goodness. "Be a good boy and eat your spinach" is a fairly common admonition to the growing child, and the further admonition to clean up one's plate also equates dietary intake with rewards and approvals from people around us. One seldom hears a parent complimented for having a "nice, skinny baby."

The literary recognition of the "fat, dumb, and happy" equation is certainly well known. Most of us had to memorize such things as:

"Let me have men about me that are fat,
Sleek-headed men, and such as sleep o' nights.
Yond Cassius has a lean and hungry look;
He thinks too much: such men are dangerous."[3]

Washington Irving said the same thing: "Who ever hears of fat men heading a riot, or herding together in turbulent mobs?—No—no, 'tis your lean, hungry men who are continually worrying society, and setting the whole community by the ears."[4]

The pleasant aspects of obesity appear also in Dryden's "The Maiden Queen": "I am resolved to grow fat, and look young till forty."[5]

This literary formulation, however, ignored entirely the fact that the obese person is not only unhealthy, but often extremely unhappy, hostile, and very much at war, both within his own personality and with his environment. Aldrich quotes from an obese young man, "I never felt that my family really wanted me around. All mother seemed interested in was getting enough food into me."[6]

Noyes and Kolb point out that "The development of obesity often occurs in a family setting in which the parents compensate for their own frustrations and disappointments through their attachment to the child." Under these circumstances, they say it is not surprising that "In many instances the obese child is not a wanted child."[7] Furthermore, no physician who handles such patients can fail to be aware that the marital situations are often poor, that alternation with alcoholism and excessive drug intake is not at all uncommon, and in some instances the attempt to lose weight may be associated with some exacerbation of these deeper difficulties.

There is not universal agreement on this last point, however. Shipman and Plesset[1] cited Stunkard, who felt that "For a large number of overweight persons, the mechanical prescription of reducing diets has had unfortunate consequences." He found that 51 percent of the obese women in a nutrition clinic reported untoward responses to previous attempts at weight reduction. Also cited[1] was Hamburger, who also noted that one-third of his 18 patients developed emotional upsets when they lost weight. Brosin[8] felt that some individuals are adjusted to a set of delicate balances, and that a change in this may lead to anxiety, depressive and other reactive states, sometimes with suicidal attempts.

Individual Treatment

Management of these patients, therefore, must be carried out in the setting which allows fairly extensive investigation of the psychologic factors involved in the overeating; and the feeling that the patient has about himself or herself. The physician must attempt to modify the patients' approaches to life situations in such a way that overeating will not be their only method of coping with environmental stress.

In exploring these factors, the physician must start with a realistic appraisal of the amount of weight reduction which may be indicated. I have seen individuals who actually were almost emaciated but insisted that they were too fat and needed to lose weight. My last such patient was a 15-year-old girl, who resembled a concentration camp victim, and who became so preoccupied with the horrors of growing "fat" that she refused to drink water. Other patients may have more realistic goals for losing weight, but the amount to be lost and the program to be adopted must, of course, be up to the physician. Various authors have stated that the general practitioners, or the patient's personal physician, is the person best suited to handle the problems of obesity. He certainly is the person to whom the patient most often turns for help in this troublesome situation. Bruch[9] comments on the fact that many such persons need the emotional support of the family physician, and indicates that the physician's previous role in dealing with other problems may place him in a more favorable position to handle the weight reduction regimen. She points out, however, that if the physician gives too much praise for losing weight, the patient may find it difficult to return for additional treatment if he becomes overweight again.

Furthermore, there are some dangers in attempting to introduce psychological elements into the discussion with the patient because he may feel that he has, to some extent, been tricked into dealing with these psychological problems. Referral to a psychiatrist often is met with a great deal of hostility and resistence for exactly the same reason. Hofling[10] also believes that "such patients are more effectively helped and handled by internists and general practitioners than by psychiatric therapy in its present state of knowledge and know-how." He, too,

points out that these patients usually do not directly seek help for their obesity, but come to the internist or general practitioner for help in other matters and discuss their obesity in general rather than specific terms.

Kroger and Freed[11] indicate also that the physician must not be facetious, sarcastic, or brusque, and must avoid criticism as he avoids excessive praise. He should not be misled by temporary weight loss, and should, in every instance, try to help the patient find other ways of coping with the environmental situation. Otherwise, an early return to overeating with subsequent disappointment and even depression may ensue.

The actual management of obesity, when once undertaken, is a matter of choice for the physician. In general, it should consist of a psychotherapeutic approach, with whatever help can be obtained from appetite-reducing medication. In my experience, diaries containing detailed descriptions of what the patient eats have had little value, although other physicians have found them useful. The problem, as I see it, is that these individuals spend a great deal of time talking about food; in fact one of the hallmarks of the obese person is the facility with which he can remember, in great detail, meals he has eaten several days previously. The diary itself is not likely to be accurate, and if the physician concentrates on the diary rather than on the psychodynamics of the overeating, he may find himself involved in a maze of contradictions and actual falsehoods which will successfully divert his attention from the more pertinent problem. In my opinion, it is more useful to ignore the actual food intake entirely, and to concentrate instead on the occasions on which the individual can identify turning to food for want of gratification in other areas.

Periodic weighing is also recommended by some physicians. I have not found this useful, and prefer to try to divert the patient's attention from the weight itself to the effect which it may have on his daily living.

Group Therapy

Group therapy has been recommended by several investigators. Holt and Winnick[12] worked intensively with a group of six women, each of whom had made at least two unsuccessful attempts to reduce. In group therapy, it was found, "The patients' relationship with their parents was similar to that found in many drug addicts, whose orality has been extensively documented. *Every one* of these patients came from homes in which the mother was overprotectingly rejecting and the father was relatively ineffectual, weak, and submissive." The sessions were conducted under psychoanalytic group therapeutic principles and the patients were told to discuss anything they wished. The project was designed to test four hypotheses: (1) that group analytic psychotherapy could improve the general adaptation and emotional well-being of obese patients; (2) that it was possible to predict weight shift by the content of the group therapy sessions; (3) that patients would lose weight as a result of group analytic treatment; and (4) that the patients would use ancillary procedures such as diet, massages, and exercise, and that the use of these procedures would be related to the content of the therapy sessions.

There was a total of 22 sessions for the six women. There was no "dieting depression," mentioned elsewhere, and there were only two somatic complications. As to the hypotheses, there was significant improvement in adaptation and self-awareness. Some prediction could be made about the gains or losses in weight, depending upon the content of the recorded sessions, but the relationship was slight. As to the weight losses, there had been a fairly consistent pattern of weight decrease, but the follow-up several months later showed that the net average change was a minus 4.3 lb. which, considering the average initial overweight of 33 lb., did not seem significant. The authors thought that the patients "established a reinforcing unconscious pattern of not losing weight and derived a kind of masochistic delight in not achieving this major goal." There was some validation of this attitude in the fact that in the group sessions, eating and food represented only 9 percent of the total responses, with dreams and other unconscious material, parents and childhood memories, interpersonal relationships, and fantasies all coming ahead of this.

The last hypothesis, that special diets, massage, and relaxation facilities which were immediately available would be used as soon as the patients accepted their roles as patients, was not confirmed.

We attempted a group therapy project with high school girls in the Salt Lake area. Three schools, each in a different socioeconomic area, were chosen. We used public health nurses as leaders for these groups, and found the most successful approach (of the three we used) was on the sample basis

of diet and exercise with constant attention to appearance and actual caloric intake. The group in the upper middle class area consisted of 35 girls, but it was impossible to keep them involved in a group psychotherapy situation. Consequently, the leaders shifted to an individual approach; this seemed to work better, although the project was not continued long enough to make any real statistical evaluation of the total project.

The girls who lived in the lower socioeconomic area were approached purely on the basis of diet and exercise. The group consisted of 75 girls, not all of whom participated in the project. Many of the girls lost weight, but frequently regained it as soon as they stopped the group sessions.

At the third high school, socioeconomically between the two others, the attempt was made to deal entirely with the emotional difficulties of the girls in the group situation, but the girls were not adequately prepared for it and the nurse herself felt uncomfortable. It was thought that in spite of the failure of the previously mentioned adult group to deal with ancillary therapy, some consideration of the "charm school" approach, having to do with posture and grooming, was useful in helping these teen-agers cope with their problems of relationships with other individuals.

Use of Drugs

The use of medication in the control of obesity deserves a certain amount of consideration. Many substances have been proposed, most of them compounds containing amphetamine sulfates and dextro-amphetamine sulfates, with dosages of 5 mg., ½ to 1 hour before meals; or longer-acting preparations given in doses of 10-15 mg. each morning. Bulky preparations like methylcellulose, which reduces hunger, or combinations of amphetamines and methylcellulose may be useful in some instances. Some patients can tolerate large doses of these appetite reducers, and dramatic amounts have been prescribed by some physicians. In my opinion, they have little value over the smaller doses, since the important thing is the attempt to help the patient gain new mechanisms to cope with the environment.

Hospitalization for rigid dietary control has also been recommended, particularly when it is used with a "crash diet" in which there is complete control, to starvation level, of the caloric intake. It has been suggested that a dramatic loss of weight, achieved while the patient is in the hospital and is allowed only vitamins and noncaloric liquids, can be most encouraging to the individual whose previous attempts at dietary control have consisted of piecemeal efforts yielding little real change. The method is worth a trial, although if it fails, the patient's disappointment may be difficult to handle.

Conclusion

Obesity is a troublesome problem which has far-reaching psychological and social roots, and is influenced by powerful psychological factors. Management of these obese patients must depend, to a large extent, upon a supportive and trusted physician-patient relationship, and the internist or family physician may find his relationship with these patients to be a favorable setting in which to provide them with adequate help. Dietary management, in and of itself, is probably largely ineffective, and the psychotherapeutic effort should be directed at helping the patient achieve better ways of coping with the environment. Usually, elements which come into the picture are fears of being unattractive, hostility toward significant individuals, a sense of unworthiness, and inadequacy in interpersonal relationships. Appetite reducing drugs such as methylcellulose may be useful. Crash weight reduction by strict dietary control in a hospital setting may be useful as may ancillary factors such as exercise and massage, but even these are likely to be ineffectual unless the patient's motivation is sustained.

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X-Rays of the Low Back In Preemployment Physical Examinations

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After one company included roentgenograms of the lumbosacral spine as part of the general physical examination for certain prospective employees, compensable claims for low back injuries dropped from 52 in 1953 to 11 in 1965.

THE EMPLOYER is legally permitted to protect himself from liability due to preexisting disabilities in a prospective employee by requiring a preemployment physical examination. Therefore, in 1953, we at General Dynamics decided that the examination would include x-rays of the back on all male applicants, according to the following criteria: (1) above 35 years of age, or younger when there was a history of previous back complaints other than a simple or mild backache lasting several days; and (2) below age 35 if there were abnormal physical findings such as scoliosis, tipped pelvis, or limitation of motion.

We chose the 35 years and over age group in order to coincide with the age group taking preemployment electrocardiograms. The reason for instituting such examinations on preemployment physicals was to eliminate as much as possible excessive compensation claims on congenital and structural spine defects which existed before employment. These x-rays were made of the lumbosacral area including anterior-posterior views as well as lateral and spot films of the L-5 and S-1 area.

In 1953, we had 52 compensable back injury cases in litigation or before the Texas Industrial Accident Board. Also, low back injury had resulted in more lost man days and more money than any other compensable condition.

These physical examinations were done on job applicants for work in a large aircraft manufacturing plant, employing factory and white collar workers, whose average age was 47 years.

Findings

During 1963, 1964, and 1965, there were 10,041 preemployment physicals performed. Back x-rays were taken in 2,098, which is 20.9 percent of the total physical examinations performed. Of the x-rays, 24 percent showed abnormal findings, and 353 applicants were rejected, representing 16.1 percent of the applicants who were x-rayed. This compares favorably with the findings of Becker[1] and Rombold[2] whose studies showed rejection of 24.5 percent and 20.3 percent, respectively, of applicants who had x-rays.

Table 1 shows the findings of the examinations in our series. Of applicants under 35 years of age with abnormal findings, 56 percent were acceptable, as compared with 57 percent in the over 35 age group.

Rowe[3] found the most frequent abnormality to be degenerative disc disease. Reiner, in a study of 1,030 candidates, found 59.3 percent to have pathological changes, and 35.8 percent of these applicants were acceptable.[4] Shevick[5] states that the low back syndrome is primarily due to orthopedic difficulties, and the most important findings were those having to do with degenerative disc disease, spondylolisthesis,

TABLE 1.

	Under age 35 (138 x-rays taken; 48 cases abnormal, 34.78%)	Over age 35 (1,960 x-rays taken; 456 cases abnormal, 23.26%)	All Ages (2,098 x-rays taken; 504 cases abnormal, 24.06%)
X-Ray Findings	%	%	%
Degenerative disc disease	10.87	10.20	10.01
Spondylolisthesis	6.52	5.09	5.20
Hypertrophic arthritis	2.90	4.02	3.97
Sacralized L-5, S-1	5.80	1.48	1.96
Lumbar scoliosis	2.90	0.65	0.77
Spina bifida	2.90	0.55	0.69
Spinal fusion	0.00	0.34	0.33
Compressed fracture	1.44	0.19	0.30
Myelogram dye	0.00	0.30	0.29
Lordosis	0.72	0.20	0.25
Acute angulation of sacrum	0.72	0.14	0.19
Spondylosis	0.00	0.10	0.10

and prior back surgery. He states that some industries reject 20 to 45 percent of applicants on the basis of preemployment x-rays.

Significance

One cannot find unanimous opinion on the significance of low back abnormality as seen in x-rays with regard to potential compensable injury, but one can say that certain conditions such as spondylolisthesis and degenerative disc disease between L-5 and S-1 are causes of an unstable low back, and that x-rays would be helpful in screening out persons likely to sustain an injury or aggravate a preexisting condition. During the period of 1953 to 1965, our compensable compensation claims for low back disorders decreased from 52 per year to 11 per year in 1965. Five applicants over 35 years old were accepted during this time; three had normal low back x-rays, and two had minimal hypertrophic arthritis of the lumbar vertebra. Four of these men filed claims with the Texas Industrial Accident Board, and one filed a lawsuit. These employees sustained low back strain when they slipped or bent over to pick something up. They therefore alleged compensable injuries.

Accepted applicants who had abnormal x-ray findings were placed in jobs, both factory and office, commensurate with their physical findings and limitations if any. We know that we cannot ascertain by these x-ray findings alone which employees will or will not have low back problems. We can only say with a degree of certainty that since we began taking preemployment back x-rays, our compensable back injury claims have decreased appreciably. We also realize that if we reject everyone who has abnormal low back findings we, as an industry, would soon find ourselves in an employment dilemma for qualified technical office and factory workers, and our line echelon would suffer. Each case was treated on an individual basis, and no so-called blanket standard was used for acceptance or rejection.

Considering the similarity of percentages in both the older and younger age groups, one might ask why we do not do preemployment back x-rays on everyone. Our reason is that the extra expense is offset by the much lower frequency of disabling back injuries in the younger age group.

1. Preemployment low back x-rays should be taken on certain applicants. As a result, compensation back injury claims will be considerably reduced.
2. The average rate of back abnormalities was 24 percent.
3. The percent of applicants rejected was 16.1 percent.
4. Serious consideration should be given to taking x-rays of the back regardless of the age of the applicant.

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Biology of Thyroid Disease

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The present survey may not cover all aspects of the "life" of the thyroid. But it is hoped that, as with every patient's history, it will help pave the way to an adequate physical examination, sensible laboratory investigation, and the correct choice of therapy.

REVIEW ARTICLES

BIOLOGY IS defined in the dictionary as "the science of life." This, in its broadest sense, "includes morphology, physiology, genetics, etc." Since life began in the sea, I shall start this survey of the life of the thyroid with its comparative anatomy and physiology. These both have been reviewed by Gorbman.[1]

All forms, even plants, namely certain algae, are able to trap iodide, a characteristic property of thyroid tissue. In the animal kingdom, invertebrates have no thyroid, but they too collect iodine. Further, they can convert trapped iodine into thyroxine within protein. This thyroxine appears mainly in the superficial cuticular structures such as shells and pharyngeal teeth.

The first suggestion of a thyroid gland capable of producing thyroxine is seen in the endostyle of the protochordates. This suggests that the first thyroids may have been exocrine rather than endocrine. The most primitive vertebrates alive today, the lampreys, show this phenomenon during the five years of larval life. The thyroglossal duct empties its secretion into the intestine where thyroxine is released by digestive enzymes. With metamorphosis, the thyroid changes from an exocrine to an endocrine structure, but before losing its duct, it acquires a protease within the thyroid lumen.

The role of thyroxine in the metamorphosis of the frog has been well studied. Interestingly, it has been claimed that various analogues of thyroxine may influence some of the steps in metamorphosis unequally compared to the normal sequence induced by thyroxine.[2] Equally interesting is the finding that unlike the tadpole, which completely deiodinates thyroxine during metamorphosis, a form of Necturus that does not metamorphose when thyroxine is introduced, fails to deiodinate the hormone.[3] This has led to a theory that the hormone is metabolized in the course of exerting its effect. The view, however, has been disputed,[4] especially since central nervous system (CNS) and molting effects of thyroxine are found in the adult frog which has no deiodinase.

In fishes, thyroxine is made and secreted by thyroid tissue although not all fish have organized thyroids. According to Gorbman, the platyfish thyroid migrates anteriorly to the choroid gland and posteriorly to the kidney. This migration is reduced by high iodide concentrations in the water. In contrast, iodine deficiency in the hagfish feeding on kale in an iodine-deficient pond results in a goiter without colloid in the follicles. The function of thyroxine itself in the fish remains obscure.

Following the discovery by Baumann in 1896 of the concentration of iodine by the mammalian thyroid; and the chemical characterization of the structure of thyroxine by Harington and Barger,[5] the nature of the thyroid hormone seemed established. However, Gross and Pitt-Rivers[6] in 1954 established the existence of a second thyroid hormone, triiodothyronine. This material is se-

creted by the gland and it is present in plasma in low concentrations. Incidentally, it was the introduction of radioiodine as an investigative tool that made this discovery possible.

Human Thyroid

Anatomically, the normal human thyroid averages between 20 and 25 gm. in weight. The gland is considerably smaller in tropical climates.[7] Size may increase under physiological stress, as with pregnancy, and recede when the stress is terminated.[8] The physiological role of the gland seems to be to manufacture its hormones, thyroxine (T4) and triiodothyronine (T3), and to store or release these into the circulation as required.

To do this, the gland must first concentrate iodide ion against the gradient established by the concentration of iodide in the blood. It must then alter this nonreactive ion to a form able to react with tyrosine to form diiodotyrosine (DIT) or monoiodotyrosine (MIT). Coupling of DIT with DIT or with MIT then occurs to form T4 or T3. Excesses of DIT can be dehalogenated within the gland. Most of these intricate chemical steps presumably are mediated enzymatically and thus lend themselves to genetic derangements. Thus, congenital goiters have been shown to result from a defect at virtually every step in the synthetic cycle,[9] as well as from a defect in hormone release when secretion of an iodinated protein instead of the free hormonal iodoamino acids may occur.[10] This protein material, an iodoalbumin, contains a considerable percentage of the circulating thyroxine in such patients,[11] and this thyroxine is unavailable to the tissues. Certain of these synthetic defects have been found in more than one member of a family, again suggesting a genetic origin.

Centers which control thyroid functioning are found in both the anterior pituitary and hypothalamus. Smith clearly established the existence of a pituitary-thyroid relation when he hypophysectomized rats without damaging the overlying hypothalamus.[12] As with all anterior pituitary-end organ relations, a "servo" or negative feedback system is involved. The pituitary secretes thyrotropin which stimulates release of T4 and T3 as well as their synthesis. The excess of hormone in the circulation in turn inhibits pituitary release of thyrotropin. Thyroid function is arrested, the blood level of thyroid hormone falls, and this allows the pituitary to secrete once more.

It has been claimed that thyrotropin is secreted only by a specific thyrotrope cell in the anterior pituitary. Evidence for this view comes from histological studies with special staining techniques.[13] The concept is also supported by work with anterior pituitary thyrotropic tumors in the mouse. Such tumors have been uniquely shown to monodeiodinate T4 to T3, unlike the total deiodination accomplished by mouse pituitary adrenotropic tumors.[14] In two normal tissues, the heart[15] and the anterior pituitary,[16] monodeiodination also has been demonstrated.

The existence in the central nervous system of a suprapituitary regulatory center has been demonstrated by experiments in which the anteromedial hypothalamus has been destroyed.[17] Thyroid activity becomes minimal as shown by negligible responses to goitrogen feeding. Implantation of thyroid hormone into these areas of the hypothalamus has been claimed to suppress the pituitary and hence the thyroid.[17] More recently, Reichlin and associates[18] transplanted rat pituitaries to the anterior chamber of the eye. The ability of the normal pituitary to monodeiodinate T4 to T3 virtually disappeared when the gland was separated from its hypothalamic connection.

In the opposite direction, Harris[19] has increased thyroid activity by electrically stimulating the anteromedial hypothalamus in the adrenalectomized rabbit. However, confirmatory evidence in other species is wanting. More recently, isolation has been accomplished of a "thyrotropin releasing factor" (TRF) from the hypothalamus which causes the pituitary to release thyrotropin.[20] Efforts to find regulatory areas higher in the central nervous system than the hypothalamus have not been too rewarding. It is of interest in this regard that systemically injected radioactive thyroxine concentrates primarily in the hypothalamus and posterior lobe of the pituitary,[21] although this varies among species.

A pituitary-like substance, long acting thyroid stimulator (LATS), has been found in the serum of patients with Graves' disease.[22] This agent behaves chemically like a 7S gamma globulin (IgG),[23] one of the immunoglobulins. Like antibody globulin, Porter fractionation produces both A and B chains with thyrotropin-like, i.e. short-act-

ing, activity found with the A chain after splitting,[24] unlike the long-acting response from the parent LATS. More recently, cultures of lymphocytes from patients with Graves' disease have been reported to release LATS into the medium. This LATS is said to react with thyroidal microsomes.[25] The claim has thus been made that LATS may be an autoimmune antibody although there is disagreement about whether LATS causes Graves' disease as a whole, or only its eye complications.

The concept that hyperthyroidism may be an autoimmune disease is startling and, on clinical grounds, difficult to adjust to. The precipitating role of emotional stress in the onset of the disease is generally acknowledged. Furthermore: (1) in our hands, serum assays for LATS are usually negative in uncomplicated hyperthyroidism until eye changes or localized myxedema supervene;[26] (2) LATS assays may be positive in the euthyroid phase of Graves' disease, when only the eye signs have appeared and not the hyperthyroidism, although it has been suggested that the gland is involved but is unable to hyperfunction;[27] (3) hyperthyroidism is cyclic and prone to spontaneous remission; and (4) it is not entirely clear that all 7S gamma globulins must be antibodies. On the other hand, prednisone, an immunosuppressive agent, can induce remission of Graves' disease and return the enlarged thyroid to normal.[28]

The autoimmune origin of chronic lymphocytic thyroiditis is possibly more secure.[29] The presence in the serum of precipitating and complement fixing antibodies is well known; the former against thyroglobulin and the latter against thyroid microsomes. A cytotoxic antibody that inhibits early cell growth in tissue culture has also been demonstrated. Less clear is how the concealed antigens within the thyroid cell become exposed to act as an antigen or react with the antibodies. Nonetheless, experimentally, the introduction into normal hosts of spleen cells and lymphocytes from thyroid-injected guinea pigs with thyroiditis will produce chronic thyroiditis and cutaneous hypersensitivity in the hosts,[30] a delayed hypersensitivity type of response.

Goiter and Thyroid Cancer

The essentiality of iodine in the synthesis of thyroid hormones places the host in close relation to the external environment. Follis has recently reviewed the ecology of the thyroid.[31] He has listed the sources of iodine and has observed that persons excreting less than 50 μg. iodine in the urine daily generally have iodine want or endemic goiter.

Certain discrepancies between iodine intake and development of goiter, however, have been demonstrated. For example, as little as 18 μg. daily intake may not be associated with goiter;[32] or there may be goiter with as much as 44 μg. iodine per day in the urine, with the nongoitrous normals in the same area excreting only 5 μg. more, an insignificant difference.[33] Apart from inadequate iodine intake, other causal factors have been hinted at: failure to absorb iodine from the gut due to infection;[34] renal losses;[35] excessive fecal losses from ingestion of certain bulky foods, as shown in the rat;[36] and finally, and perhaps ultimately the most significant, consanguinity.

The polemic as to whether prolonged thyroid hyperplasia from iodine want or other causes leads to neoplasia has not been entirely settled. Experimentally, prolonged use of antithyroid drugs leads to cancer in the rat.[37] Removal of all but the isthmus of the mouse thyroid, however, leads only to changes like those of nontoxic nodular goiter.[38] Here, as in man, with the appearance of nodular changes, the administration of thyroid hormone no longer induces the regression it causes in the diffuse stage. Sloan[39] has pointed out, clinically, that the peak incidence for thyroid carcinoma is before age 40, whereas for nontoxic nodular goiter, the incidence begins to rise after this age.

In man, the possible importance of genetic factors in the induction of nontoxic nodular goiter and thyroid cancer has been suggested by the strong predilection of certain families to develop thyroid disease. Changes of recent interest, functionally, in the transition to cancer are, (1) the loss of the microsomal antigen present in normal thyroid tissue so that cancer cells can grow in Hashimoto thyroiditis serum, whereas normal thyroid cells cannot;[40] (2) the continued production of thyroglobulin by cancer cells, but of very low iodine content;[41] and (3) the release into the circulation of an iodinated albumin by many papillary thyroid cancers.[42]

The mere presence of nodular goiter would not be a source of concern to the clinician in most instances were it not for fear of cancer. As a result, the internist and surgeon often find themselves diametrically opposed about therapy. The relatively high

incidence of cancer in, or associated with, the solitary thyroid nodule has been well publicized. Recently, the therapeutic significance of the association of thyroid cancer and nontoxic multinodular goiter has been carefully considered,[43] and the conclusion drawn that surgery or medical therapy have equal arguments on their sides.

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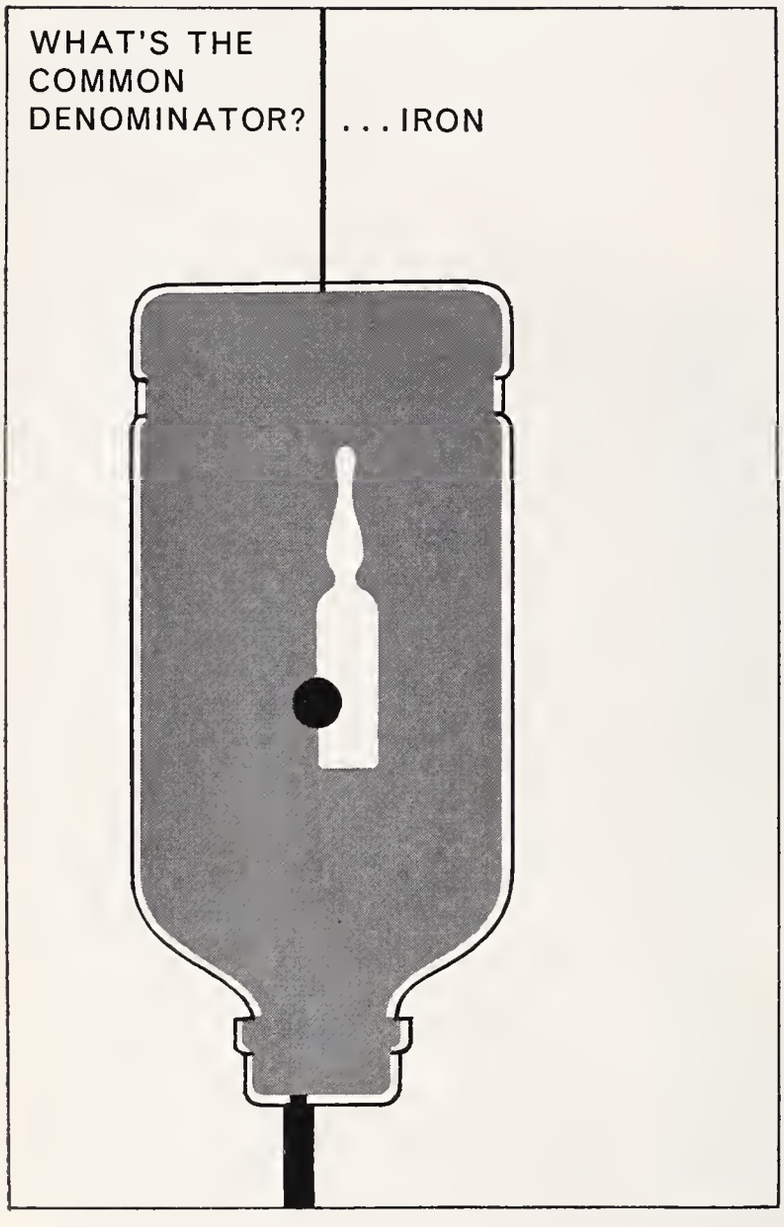

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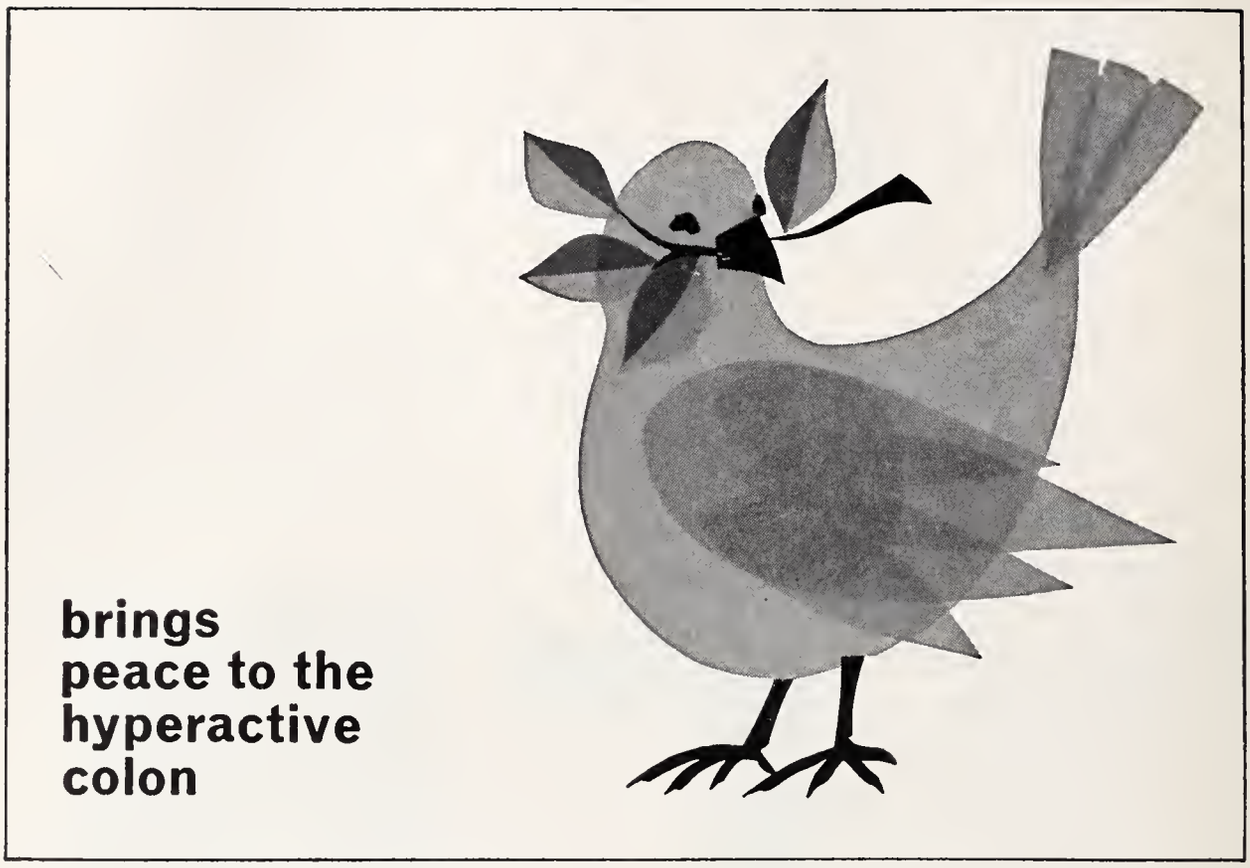

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Clinicopathologic Conference

George Peacock, M.D.; Ed Winga, M.D.; John T. Davis, M.D.; Norman M. Kaplan, M.D.; Leonard Madison, M.D.; Daniel W. Foster, M.D.; Donald Seldin, M.D.; and James B. Ketchersid, M.D.

The patient, age 80, had been hospitalized 11 times in four years. There was evidence of cholelithiasis, chronic bronchopulmonary disease, coronary arteriosclerosis, panhypopituitarism, and hypothyroidism. The sella turcica was enlarged. Finally, hemiparesis developed. The patient became comatose; his temperature rose, and he died.

DR. ED WINGA: This 80-year-old white man was admitted to Parkland Memorial Hospital for the 12th and last time on Jan. 1, 1966, with the chief complaint of a "painful right hip."

The patient's first admission, for fracture of the left mandible, was long ago (record lost). His second admission was to the surgical service in August, 1962, because of abdominal pain. He had a long history of cigarette smoking and chronic productive cough, but had worked as a night watchman until about two months prior to admission when he retired because of weakness and shortness of breath. At that time he was seen in another hospital and given oxygen and vitamin B_{12} for a "heart murmur and anemia." For two days before admission he had pains in the right upper and lower quadrants, with nausea and vomiting. He had normal vital signs, smooth and waxy skin, mild arteriovenous nicking in the fundi, distended neck veins, a nonpalpable thyroid, no cardiomegaly or heart murmurs, dullness of the right chest base posteriorly with bilateral basilar rales, hepatomegaly and tenderness in the right upper quadrant of the abdomen, and 4+ guaiac stool. Initial laboratory data revealed Hb., 11 gm. per 100 ml., decreasing to 8.9 gm., although subsequent stools were guaiac negative. A gastrointestinal workup revealed sigmoid diverticulosis and nonvisualizing gallbladder. A chest roentgenogram showed borderline cardiomegaly, apical pleural thickening on the left side, increased pulmonary vascular markings, a fine reticular pattern, and bilateral pleural effusions. A first degree heart block, low voltage, and nonspecific S-T changes were seen on electrocardiogram. The patient was given blood and digitalis, with resolution of the pleural effusions, before being transferred to the medical service because of suspected decreased pituitary function. The patient had a lack of axillary and pubic hair, slow reflexes, decreased serum sodium concentration, and an enlarged sella turcica. The protein-bound iodine (PBI) and ^{131}I uptake determinations were unreliable because of previous use of Telepaque, but the ^{131}I uptake increased from 7.5 percent to 12.7 percent after three days of thyroid stimulating

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This is the sixth in a series of clinicopathologic conferences from The University of Texas Southwestern Medical School, Dallas. Dr. Bruce D. Fallis, professor of pathology, is guest editor for the series. Participants this month are Drs. George Peacock, resident in pathology; Ed Winga, resident in internal medicine; John T. Davis, assistant professor in radiology; Norman M. Kaplan and Daniel W. Foster, assistant professors in internal medicine; Leonard Madison, professor in internal medicine; Donald Seldin, professor and chairman, Department of Internal Medicine; and James B. Ketchersid, clinical assistant professor in pathology.

hormone (TSH). Urine 17-hydroxycorticoids were normal with no response to Metopirone. An intermediate PPD skin test was positive, but sputum cultures for acid-fast bacilli were negative. The patient was thought to have anemia secondary to diverticulosis, pleural effusions of unknown etiology, and anterior pituitary hypofunction of unknown etiology. Urine 17-hydroxycorticoids per 24 hours were 5.9 mg. (control), 6.7 mg. after Metopirone, and 16.3 mg. two days after administration of ACTH. He was discharged; prescribed medications were thyroid, 15 mg. per day; hydrocortisone, 10 mg. twice a day; methyltestosterone, 10 mg. per day; Digitoxin, 0.1 mg. every other day; and Colace.

His third admission was in November, 1962, because of acute cholecystitis, cholelithiasis, biliary calculus of the common bile duct, cholangitis, and acute pancreatitis. An initial cholecystostomy was done, but definitive operative procedures were deferred because of an acute anteroseptal myocardial infarct. On Jan. 10, 1963, a cholecystectomy was done. Because of a slow heart rate and atrioventricular block, digitalis was stopped and never resumed. Other medications were unchanged except for the addition of 0.1 mg. Thyroxine per day.

In February, 1963, the patient was seen in the emergency room, with a history of tremors on the right side for two weeks, and urinary retention (775 cc.). The next day, grand mal seizures occurred. This precipitated his fourth admission, during which his neurological symptoms disappeared within a few days. Dilantin was prescribed. Results of a lumbar puncture were normal, and an electroencephalogram revealed a pattern compatible with tumor on the right. It was considered that the seizures might have been secondary to catheterization with the abrupt removal of 1,300 cc. of urine. Visual fields showed no hemianopsia, and it was felt that radiation therapy was not indicated, although more enlargement (since August, 1962) of the sella turcica was noted.

His fifth admission was in February, 1963, because of hematuria and nodular hyperplasia of the prostate. Because of heart failure which was precipitated by taking excess hydrocortisone, an operation was deferred and later refused by the patient. Results of an intravenous pyelogram were normal, and the sella turcica was reported as "not enlarged." A sinus tract in the right upper quadrant scar was persistently draining purulent material. The patient returned the following month for his sixth admission, during which a transurethral prostatectomy revealed nodular hyperplasia. The seventh admission was in June, 1963, for bilateral basilar pneumonitis, which cleared with penicillin. One sputum culture grew scotochromogens.

His eighth admission was in August, 1963, because of a subendocardial myocardial infarct. The PBI was 5.5 μg. per 100 ml. Thyroxine was discontinued. Hydrocortisone was increased to 40 mg. per day because of minimal hyponatremia. His ninth admission was in December, 1963, because of a right hilar pulmonary infiltrate which cleared after treatment with penicillin. Following this, jaundice developed and the patient was readmitted in January, 1964. It was thought that the jaundice was secondary to methyltestosterone therapy. Because of a cholesterol level of 563 mg. per 100 ml., the question of myxedema arose, and l-Thyroxine was given in low doses. His eleventh admission was in May, 1964, for acute pyelonephritis, which was treated with tetracycline. The right upper quadrant sinus was still draining, and finally in September, 1965, a stitch was found and removed, and the lesion healed. The l-Thyroxine was increased to 1.25 mg. per day on Nov. 19, 1965.

The final admission was on Jan. 1, 1966. The patient fell early that morning, and had hemiparesis on the left side. He had weakness, frontal headache, diarrhea, and nausea on the day prior to admission, but no other symptoms. He was taking cortisol (20 mg. per day), Peritrate, Dilantin, l-Thyroxine, and trinitroglycerine. Physical examination revealed temperature, 99.6 F.; pulse, 72 and irregular; respirations, 18; blood pressure, 164/78 mm. Hg. The patient was oriented and in no acute distress, and his speech was minimally slurred. New physical findings included nystagmus to the right, left facial weakness, weak gag reflex, poor sphincter tone, absent pedal pulses, decreased sensation, and motor weakness of the left arm and leg with a left Babinski response. Laboratory studies were essentially within normal limits. Lumbar puncture revealed clear fluid with an opening pressure of 10 cm. and no cells. Electrocardiogram showed atrial fibrillation and nonspecific S-T and T wave changes. The patient did well until that night, when his tempera-

ture rose to 103.6 F. The patient died shortly thereafter, on Jan. 2, 1966, without any noted aspiration.

Radiologic Findings

DR. DAVIS: A skull series (September, 1965) shows an enlarged sella turcica; the anteroposterior measurement is 19 mm., and the depth is 12 mm. The posterior clinoid processes are eroded and almost invisible; the anterior clinoids are asymmetrical and appear undercut. The sellar floor is eroded, and there is soft tissue apparently extending minimally into the sphenoid sinus. There is no calcification within the sella or suprasellar region. The calcified pineal gland is in normal position. The findings are those of a pituitary mass. In the absence of calcification, or signs or acromegaly, this probably represents a chromophobe adenoma of the pituitary.

Films of the chest made in January of 1966 show the heart to be minimally enlarged in transverse diameter with a contour which is not specific for any individual chamber enlargement. The diaphragm is slightly low but not completely flat, and there are rather prominent, coarse, reticular markings throughout both lung fields in addition to the normal vascular markings. There is no confluent infiltrate in any location, and these findings may indicate the effects of chronic bronchopulmonary disease with repeated low grade episodes of bronchitis, bronchopneumonia, or both.

Clinical Discussion

DR. KAPLAN: This elderly man was apparently in good health until just before he came here in August of 1962. His initial hospitalization could have been related to primary gallbladder disease. Subsequently, he had acute cholecystitis, and later a cholecystectomy revealed cholelithiasis. In addition, on his initial admission, evidence of chronic bronchopulmonary disease with infiltration was noted. Presumably this represented recurrent bronchitis and some degree of pulmonary fibrosis. He also had arteriosclerotic heart disease with electrocardiographic manifestations of myocardial infarction and a history of angina pectoris.

When the patient was first seen, there were clinical features that suggested panhypopituitarism, and he was evaluated for this. The patient was pale, had little axillary and pubic hair, slow reflexes, hyponatremia, low voltage on electrocardiogram, and an enlarged sella turcica. The initial measurements of thyroid function were unreliable because of the recent use of Telepaque for the gallbladder series. Uptake of ^{131}I was initially low, 7.5 percent, and after three days of treatment with TSH the uptake was 12.7 percent. This is a questionable, borderline response to three days' treatment with TSH. Perhaps the initial low value was the result of the iodine released from the Telepaque. But, in general, TSH is able to overcome this, as well as any suppression from exogenous thyroid hormone therapy. This response is definitely more than should be seen with primary hypothyroidism, but not as good a response to TSH as one would want before making a definite diagnosis of secondary hypothyroidism due to pituitary insufficiency.

But, a poor response to TSH is seen in a few patients with hypopituitarism; perhaps it is related to long-standing atrophy and fibrosis of the thyroid.[1] The patient had initial basal corticoid values which were normal. This is compatible with panhypopituitarism. In making the diagnosis of hypopituitarism, the basal 17-ketosteroids are oftentimes of more value than a basal corticoid level, because there is a floor to the 17-ketosteroid determination in a male, with a normal lower limit around 8 mg. per 24 hours. With pituitary or adrenal deficiency, the 17-ketosteroids level is below 8 mg. On the other hand, corticoid values in normal patients may be as low as 2 or 3 mg., and even in patients who have had total adrenalectomy we may find 1 or 2 mg. of presumably nonspecific chromogen showing up on the corticoid determinations. Thus, normal basal corticoid levels are often seen in panhypopituitarism. This patient's failure to respond to Metopirone can be taken as evidence that he did not have a normal ACTH reserve, and this would be compatible with a diagnosis of hypopituitarism.

Growth hormone assay after the provocation of hypoglycemia was not available when this patient was first studied. This is a sensitive assay. If it is properly performed with the patient in a basal state initially and with a good hypoglycemic response provoked by insulin, a rise in growth hormone levels should be observed, providing a specific measurement of pituitary function. Of course, there is an obvious danger in performing any type of insulin tolerance test

to provoke hypoglycemia in such patients, but if done carefully, this test is valuable.

The patient apparently did have an enlarged sella. The most likely cause of panhypopituitarism with enlarged sella is chromophobe adenoma. If all cases of hypopituitarism are considered, postpartum necrosis of the pituitary, or Sheehan's syndrome, has been the most common cause. However, as better obstetric care has become available, this is becoming less common, and now chromophobe adenoma is the most common cause overall.

Two other possibilities in this patient should be brought to mind, although I think they are much less likely, purely on a statistical basis, than chromophobe adenoma. First, he could have had metastatic carcinoma in the pituitary. This occurs and can lead to panhypopituitarism. The fact that this patient lived for three and a half years makes metastatic tumor unlikely, but there are some suggestive elements to his story which would support this diagnosis. He was, at one time, thought to have a lesion on the right side of the brain; focal neurological findings could have represented metastatic carcinoma. He also had evidence of obstructive jaundice which could represent liver involvement; however, since the jaundice cleared without specific therapy, it is unlikely that it was related to metastatic liver disease. In addition, recurrent pulmonary infiltrates were seen on chest x-rays. Apparently, these also cleared without treatment. Thus, there is no strong evidence for cancer elsewhere, and the possibility of metastatic carcinoma in the pituitary is slight. The second possibility, also unlikely, is some type of granulomatous involvement of the pituitary, such as tuberculosis or sarcoidosis. He did have atypical mycobacteria in his sputum, positive reaction to tuberculin skin tests, and pulmonary infiltrates. But all in all, I think it is extremely unlikely that he had granulomatous disease of the pituitary. We are left with the most probable diagnosis: chromophobe adenoma with mild but definite panhypopituitarism, for which he was treated.

I doubt that his terminal episode was related to panhypopituitarism. Probably the most common cause of death in patients with panhypopituitarism is hypoglycemia. The majority of patients who die from hypopituitarism become comatose, and in the series reported by Sheehan and Summers,[2] hypoglycemia was by far the most common mechanism for this. Our patient apparently had a cerebrovascular accident. While he was comatose, fever developed, and it is conceivable that in the absence of adequate food intake, terminal hypoglycemia could have developed as one of the factors leading to his death.

DR. MADISON: I agree with Dr. Kaplan's diagnosis and with his analysis of the case. On the patient's initial admission, if indeed he was myxedematous and I believe he was, the pleural effusion could be ascribed to the myxedema, because both pleural effusion and ascites can occur in myxedema. If there were no other apparent explanation (it is difficult to tell from the protocol whether or not the pleural effusion could be ascribed to another disease), then myxedema is an adequate enough cause. The pleural fluid in myxedema has a high protein content. On the other hand, the hydrothorax also could be explained by congestive heart failure, especially if the venous pressure was high. I don't see why it was stated in the protocol that there was no reason for his having bilateral pleural effusions on his first admission. Later, the l-Thyroxine was discontinued, and the hydrocortisone dosage was increased because of hyponatremia. However, hyponatremia also is a feature of myxedema and responds to thyroid therapy.

On his last admission there are several things of interest. On the day before hemiparesis developed, he complained of weakness, diarrhea, and nausea; it is possible that the hydrocortisone was not absorbed, that he vomited it, or perhaps didn't take it because of nausea. If extracellular fluid depletion was present at that time, he would be extremely sensitive to orthostatic hypotension. This could lead to infarction of the brain without thrombosis of a cerebral vessel. In other words, peripheral circulatory collapse could have produced hemiparesis. This, however, does not seem to be the case, judging from his final blood pressure, which was recorded in the normal range. However, that blood pressure may have been obtained while the patient was recumbent, which would obscure the presence of orthostatic hypotension.

DR. FOSTER: Apparently the patient died from a cerebrovascular accident involving the right middle cerebral artery, and I suppose that on a statistical basis this would

be overwhelmingly likely. However, the patient was described as having nystagmus to the right at the time he was examined. This being true, one would have to offer as an alternative possibility a lesion which was lower in the central nervous system, specifically in the lower portion of the pons involving the vestibular nucleus.

DR. SELDIN: The protocol mentions that the patient had neither pubic nor axillary hair. This is of considerable clinical importance. In old people it is common for axillary hair to be scanty, but total absence of both axillary and pubic hair is distinctly uncommon. This finding in our case is evidence for pituitary disease.

There are two causes that one might consider for sellar enlargement. One is that he has a lesion of the sella, and a chromophobe adenoma would be most likely. The second possibility is that he had primary myxedema. In animals, primary myxedema not uncommonly gives rise to sellar enlargement, and recently several cases have been reported of children with primary myxedema whose sella became enlarged. Thus, primary myxedema may cause secondary sellar enlargement. We had one patient whom we thought was in this category a number of years ago. It is important, however, to recognize that in those instances where primary myxedema gives rise to secondary sellar enlargement, one either finds no functional pituitary disturbance or a hypersecretory pituitary syndrome, but not overall pituitary failure. For this reason, then, the presumptive evidence of pituitary panhypofunction with deficiency of other trophic hormones would militate strongly against primary myxedema with secondary pituitary insufficiency. It seems quite likely, therefore, that the patient had a chromophobe adenoma.

The notion that the serum cholesterol level is an important distinguishing feature between primary and secondary myxedema is now more or less discarded. The most important evidence for secondary myxedema in this case is the ^{131}I of 7.5 percent with a rise, albeit modest, after TSH treatment. The rest of the picture as well as the sellar enlargement is impressive, and I think it is clear that the patient had a sellar tumor.

With regard to his terminal event, the evidence that this lesion involved the lenticulostriate artery is poor, for the reasons that Dr. Foster mentioned. I agree with him that it was more likely a lesion in the pons, either a thrombosis or a direct extension of the tumor.

The patient was given maintenance doses of 20 mg. of hydrocortisone, spaced regularly; this is a low dose, and not properly proportioned. The diurnal variation in hydrocortisone secretion is such that higher doses should be given in the morning than in the evening to simulate the normal pattern of hydrocortisone secretion. The average secretory rate in a normal active individual would be about 25 to 30 mg. a day, and a sick person would need more. It is clear that the total dose of hydrocortisone as well as the way it was divided up during the day was not optimum.

Pathologic Findings

DRS. PEACOCK and KETCHERSID: At necropsy the body was that of a thin, elderly, white man with thin, smooth skin and no appreciable axillary or pubic hair. Examination of the cranial cavity and its contents revealed, as has been clinically suggested, a pituitary tumor (Fig. 1). This tumor was soft and brown with a dumbbell shape produced by the diaphragm of the sella. The inferior portion of the tumor measured 3.4 by 3.2 by 1.9 cm., while the superior portion measured 2.2 by 1.8 by 1.4 cm. The sella turcica was expanded, and the posterior

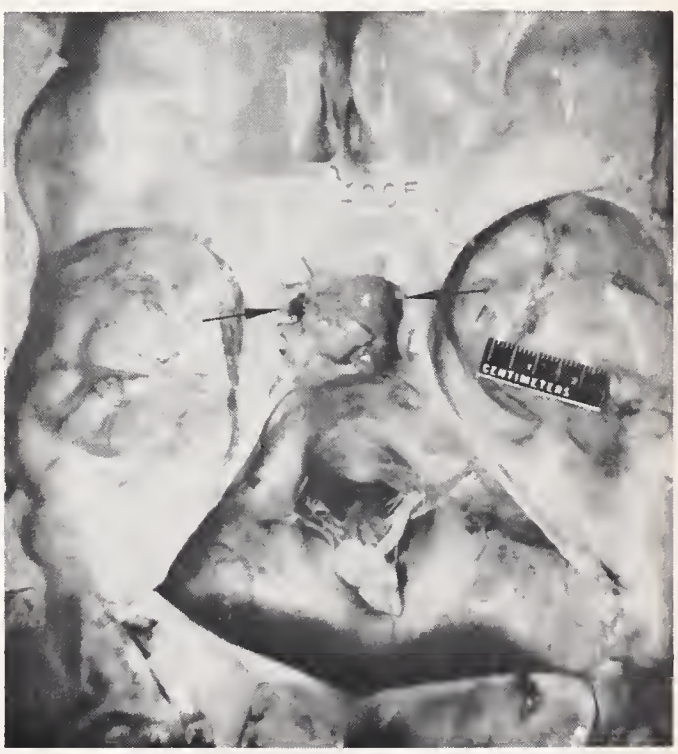

Fig. 1. Base of skull after removal of brain. Note the internal carotid arteries (arrows), between which lies the pituitary tumor. The right internal carotid artery is barely visible because of compression by the pituitary tumor.

clinoid processes were short and blunt. However, there was no extension of the tumor into the sphenoid air cells. The middle clinoid processes were blunt. Laterally, the pituitary tumor displaced and compressed both internal carotid arteries, the right more than the left. Although there was modest anterior displacement of the optic chiasm by the superior portion of the tumor, no optic atrophy could be demonstrated microscopically, and visual defects were presumably absent. Microscopic examination of the tumor revealed a chromophobe adenoma composed of a sheet of small, round cells with round to oval, dark nuclei and poorly demarcated, lavender-staining cytoplasm (Fig. 2). No residual normal pituitary tissue was present, and the patient did have hypopituitarism. As a result of loss of the trophic hormones of the adenohypophysis, the thyroid gland was atrophic with interstitial fibrosis. The adrenal cortices were likewise atrophic.

Examination of the carotid arteries and circle of Willis revealed mild atherosclerosis but no recent or old occlusion. There were no thrombi in the terminal branches of the right middle cerebral artery. However, there was an infarct, 36 to 48 hours old, measuring 3 by 3 by 2 cm., involving the posterior limb of the right internal capsule (Fig. 3). In the absence of severe atherosclerosis or thrombosis of the cerebral arteries, the compression of the right internal carotid artery by the pituitary tumor coupled with a transient episode of hypotension is the probable cause of the infarct.

The heart was moderately hypertrophied, weighed 500 gm., and was the seat of severe coronary atherosclerosis. Both right and left coronary arteries were severely narrowed by roughened, friable, yellowish-tan atherosclerotic plaques, but no old or recent complete occlusion was present. The anterior descending branch of the left coronary artery was most severely involved by atherosclerosis and, correspondingly, there was a healed, subendocardial, anteroseptal myocardial infarct. Microscopic examination revealed foci of fibrosis throughout the myocardium, particularly in the posterior basal region in the area of the bundle of His. This fibrosis, resulting from ischemia, interrupted the conduction mechanism and caused the clinically appreciated conduction defects. All four chambers of the heart were dilated; this probably occurred terminally, because there was no evidence of chronic congestive heart failure in the other organs. There was severe pulmonary congestion and edema, indicating terminal cardiac decompensation.

The gallbladder was absent, and the common bile duct was dilated but contained no stones. Whether the episode of jaundice in January, 1964, was the result of choledocholithiasis or was drug-induced is uncertain. The liver was unremarkable.

Although there was no evidence of recent bleeding within the bowel, there were two lesions which could have caused the clinically manifest guaiac-positive stools and anemia during the patient's previous hospitalization. Colonic diverticulosis was present; in addition, there was a small hemangioma within the jejunum. Although this hemangioma was covered by an intact epithelium, it could have ulcerated and bled in the past.

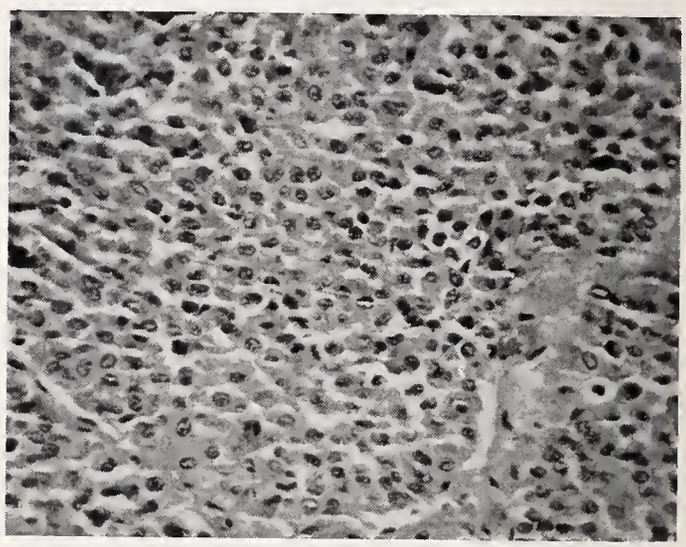

Fig. 2. Photomicrograph of pituitary tumor. X 344.

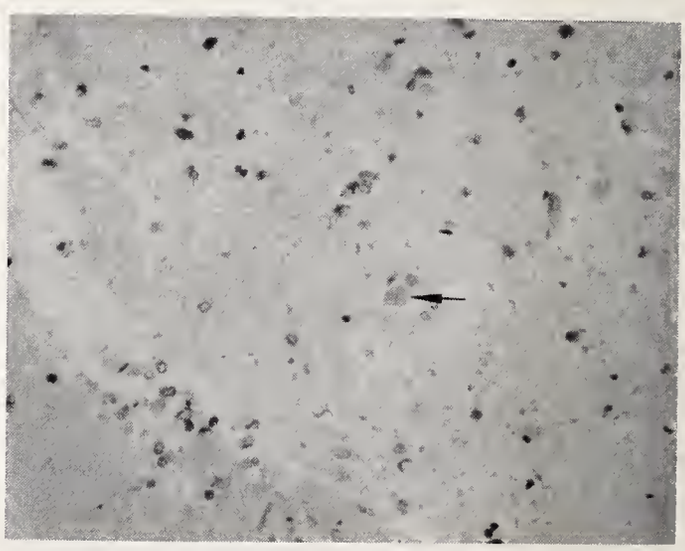

Fig. 3. Photomicrograph of infarct of brain. X 344. The arrow indicates a necrotic neuron. Note the beginning disintegration of the cerebral tissue (lower left) and the leukocytes which are scattered through the tissue.

The lungs were the seat of chronic bronchitis and obstructive emphysema of moderate severity. In addition, bilateral apical fibrous scars as well as a calcified hilar node were identified, probably the result of old tuberculosis. Finally, the terminal elevation in temperature was most likely the result of acute purulent bronchiolitis in the lower lobe of the right lung.

In summary, this 80-year-old white man had longstanding panhypopituitarism which resulted from a chromophobe adenoma of the pituitary. This adenoma had grown around and compressed the right internal carotid artery, and an anemic infarct of the right cerebral hemisphere occurred 36 to 48 hours before the patient's death. The patient also had severe coronary disease and chronic bronchopulmonary disease.

DR. KAPLAN: In retrospect, it was probably an error not to give this patient radiotherapy when he was first seen. In a patient with a chromophobe adenoma who has an enlarged sella with or without evidence of panhypopituitarism, but without evidence of extension of the tumor to the optic nerves, irradiation is probably the therapy of choice.[3] In the near future, however, cryohypophysectomy will probably be the therapy of choice. If the tumor is large, has extended outside the sella, and has definitely involved the optic nerve, then craniotomy and surgical removal of the pituitary tumor is the treatment of choice. I think this patient should have received external pituitary radiation in August of 1962.

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The authors, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas 75235.

Too Many Seat Belts Go Unused

The U.S. Department of Health, Education, and Welfare reports that more people have seat belts in their cars than ever before. Many riders, however, the Department says, especially occupants of newer models, risk death by failing to wear these belts.

Researchers at Cornell Aeronautical Laboratory, Inc. of Buffalo, New York, report this finding from a study of a selected group of automobile accidents from 1956 to 1965. The study was supported by the US Public Health Service's Division of Accident Prevention and the Automobile Manufacturers Association.

Since 1964, new cars have had seat belts as standard equipment in the front seat. Seat belts became standard equipment in front and back seats of 1966 models.

The report, however, found that only 30 percent of the people in 1965 model automobiles were wearing their seat belts in the accidents under study. Thirty-seven percent of the occupants of 1961-62 models were wearing their belts.

"It has been estimated that the lives of 5,000 persons would be saved every year if everyone wore seat belts," said Dr. Paul Joliet, chief of the Division of Accident Prevention. "The deaths of persons in automobile accidents are even more tragic when the seat belts that might have saved them are found lying unused," he added.

The "Socio-geographic" mystery

Why is one man's gastric ulcer another man's duodenal?

Geographic variation in the *incidence* of peptic ulcer is a familiar fact. But the proclivity of certain *kinds* of ulcer for certain geographic areas is a recently recognized phenomenon.[1,2]

For example, in one particular Norwegian fishing village there is a tendency for patients to develop a gastric ulcer; anywhere else in Norway, ulcers are usually duodenal. Peruvians high in the Andes have more gastric ulcers than their compatriots in the lowlands. Why? Nobody knows.

Social variations, too. Even in the same geographic areas there are interesting variations. An Englishman's ulcer depends on his social standing—professional men suffer with duodenal ulcers, while workingmen have more of the gastric variety. In southern India the pattern is reversed. Here, duodenal ulcers are common among laborers and agricultural workers and rare among the upper classes.

Investigators are exploring every possible theoretical avenue in their search for the cause of peptic ulcer. Of all the factors implicated in ulcerogenesis, the one that is generally acknowledged to be of primary importance is hypersecretion of gastric acid.[3-8] Or, as one author states it: "The medical management of peptic ulcer pharmacologically is, in the final analysis, concerned largely with the effective inhibition of peptic activity."[3]

Robinul (glycopyrrolate) provides potent, rapid, specific antisecretory action confirmed by gastric analyses and x-ray evidence of clinical effectiveness.[3,7,9-12] Relieves pain with "impressive" promptness.[8] Quickly alleviates acute discomfort, and effectively counteracts gnawing pain, preprandial midepigastric pain, belching and other ulcer symptoms.[7] Suppression of nocturnal pain is "outstanding."[13] Maximally effective doses may be given with minimal side reactions, and the incidence of unwanted anticholinergic effects is negligible.[3,7-14]

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promotes the essential ulcer-healing environment

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Contraindications: Glaucoma, urinary bladder neck obstruction, pyloric obstruction, stenosis with significant gastric retention, prostatic hypertrophy, duodenal obstruction, cardiospasm (megaesophagus), and achalasia of the esophagus, and in the case of Robinul-PH Forte, sensitivity to phenobarbital.

Precautions: Administer with caution in the presence of incipient glaucoma.

Adverse Reactions: Dryness of the mouth, blurred vision, urinary difficultics, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.

Dosage: Dosage should be adjusted according to individual patient response. Average and maximum recommended dose is 1 tablet 3 times a day: in the a.m., early p.m., and at bedtime. *See product literature for full prescribing information.*

Supply: Robinul (glycopyrrolate 1 mg.); Robinul Forte (glycopyrrolate 2 mg.); Robinul-PH (glycopyrrolate 1 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming); Robinul-PH Forte (glycopyrrolate 2 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming.) In bottles of 100 and 500 scored tablets.

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I certify that the statements made by me above are correct and complete.

E. Moxwell Jones, Business Manoger

NEWS

Plans Announced for TMA Winter Meeting; Other Texas Groups Ready for December

TMA Legislation Conference Scheduled for Jan. 21

Five main speakers have been scheduled to participate in the Texas Medical Association's 1967 Conference on Legislation and Symposium for County Medical Society Officers. The Conference is planned for the TMA Headquarters Building, 1801 North Lamar Blvd., Austin.

Scheduled to speak are Mr. C. Joseph Stetler, president of the Pharmaceutical Manufacturers Association, and Dr. Ernest B. Howard, assistant executive vice-president of the American Medical Association. Mr. Stetler will speak to the Conference on issues related to drugs which will be placed before the 90th Congress. Dr. Howard will speak on "Socio-Economic and Medical Practice Trends."

The three other scheduled speakers are Hon. Preston Smith, Lieutenant Governor of Texas, Hon. Ben Barnes, speaker of the Texas House of Representatives; and Hon. Omar Burleson, Anson, Tex., Representative, Congress of the United States, 17th District of Texas.

The program will also include two panel discussions: "Medical and Health Issues Before the 1967 State Legislature," and "Stimulating and Evaluating County Medical Society Activities."

Conference objectives are to inform those in attendance of their obligations and responsibilities as medical society leaders and to inform them of the issues and problems which they may face in the year ahead. The Conference will be attended by presidents, presidents-elect, secretaries, executive secretaries, delegates, and legislative chairmen of the 115 component county medical societies, and by officers and committee members of TMA and the Woman's Auxiliary to TMA.

Ophthalmology, Otolaryngology Society To Convene Dec. 2-3

The Texas Society of Ophthalmology and Otolaryngology will hold its 41st Annual Meeting Dec. 2 and 3 at the Inn of the Six Flags, Arlington.

Guest speakers for ophthalmology will be Dr. Graham Clark of New York City and Dr. P. R. McDonald of Philadelphia. Guest speakers for otolaryngology will be Dr. Hollie E. McHugh of Montreal, Canada, and Dr. Frank D. Lathrop of Boston.

There will be a ladies' luncheon for doctors' wives and a formal dinner-dance on Friday. A business meeting will take place Saturday afternoon immediately following the scientific session.

The meeting is open only to members of the society. Members may bring guests if approval of the society president is obtained.

M. D. Anderson To Hold 11th Clinical Conference

"Cancer of the Uterus and Ovary" will be the subject of the 11th Annual Clinical Conference to be held at The University of Texas M.D. Anderson Hospital and Tumor Institute in Houston, on Dec. 1 and 2.

Outstanding scientists and physicians from the United States and other countries will join staff members from M.D. Anderson Hospital in presenting current information on diagnostic procedures and treatment for carcinoma of the uterus, uterine cervix, and ovary.

Co-sponsor of the conference is the Division of Continuing Education of The University of Texas Graduate School of Biomedical Sciences at Houston.

Presentations will include: "Treatment at M.D. Anderson Hospital," and "Corpus et Collum," Dr. Richard C. Boronow, Chicago; "Special Aspects of Radiation Treatment," Dr. Luis Delclos, M. D. Anderson Hospital; "Hormone Therapy," Dr. Julian P. Smith, M. D. Anderson Hospital; "Undifferentiated Tumors," Dr. Creighton

L. Edwards, M.D. Anderson Hospital; "Value of Pre- and Postoperative Irradiation," Dr. John M. Morris, New Haven, Conn.; "Individualization of Therapy with Regard to Extension and Microscopic Structure," Dr. H. L. Kottmeier, Stockholm, Sweden; and "Role of Surgery," Dr. Hugh R. K. Barber, New York City.

Papers to be presented dealing with diagnosis and treatment for carcinoma of the ovary include: "Treatment," Dr. Beaury C. Burns, Jr., and Dr. Paul B. Underwood, M.D. Anderson Hospital; "Pathological Findings after Chemotherapy," Dr. H. Stephen Gallager, M. D. Anderson Hospital; "Special Tumors," Dr. Dean J. Candis, M.D. Anderson Hospital; and "Trends in Cooperative Management of Cancer of the Cervix," Dr. Fernando G. Bloedorn and Dr. James P. Gallaher, both of Baltimore.

Carcinoma of the cervix presentations will include "Shifts in Interest in Radiotherapy," Dr. Gilbert Fletcher; "Radium Dosimetry to Regional Lymph Nodes," and "Treatment following Inadequate Primary Surgery," Dr. Fred Durrance; "Combination Irradiation and Surgery," Dr. Felix N. Rutledge; "G. I. Tract Complications," Dr. Julian Smith and Dr. Patrick E. Golden; "G. U. Tract Complications," Dr. Beaury Burns and Dr. Richard T. Upton; "Complications of Treatment," Dr. John Morris; and "Sites and Mechanism of Recurrences," Dr. R. Vernon Colpitts, and Dr. Robert E. Rogers. All these speakers are from M.D. Anderson Hospital.

Other papers to be presented include "Treatment of Recurrent Carcinoma," Dr. H. L. Kottmeier; "Surgery for Recurrence," Dr. Hugh Barber; "Regional Chemotherapy," Dr. Julian Smith; "Palliation with Radiogold Grains for Postirradiation Recurrences," Dr. Lowell Miller, M.D. Anderson Hospital; "Low Vaginal Metastases," Dr. Luis Delclos; and "Distant Metastases," Dr. Delclos and Dr. Victor Carlson, M.D. Anderson Hospital.

The annual clinical conferences are designed to give the physicians of Texas and other interested physicians the most current diagnostic and therapeutic procedures for the treatment of cancer patients. A particular aspects of cancer is chosen for each conference.

Partial support for this year's clinical conference was contributed by the American Cancer Society, Texas Division, and a Community Cancer Demonstration Project Grant from the Cancer Control Program, USPHS.

AMA, TMA Cosponsoring Clinical Nutrition Lectures

Texas Medical Association, in cooperation with the American Medical Association, is cosponsoring clinical nutrition lectures at ten colleges and universities during the current academic year.

The AMA Council on Foods and Nutrition initiated the clinical nutrition lecture program in the fall of 1964. The program is being carried out on a regional basis with several lecturers participating.

The lectures are designed to stimulate undergraduate students to consider careers in the health sciences, as well as to inform the audience of recent developments in the field of nutrition.

A total of 36 lectures this year have been scheduled in the eight-state area which includes Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, South Carolina, and Texas.

Dr. Robert E. Olson, director of the Department of Biochemistry at St. Louis University School of Medicine, will lecture at North Texas State University in Denton Nov. 21 and at Abilene Christian College in Abilene Nov. 22.

Dr. C. E. Butterworth, Jr., director, Nutrition Division, University of Alabama Medical Center, Birmingham, will speak at Southwest Texas State College in San Marcos on Nov. 21 and at Lamar State College of Technology in Beaumont on Nov. 22.

Texas ACP Internists Plan Galveston Meeting Dec. 8

The American College of Physicians (ACP) will hold a scientific regional meeting Dec. 8 for internal medicine specialists in Texas at the Flagship Hotel in Galveston.

The regional meeting is one of 29 scientific sessions sponsored each year by the ACP throughout the United States and Canada.

Dr. Edward C. Rosenow, Jr., of Philadelphia, ACP Executive Director, will be a special guest. Dr. Hatch W. Cummings, Jr., of Houston, ACP governor for Texas and professor of clinical medicine at Baylor University College of Medicine, is in general charge of the meeting.

Pathologists To Hold Seminar

The San Antonio Society of Pathologists will conduct its 23rd Annual Tumor Seminar on Saturday, Dec. 3, at Brooke General Hospital, Fort Sam Houston.

Guest speaker will be Dr. Saul Kay of the Medical College of Virginia in Richmond.

Hospitals, Clinics To Meet

The annual convention of the Private Clinics and Hospitals Association of Texas will take place at the Driskill Hotel in Austin, Dec. 9-10.

Dr. H. A. Baker, Box 267, Wills Point, Tex., is president.

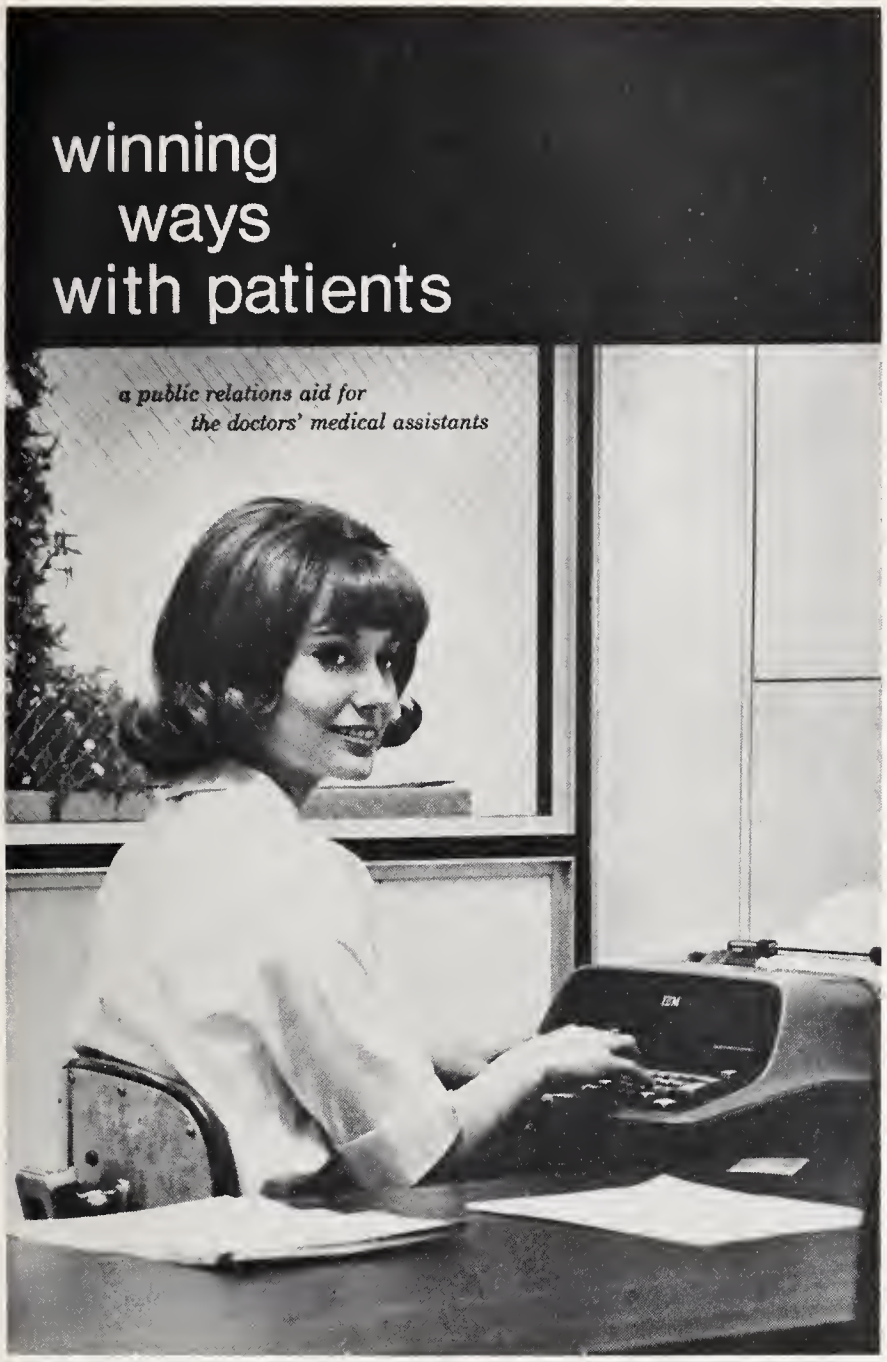
Radiological Society Plans Annual Meeting in Chicago

The Radiological Society of North America will hold its 52nd annual meeting at the Palmer House in Chicago Nov. 27-Dec. 2.

The session will include a business meeting, a meeting of counselors, and scientific sessions. Topics to be taken up in scientific sessions include neuroradiology, radiotherapy, cardiovascular radiology, nuclear medicine, pediatric radiology, gastrointestinal radiology, radiobiology, and physics.

Texas Drs. Gilbert H. Fletcher, Charles A. Hill, John A. Burdine, Jr., Thomas S. Harle, James R. Stevenson, Victor Carlson, Luis Delclos, Magdalena J. Pogonowska, Lois Cowan Collins, Harold L. Dobson, M. L. M. Boone, and Norah duV. Tapley, all of Houston, will participate in the program.

The refresher courses will begin



MUST READING FOR YOUR MEDICAL ASSISTANT

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Everybody gains when your medical assistants follow the helpful hints given in this 20-page AMA publication—you, your patients and the office staff, too.

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Specifically designed as a guide advising today's medical assistants how to perform their services most effectively, "Winning Ways With Patients" is loaded with practical "how-to-do-it" steps and techniques. Here are just a few of the important areas covered:

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on Sunday, Nov. 27, and be held throughout the meeting. Attendance at these courses is limited to the medical profession, including graduate students and residents in radiology, radiation physicists, radiobiologists, chemists, medical students certified by the deans of their respective medical schools, and others closely allied with the science of radiology. A registration fee of $25 will be charged to non-members of the society.

Health Service Schedules Air Pollution Conference

More than 2,000 persons will gather in Washington, D.C., Dec. 12-14 to discuss problems of air pollution control and to develop guidelines for action in improving and speeding air pollution control.

The National Conference on Air Pollution, called by John W. Gardner, Secretary of Health, Education, and Welfare, will take place at the Sheraton Park Hotel. Persons in attendance will include physicians, scientists, engineers, businessmen, labor leaders, government officials, researchers, community representatives, and public-spirited citizens.

The conference is being sponsored by the Division of Air Pollution, the Public Health Service, the US Department of Health, Education, and Welfare. A steering committee of 21 organizations, representing our nation's communities, industries, and various levels of government, has helped develop the program.

The program will include discussions of air pollution as it affects health, economy, technology, and communities. Panel discussion groups will take up specific aspects of air pollution problems and their controls, such as automobiles, incinerators, factories, and industrial plants. What controls are needed, how they should be paid for, and existing programs will be examined.

Registration for the conference is free. Participants will, however, be responsible for their travel, food, lodging, and other personal expenses. The conference will be open to the public.

MISCELLANY

Sir Hans Krebs, Whitley Professor of Biochemistry at England's Oxford University, is appearing as Ashbel Smith Distinguished Visiting Professor at the University of Texas Medical Branch from Nov. 6-Dec. 15. He was awarded the Nobel Prize in 1953 for his concept of the citric acid cycle. Commonly known as the Krebs Cycle, the popular theory explains the final steps in the conversion of foodstuffs to certain carbon dioxide, water and energy.

Dr. Krebs, who was born in Germany, holds M.D. and Ph.D. degrees in addition to honorary degrees from many famous universities. He was knighted by the Queen of England in 1958.

Commissioner of Social Security Robert M. Ball has announced the selection of 11 organizations which will act as fiscal intermediaries under Medicare. The selected intermediaries will determine the amounts of payments due nursing homes accepted for participation in Medicare. They will pay the nursing homes under two-year renewable contracts.

The Blue Cross Association will act in all states except Hawaii and Nevada, in all territories except the Virgin Islands, and in the District of Columbia. Nursing homes in Texas and in 13 other states will have the alternative of choosing the services of Mutual of Omaha.

The companies available also as carriers in other states are The Travelers Insurance Co., the Aetna Life and Casualty Co., the Kaiser Foundation Health Plan, Prudential Insurance Co., Nationwide Mutual Life Insurance Co., Hawaii Medical Service Association, Intercounty Hospitalization Plan, Inc.; Cooperativa de Salud; and the Community Health Association.

This extended portion of Medicare goes into effect on Jan. 1.

The 89th Congress has been the most health-oriented Congress to date. Of the more than 24,000 bills introduced before the end of July, 1,440 concerned medical care, the practice of medicine, or public health.

The 88th Congress had seen 18,000 bills introduced by this same time, 836 of which were of interest to medicine. The 87th Congress had had 20,000 bills introduced, and only 494 were medically oriented.

Bills passed by the 89th Congress and signed into law by the President included Medicare; the Drug Abuse Control Amendments; the Mental Retardation Facilities Construction Act; Community Health Services Extension Amendments; Health Research Facilities Amendments; Heart Disease, Cancer, and Stroke Amendments; Health Professions Educational Assistance Amendments; laws on air pollution; and laws on water pollution.

Still pending are the Allied Health Professions Personnel Training Act; Comprehensive Health Planning and Public Health Service Amendments; Hospital and Medical Facilities Modernization Amendments; the Child Safety Act; Keogh Act Amendments; and the Medical Restraint of Trade Act.

More than half of the nation's radiologists are sending and collecting bills for their professional services to patients in hospital x-ray departments, according to returns from a September survey of its members by the American College of Radiology.

Based upon a total of 2,768 replies from a questionnaire sent to some 5,800 radiologists in active practice, 1,031 radiologists or radiological groups are billing for all services, and an additional 295 now bill for services in one or more of several hospitals covered. Another 642 radiologists reported negotiations with hospitals to begin separate billing underway.

Some 250 respondents indicated no intention to seek separate billing, and 540 said that their practice did not include a voluntary hospital department.

Bexar County Plans Fair Pavilion, Medical Health Education Center

Plans are underway for the construction of a Health Education Center of the Americas in San Antonio.

A permanent structure, primarily designed to educate the public of the Western Hemisphere in matters of health, will be built as a part of the Hemisfair to be held from April through October, 1968, in San Antonio. The building will house facilities and exhibits to aid the study of both physicians and laymen from throughout the western world.

The proposed health center is a long-time dream of Dr. A. P. Thaddeus, a San Antonio physician instrumental in the presenting of San Antonio health fairs in 1963 and 1965, and of Mr. Robert B. Hitchcock, public relations director for Baptist Memorial Hospital. Plans call for the center to be constructed under the supervision of the Bexar County Medical Society. An autonomous, nonprofit corporation, The Health Education Center of the Americas has been set up and chartered in the state of Texas to oversee the operation of the center.

Design director for the project is Victor Klein who formerly designed the Dallas Health Museum. Mr. Hitchcock will serve as executive director for the center.

Features of the Hemisfair pavilion will include exhibits and displays by commercial firms, medical schools, world nations, health agencies, and other interested parties. The most spectacular feature planned for the center will be a gigantic flame intended to symbolize the ebb and flow of human life, the theme of the center. The flame will leap toward a domed ceiling of artificial sky and stars. As the flame subsides, the artificial sky will fade out and the story of life—its origin, preservation, and perpetuation—will be projected in color on the domed ceiling.

The permanent building will house, in addition to exhibit space, meeting and conference rooms for medical and paramedical personnel. Plans for the center also include using it for short courses for physicians, student health fairs, and scientific demonstrations.

The Health Education Center of the Americas building will be located between the proposed state and federal pavilions which will also be permanent structures. The building will occupy approximately 40,000 square feet on the exposition site. Construction of the pavilion will begin in the summer of 1967, and opening is scheduled for April of 1968.

Bexar County Medical Society hopes to finance the completion of the center from contributions of national, state, and local health agencies, private corporations, medical and paramedical organizations, and from individual members of the medical and paramedical fields. It is hoped that the center will become self-supporting, once the fair has opened.

Signers of the original charter for the Health Education Center of the Americas, Inc., were Dr. Francis E. O'Neill, president of Bexar County Medical Society; Drs. John M. Smith, Jr., Bernard T. Fein, Augustus F. Herff, Jr., William F. Pipes, Max E. Johnson, R. F. Gossett, Jack M. Partain, and Thaddeus; Mr. Hitchcock; and S. E. Cockrell, Jr., executive director of Bexar County Medical Society.

Members of the Board of Trustees of the corporation include Drs. John H. Bohmfalk, William M. Center, J. B. Gonzales, A. H. Hill, Peter B. Kamin, Jack B. Lee, B. B. Markette, E. A. Maxwell, E. L. Mueller, Sr., F. Carter Pannill, J. L. Pipkin, Arthur Ressmann, William Schlattner, Jr., M. L. Thornton, Walter Walthall, Thaddeus, Mr. S. L. Fickel, consultant Mr. Hitchcock, and Mr. Cockrell.

Designer Victor Klein's concept of the proposed exterior of the Health Education Center of the Americas as the building will appear upon completion.

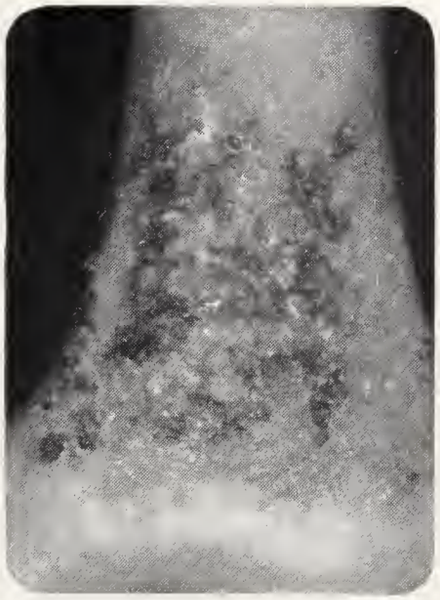
High School To Be Named For Dr. Foy H. Moody

A new high school in Corpus Christi will be named for Dr. Foy H. Moody, who died in 1965. The school is in Inwood Village and will be named Foy H. Moody High School. The school is due to open in the fall of 1967.

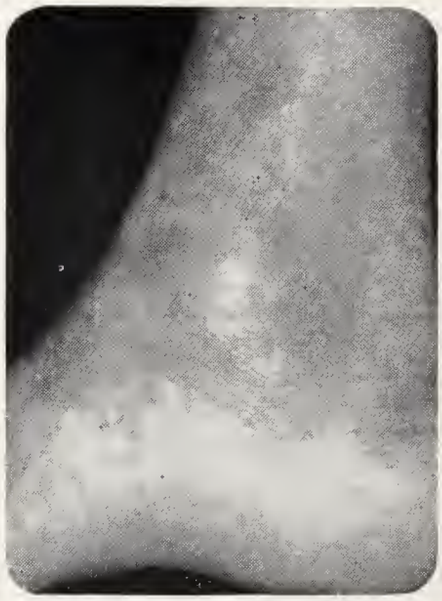
Dr. Moody served seven years on the school board in Corpus Christi. He was vice-president when he resigned. He also served as a director of the Texas School Board Association.

Dr. Moody had been vice-president of Texas Medical Association, president of the Nueces County Medical Society, and secretary of the Fifth and Sixth Districts Medical Societies.

eczema: scourge of childhood



R. R., Age 11—Before treatment—atopic eczema of long standing



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ARISTOCORT® Triamcinolone Acetonide Topicals have proved exceptionally effective in the control of various forms of childhood eczema: allergic, atopic, nummular, psoriatic, and mycotic.

In most cases responsive to topical ARISTOCORT, the 0.1% concentration is sufficiently potent. The 0.5% concentration provides enhanced topical activity for patients requiring additional potency for proper relief.

Administration and Dosage: Apply sparingly to the affected area 3 or 4 times daily. Some cases of psoriasis may be more effectively treated if the 0.1% Cream or Ointment is applied under an occlusive dressing.

Contraindications: Tuberculosis of the skin, herpes simplex, chicken pox and vaccinia.

Precautions and Side Effects: Do not use in the eyes or in the ear (if drum is perforated). A few individuals react unfavorably under certain conditions. If side effects are encountered, the drug should be discontinued and appropriate measures taken. Use on infected areas should be attended with caution and observation, bearing in mind the potential spreading of infection and the advisability of discontinuing therapy and/or initiating antibacterial measures. Generalized dermatological conditions may require systemic corticosteroid therapy. Steroid therapy, although responsible for remissions of dermatoses, especially of allergic origin cannot be expected to prevent recurrence. The use over extensive body areas, with or without occlusive nonpermeable dressings, may result in systemic absorption. Appropriate precautions should be taken. When occlusive nonpermeable dressings are used, miliaria, folliculitis and pyodermas will sometimes develop. Localized atrophy and striae have been reported with the use of steroids by the occlusive technique. When occlusive nonpermeable dressings are used, the physician should be aware of the hazards of suffocation and flammability. The safety of use on pregnant patients has not been firmly established. Thus, do not use in large amounts or for long periods of time on pregnant patients.

Packages: Tubes of 5 Gm. and 15 Gm.; ½ lb. jar.

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Medicare in Texas: The First 120 Days

On the basis of experiences to date, it is apparent that Medicare will have a tremendous effect upon medical service and upon patient care. Certainly no innovation in our lifetime will have a greater impact upon the financing of medical care.

July 1 was a day of major transition, not so much from the standpoint of the impact of Medicare upon patient care as the change in philosophy and the scope of coverage for the aged. Since 1962 our state had been providing a comprehensive program of hospital, medical, surgical, and nursing home benefits for 230,000 Texans on Old Age Assistance. The Kerr-Mills program was extremely well received by the elderly, and it generally was regarded as one of the finest in the nation. The main features of that program for the needy aged came to an abrupt halt on June 30. The emphasis now is on providing benefits for all of the aged, regardless of need or ability to pay.

Most of the 886,000 aged in Texas now are eligible for Medicare's basic hospital, nursing home, and home care benefits. Five hundred fifty-three hospitals in Texas have been approved for Medicare. Twenty-four hospitals in our state have been turned down for participation, most of them because of a shortage of nurses. Unlike some states in the South, there have been relatively few problems related to racial integration and civil rights requirements. Approximately 93.5 percent of the state's 65-and-over population, 827,000 aged Texans, have signed up for Part B, the optional supplementary insurance plan which covers professional services by physicians.

Increase in Demand

With the advent of Medicare, some had anticipated an initial surge in the demand for medical services. It was only logical to assume that many of the elderly would be eager to try out their new benefits. Surprising to some, there was no great immediate increase in the number of patients admitted to hospitals during the first days of Medicare. Since that time, however, the increase in demand for medical services is beginning to be realized. Physicians now report having seen many Medicare patients who are acutely ill as well as some who had postponed elective procedures and treatment for chronic conditions. Starting in mid-July, hospitals have been reporting a larger number of admissions and a higher census of patients over 65.

Even so, these early returns represent only the start. When the first snows fill the air this winter, the impact upon hospital admissions promises to be even greater. January, February, and March are the traditional heavy hospital occupancy months for the year. For the first 12 months of the program, it is probable that about 40 percent of those eligible will be hospitalized sometime during the year.

The first 120 days of Medicare in Texas also have given rise to several other general observations. Despite the tremendous advance promotion and information, there has been a general lack of understanding on the part of the patient about benefits and about the operation of the Medicare program. Many physicians report that they and their office assistants are devoting a significant amount of their time to answering questions and to providing information. This lack of understanding appears to be compounded when the Medicare patient also is eligible for Old Age Assistance benefits. Not only are the beneficiaries somewhat confused, but physicians indicate that they themselves are not fully informed, particularly in the realm of billing and reimbursement procedures.

Many doctors also have pointed out that Medicare has generated an increased volume of paper work, and along with it, higher office and administrative costs. Some clinics have reported that they have employed a full-time assistant just to open and to post Medicare accounts.

Payments Delayed

Along with the attending increase in paper work, some doctors have encountered delays in the payment of claims. Some physicians have expressed concern that the delay might well be due to the close scrutiny of medical charges by the fiscal intermediary. According to information received from Blue Shield, however, this supposition would not seem to be fully justified. Blue Shield reports that 96 percent of all claims so far have satisfied the criteria of reasonable charges. The remaining four percent of some 15,000 claims which are being received daily must be reviewed individually. Blue Shield has indicated that incorrect billing and the lack of itemization or breakdown of charges have been responsible for many of the delays.

Since the start of Medicare, considerable attention has been given to the two options given to physicians for collection of fees: (1) direct billing of the patient or (2) accepting an assignment from the patient for services rendered.

In this connection, Blue Shield reports that 60 percent of the claims which it has received have been filed by physicians, the remainder by patients. These figures probably do not represent an entirely accurate barometer of the percentage of physicians who are taking assignments, and those who are billing directly. Medicare does not provide reimbursement for the

first $50 of professional services; therefore, claims received by Blue Shield undoubtedly represent only a portion of the bills rendered by physicians for professional services. At any rate, it is evident that the philosophy of individual responsibility has had its impact. Many more physicians than ever before are rendering their bills directly to patients.

In regard to physician-hospital relations, Medicare has created several problems. Some are of a transitory nature; others have deep and scarring effects. At the start of Medicare, considerable confusion was caused by the requirement of a special form for certifying the medical necessity for admission of patients, and for recertifying the patient on or before 14 days and thereafter. This problem was accentuated at the time when the American Hospital Association distributed a sample form to its member hospitals. Fortunately, that issue has been alleviated by the pronouncement by the Department of Health, Education, and Welfare (HEW) that no special form is required. Nevertheless, HEW has stipulated that physician certification must include the words, "medically necessary."

Some other rough spots which were anticipated fortunately have not materialized. One of these relates to utilization committees. We have heard of relatively few problems on the operation of these committees in hospitals throughout the state.

Separation of Fees

The most pressing problem which has been brought into sharper focus by Medicare is the controversy involving hospital-based specialists. TMA has been working diligently to assist hospital-based physicians in their effort to separate their professional fees from hospital charges. The House of Delegates has supported the position that radiologists and pathologists, like other physicians, are rendering professional services, and that all doctors should accept full responsibility for establishing, billing, and collecting fees. Significant progress has been made in some communities throughout the state, particularly as it applies to Medicare patients. Nevertheless, this is a deep-seated problem with many complex ramifications. It will be difficult to resolve, particularly as long as so many divergent views remain among some of the specialists themselves, the hospitals, and the insurance companies.

From these reports, and despite some of these problems, we would have to conclude that the initial impact of Medicare has been somewhat less than expected. The new program generally has been handled in stride, and this reflects great credit upon physicians, hospitals, Blue Cross-Blue Shield, and their dedication to patient care.

Increase in Medical Needs

Nevertheless, the long-term outlook is not nearly as reassuring. As the aged become more aware of their benefits, and as they satisfy the initial deductibles each year, they most certainly will utilize more fully this new federal program. As an illustration, perhaps these figures reflect the impact of government financed care on hospital utilization. In 1962, at the start of the Kerr-Mills program, the elderly occupied 24 percent of the hospital beds in Texas. This past May, just prior to Medicare, 26 percent of the patients in Texas hospitals were 65 years of age or older. Two months later, in July, after the start of Medicare, occupancy by the aged had risen to 30 percent. According to the latest Blue Cross-Blue Shield survey, bed occupancy by the elderly climbed to 31 percent in September. Thus, in a relatively brief period, hospital bed occupancy by the aged has gone up from 24 to 31 percent. For this specific age group, this represents an increase of 29 percent.

We anticipate that the demand for medical service will continue to increase, gradually but substantially, over the years ahead. We also believe that the cost of the program (estimated at 3 billion, 900 million dollars the very first year) will be considerably greater than had been anticipated.

It is clear that profound changes will occur in medicine. Perhaps they could be summarized in this way:

Medicare will accentuate the trend away from solo practice and toward group and clinic medicine. It very well could promote the exploration of new ways of organizing and delivering medical care. There will be more emphasis on the team approach and on hospital-centered medicine. It can be anticipated that Medicare will bring about acute bed shortages in some communities. Physicians thus will encounter more difficulty in procuring beds for their patients, both for the elderly and for those who are younger. As the Medicare program progresses, physicians' practices will become increasingly oriented to the hospital. Doctors will spend more time in hospitals and in nursing homes because many of their elderly patients will be there. Medicare will give a strong push in the direction of more home care services for the aged. It also will have an impact upon physicians' fees. It is only realistic to assume that physicians' fees will come under greater scrutiny than ever before.

And finally, it seems evident that Medicare and the accompanying increased demands for medical services will place greater stress and pressures upon physicians. We are extremely concerned about the modest supply of health manpower, particularly in Texas. The training of doctors, nurses, and technicians already is lagging behind the upsurge in population and the increased public demand for health services.

Physicians, Nurses Needed

In Texas, we now have some 10,700 physicians who are actively practicing their profession. We have a ratio of one active physician for every 997 persons. This is considerably less favorable than the national ratio of one doctor for every 681 persons. On the basis of continuing surveys, we frankly believe that there is a need for a greater number of physicians, particularly in some of the smaller communities and in many rural areas. The great need is for more general practitioners, and for those who desire to practice family medicine.

The need for more physicians is relatively insignificant, however, when compared with nursing. The Surgeon General's Consulting Group reports that this country will need 300,000 more professional nurses by 1970. In Texas, we presently have some 17,700 active nurses. Our state unfortunately ranks 46th in the nation in the number of nurses per 100,000 population. According to recent estimates, we need 10,500 nurses right now just to catch up with the present demand. There are estimates that Texas will need a total of

50,000 nurses by 1980. This is three times the number we have at present.

With the outlook already somewhat dismal, Medicare can only make more severe the discrepancy between supply and demand. It will place even greater pressures upon physicians and upon those dedicated to patient care. The most difficult challenge, without question, will be to maintain the present high quality of medical care.

—C. Lincoln Williston, Austin,
Executive Secretary,
Texas Medical Association.

Nondiscrimination Guidelines Listed for Nursing Homes

Guidelines to help skilled nursing homes determine if they are in compliance with the nondiscrimination provisions of the Civil Rights Act of 1964 have been released by the US Public Health Service.

Nursing homes that wish to take part in the Medicare program as extended care facilities beginning Jan. 1, 1967, must meet the quality of care standards set by the Social Security Administration and the nondiscrimination provisions of Title VI of the Civil Rights Act.

Title VI provides that no person in the United States shall be subject to discrimination under any program receiving federal financial assistance.

Nursing homes that wish to take part in Medicare or welfare programs must demonstrate that they provide services to all people without regard to race, color, or national origin.

The Office of Equal Health Opportunity will make the determinations of compliance with the assistance of other units in the Department of Health, Education, and Welfare.

The guidelines call for:

1. Open admission policies. Skilled nursing homes which are excluding patients of one race must take corrective action.

2. Nursing homes must accept patients only from persons or agencies which do not practice discrimination in referring patients.

3. Nursing homes must make assignment of rooms without asking for patient preference regarding race and not using patient transfer as a device to evade compliance.

4. All patients must receive services by employees and medical staff without regard to race, color, or national origin.

5. Nursing homes must provide opportunities for medical staff to practice in the nursing home without discrimination.

6. Recruitment and selection of candidates for training programs must be carried out without regard to race, color, or national origin.

7. All facilities and services must be operated within the home without discrimination.

8. Written policies concerning nondiscrimination must be adopted, practiced, and communicated to staff members and employees.

9. Nursing homes must notify the community and source of patient referral that the home is available to all without discrimination.

Doman-Delacato Treatment Pamphlet Available at TMA

In response to requests of physicians whose patients have asked about the Doman-Delacato Treatment of Neurologically Handicapped Children, United Cerebral Palsy Association of Texas has prepared an information pamphlet on the subject.

During the past few years, articles have appeared in newspapers and magazines throughout the country concerning methods for treatment of brain-damaged children conducted by the Institute for the Achievement of Human Potential, located in Philadelphia. An article by R. J. Doman, and others, appeared in the Sept. 17, 1960, issue of the *Journal of the American Medical Association*, and described the Institute's program and the methods utilized.

Pediatricians and other physicians have been asked by patients about the Institute which has franchised operations at San Antonio and at the University of Plano.

The pamphlet published by United Cerebral Palsy discusses the Doman-Delacato Treatment. The treatment is reviewed by the Executive Board of the American Academy of Pediatrics; the American Academy for Cerebral Palsy; Dr. William Silverman, New York pediatrician; and by Dr. (Ph.D.) Alfred Stern, director, Louisiana State University Special Education Center, New Orleans.

This pamphlet is available to physicians without charge from the Texas Medical Association Memorial Library, 1801 North Lamar Blvd., Austin 78701.

Fifty-five Texas Surgeons Elected to American College

Fifty-five Texas doctors were inducted as fellows of the American College of Surgeons on Friday, Oct. 14.

Texas physicians inducted were Dr. Elliott Mendenhall, Jr., of Abilene; Dr. Henry W. Bendel, Jr., of Arlington; Drs. Joseph M. Abell, Jr., William E. Barnes, Jr., and Jimmie H. Calhoon, all of Austin; Dr. Thomas F. Camp of Brooks Air Force Base; and Drs. Roger S. Johnson, William P. King, and Craig W. Norstrom, all of Corpus Christi.

Dallas physicians inducted were Drs. William J. Bennett, William A. Cook, James E. Dimmette, E. Frank Dunton, Raymond R. Gibbons, Leonard Graivier, Dean C. Kipp, Malcolm McNatt, R. Don Patman, Fred W. Pauling, III, Sydnie G. Smith, Clarence Gene Wheeler, and David W. Young.

Other new fellows include Dr. Richard J. Harris of El Paso; Drs. Basil A. Pruitt, John R. Simmons, and Robert G. Stanek, all of Fort Sam Houston; Drs. Henry V. Birdwell, Jr., Jackson E. Cagle, Jr., Dick G. Ellis, and John H. Smith, all of Fort Worth; Dr. Duane L. Larson of Galveston; Dr. Frank J. Little of Greenville; and Drs. Peter B. Fisher, John B. Fitzgerald, Adeeb Ed A. Hanna, Jimmy F. Howell, Raymond H. Kaufman, Conrad D. Moore, and Don C. Quast, all of Houston.

Also inducted were Drs. William A. Hendricks of Irving; Byron N. Dooley and Jack R. Robison of Lackland Air Force Base; Dan C. English of Lubbock; J. Patrick Hooker of Midland; Tom P. Williamson of Nacogdoches; Hylmar E. Karbach, Jr., of New Braunfels; Irwin Lehman of Richardson; Dr. Wendell R. Sylvester of Sherman; Dr. Sherroll A. Neill, of Tyler; and Dr. Robert H. Saxton of Waco.

New members from San Antonio include Drs. Everett M. Boyd, Vincent T. Caldarola, Efraim A. Gonzalez, Jr., Perry W. Nadig, Jaime Pankowsky, Spencer A. Rowland, and Hugh L. Wolff.

"'Tranquilizer' is not a good word"[1]

"THIS classification is psychologically too seductive, pharmacologically too unspecific, and in terms of results not infrequently untrue."[2]

What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there are differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1] This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Trifluprомazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

Prolixin is therapeutically effective without excessive sedation

When used as a 'tranquilizer' in general medical practice, in many patients Prolixin (Squibb Fluphenazine Hydrochloride) suppresses anxiety, but not normal activity. These two features are of particular importance to patients who must be able to live and work without their normal daily activities being restricted.

Because of its longer duration of action, Prolixin, in doses of as little as one to three milligrams in adults, generally taken once a day, is effective in maintaining many patients free of their symptoms of anxiety and tension.

Contraindications: Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use with caution in patients with a history of convulsive disorders. Severe reactions may occur in patients with idiosyncrasy to other centrally-acting drugs, and severe hypotension may occur in patients with special medical disorders, e.g. mitral insufficiency and pheochromocytoma.

Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

Available: 1 mg. tablets. Bottles of 50 and 500.

For full prescribing information, see package insert

References: 1. Simpson, G.M.: Postgrad. Med. *39*:557, 1966. 2. Freyhan, F.A.: Am. J. Psychiat. *115*:577, 1959. 3. Dorland's Illustrated Medical Dictionary, ed. 24, Philadelphia, W. B. Saunders Co., 1965, p. 1603. 4. Physicians' Desk Reference, 1966, Oradell, N.J., 1965, p. 310. 5. Cohen, S.: Northwest Med.: 65:197, 1966. 6. Detre, T., and Jarecki, H.: Connecticut Med. *25*:553, 1961. 7. Sainz, A.: Psychosomatics *5*:167, 1964.

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Declaration of Helsinki: Recommendations Guiding Doctors in Clinical Research

Prior to World War II, guidance for physicians in clinical research rested with the "scientific community." When reports of the uncontrolled experiments (which the Nazi Government encouraged) with human beings became available; the need for a formal set of rules became apparent.

The World Medical Association prepared the Declaration of Helsinki in 1954. This document has been approved by the House of Delegates of the American Medical Association. The Board of Councilors, through the Executive Board of the Texas Medical Association, commends the reading of this statement.

—Dick Cason, M.D., Secretary, Board of Councilors.

It is the mission of the doctor to safeguard the health of the people. His knowledge and conscience are dedicated to the fulfillment of this mission. The Declaration of Geneva of the World Medical Association binds the doctor with the words: "The health of my patient will be my first consideration" and the International Code of Medical Ethics declares that "Any act or advice which could weaken physical or mental resistance of a human being may be used only in his interest." Because it is essential that the results of laboratory experiments be applied to human beings to further scientific knowledge and to help suffering humanity, the World Medical Association has prepared the following recommendations as a guide to each doctor in clinical research. It must be stressed that the standards as drafted are only a guide to physicians all over the world. Doctors are not relieved from criminal, civil, and ethical responsibilities under the laws of their own countries.

In the field of clinical research, a fundamental distinction must be recognized between clinical research in which the aim is essentially therapeutic for a patient, and the clinical research, the essential object of which is purely scientific and without therapeutic value to the person subjected to the research.

I. Basic Principles

1. Clinical research must conform to the moral and scientific principles that justify medical research and should be based on laboratory and animal experiments or other scientifically established facts.

2. Clinical research should be conducted only by scientifically qualified persons and under the supervision of a qualified medical man.

3. Clinical research cannot legitimately be carried out unless the importance of the objective is in proportion to the inherent risk to the subject.

4. Every clinical research project should be preceded by careful assessment of inherent risks in comparison to foreseeable benefits to the subject or to others.

5. Special caution should be exercised by the doctor in performing clinical research in which the personality of the subject if liable to be altered by drugs or experimental procedure.

II. Clinical Research Combined With Professional Care

1. In the treatment of the sick person, the doctor must be free to use a new therapeutic measure, if in his judgment it offers hope of saving life, re-establishing health, or alleviating suffering. If at all possible, consistent with patient psychology, the doctor should obtain the patient's freely given consent after the patient has been given a full explanation. In case of legal incapacity, consent should also be procured from the legal guardian; in case of physical incapacity, the permission of the legal guardian replaces that of the patient.

2. The doctor can combine clinical research with professional care, the objective being the acquisition of new medical knowledge, only to the extent that clinical research is justified by its therapeutic value for the patient.

III. Non-Therapeutic Clinical Research

1. In the purely scientific application of clinical research carried out on a human being, it is the duty of the doctor to remain the protector of the life and health of that person on whom clinical research is being carried out.

2. The nature, the purpose, and the risk of clinical research must be explained to the subject by the doctor.

3a. Clinical research on a human being cannot be undertaken without his free consent after he has been informed; if he is legally incompetent, the consent of the legal guardian should be procured.

3b. The subject of clinical research should be in such a mental, physical, and legal state as to be able to exercise fully his power of choice.

3c. Consent should, as a rule, be obtained in writing. However, the responsibility for clinical research always remains with the research worker; it never falls on the subject even after consent is obtained.

4a. The investigator must respect the right of each individual to safeguard his personal integrity, especially if the subject is in a dependent relationship to the investigator.

4b. At any time during the course of clinical research the subject or his guardian should be free to withdraw permission for research to be continued. The investigator or the investigating team should discontinue the research if, in his or their judgment, it may, if continued, be harmful to the individual.

Endowed T. J. Walthall Chair Established at South Texas

An endowed chair has been established at the University of Texas South Texas Medical School, to be known as the Dr. and Mrs. T. J. Walthall Chair in Ear, Nose and Throat.

Chairman W. W. Heath of the Board of Regents of the University of Texas noted that the Walthall Chair at the South Texas Medical School is a perpetual endowment and living memorial to the foresight and generosity of the Walthall family. The endowment is a gift made by Mrs. Marjorie T. Walthall of San Antonio in memory of her husband, Dr. Thomas J. Walthall, and will provide support for teaching and research in the areas of ear, nose, and throat diseases.

Dr. T. J. Walthall, an otolaryngologist, was a native of Alabama who moved to San Antonio at an early age. He completed his education at The University of the South at Sewanee, Tenn., served in World War I, and subsequently spent over 20 years in active practice in San Antonio. He died in 1938.

In making this contribution to the development of this new unit of The University of Texas System, Mrs. Walthall is following a long tradition of support of education in the Walthall family. In his will dated July 16, 1608, William Walthall, Alderman of London, England, left in trust with the Mercers Company a sum to send two poor scholars each year to Cambridge University for a master of arts degree.

A few years ago when Mrs. Walthall visited London, she called at the Mercers Company and was shown the book containing the names of the recipients from the time the fund was created to the present. At the time of her visit, Mrs. Walthall learned that the fund was almost sufficient to send three students instead of two, and in memory of her husband she gave a sum to make this possible.

Baylor Receives NIH Grant For Clinical Research Center

A new 28-bed general clinical research center will be established under a National Institutes of Health (NIH) award to Baylor University College of Medicine in Houston.

The improvement of blood flow to the heart muscle in patients with heart disease, investigations to combat graft rejection in kidney transplants, and clinical research of respiratory virus diseases are among the problems to be investigated in the center.

The award is one of six totaling $1,258,574 given by the Department of Health, Education, and Welfare, to establish three new general clinical research centers and to provide funds to three existing centers for expansion or continued support.

Authorized by Congress in 1960, the general clinical research center program will be administered by the NIH Division of Research Facilities and Resources. The program provides a research environment in which scientists from many biomedical disciplines can conduct precise clinical studies on selected patients. The centers, essentially small research hospitals within a larger hospital complex, provide the controlled setting essential for clinical research. Each center has its own research beds, staff, laboratories, and other facilities.

In addition to the $502,134 award to Baylor, other grants to establish new clinical centers went to the University of Oregon Medical School, Portland, Ore., and the Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, N.C.

Institutions with existing general clinical research centers receiving grants were the University of Miami School of Medicine, Coral Gables, Fla.; Jefferson Medical College of Philadelphia; and the University of Michigan Medical School, Ann Arbor.

The new awards bring to 91 the number of general clinical research centers in the nation with a total complement of 1,124 beds.

April 1 Is Entry Deadline For *Texas Medicine* Award

The second annual *Texas Medicine* award for scientific writing by a Texas medical student will be presented in 1967. Deadline for submitting entries is April 1, 1967.

The award is given to a medical student for writing a scientific article. He must be enrolled in a Texas medical school at the time of preparation of the submitted article. The author of the winning article will receive $300. In addition, the winning entry will be published in *Texas Medicine*.

Articles must deal with a subject within the realm of the basic sciences or medicine and must be original and unpublished. No minimum or maximum word length is required.

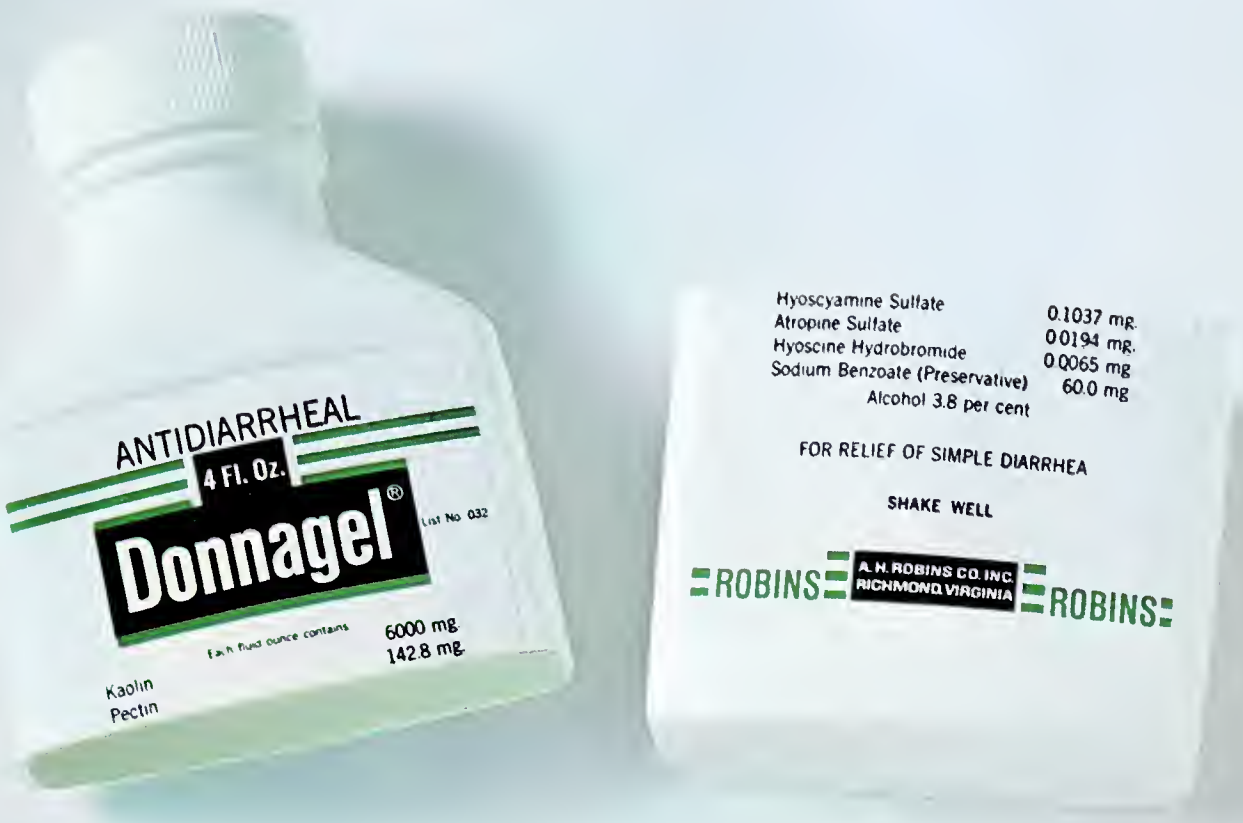
Criteria for judging will be excellence of scientific work and competence in the writing craft. Each entry will be reviewed by the Journal Advisory Committee.

All entries must be in the editorial offices of *Texas Medicine*, 1801 North Lamar Blvd., Austin 78701, by the April 1 deadline. Work on the entry may already be in progress. Brochures listing manuscript requirements for article entries will be forwarded to Texas medical schools in the near future.

The Board of Publication and the Journal Advisory Committee established the award in 1966 to encourage excellence in scientific writing.

Winner of the first annual *Texas Medicine* award was Dr. Charles F. Johnson III, now an intern at John Peter Smith Hospital in Fort Worth. He was presented the award at a meeting of the Tarrant County Medical Society in Fort Worth, Oct. 4. He received the award for the article, "The Effects of Acute Ethanol Intoxication on Hepatic Protein Synthesis," (see p. 63) written under the direction of Dr. Charles T. Ashworth, chairman of the Department of Pathology at the University of Texas Southwestern Medical School, Dallas.

Dr. Johnson was studying under a grant from Dr. F. L. Jennings, chairman of the Department of Pathology at the University of Texas Medical Branch at Galveston. He was enrolled in the Medical Branch.



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References: 1. Kramer, P., and Ingelfinger, F.J.: Med. Clin. N. Amer., *32*:1227, 1948. 2. Hock, C.W.: Clin. Med., *8*:1932, 1961. 3. Winfield, I.W.: Am. J. Gastroent., *31*:438, 1959.

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Work in Ambur, India, gave Dr. Kurt Lekisch an opportunity to observe native customs. He pauses here (second from left) with the "Holy Man on the Hill" Vellimalai Mounaguru (the silent Teacher of Vellimalai); Mr. G. Shanmugam, a local businessman; Mr. Rao, member of a nearby Ashran (an institution of learning and good will), and two native boys.

The Story of a Homeless Wanderer

Kurt Lekisch, M.D.

[Editor's Note: A devotee of international medicine, Dr. Kurt Lekisch of Midland served at the Bethesda Lutheran Hospital, Ambur, NADT, Madras State, South India, from September, 1962, through August, 1963. He was in charge of a 75-bed tuberculosis ward and of a tuberculosis outpatient department, directed a tuberculosis survey program covering the entire population of Ambur (45,000 people); and originated the first house-to-house campaign for prevention of tuberculosis with Bacillus Calmette-Guerin vaccine. Dr. Lekisch was also one of ten Texas physicians who were honored Oct. 1 by Texas Medical Association for service in civilian capacities in Vietnam. In December, 1965, following his tour of duty in Vietnam, he visited the Bethesda Hospital again and served as a short-term consultant there. The following article draws upon his observations of Indian native culture.]

MEDICINE represents powerful diplomacy, offering an almost ideal method of communicating with people of different ideologies. My participation in the field of international medicine allowed me the opportunity to inquire into and examine certain sources of India's social customs.

According to the letter of the sacred law in India, when a householder's hair turns white and he sees his sons' sons, he should become a hermit, either leaving his wife to the care of their children or else taking her to the forest with him. There, he lives in a little hut on the alms of the villagers or on food collected from the wilds. He should perform regular rites at his sacred fire and should study the Upanisads to stress union with the Divine, in order to raise his soul above earthly things.

Even today, it is not unusual for an elderly man to follow the course laid down by the ancient sages and to end his days in asceticism and become a homeless wanderer, a

sannyasin, with nothing but a staff, a begging bowl, and a few rags of clothing.

I met such a man. He is called Vellimalai Mounaguru—the silent Teacher of Vellimalai. He visited, on special occasions, Hindu festivals in Ambur, North Arcot District, South India. People called him Mouni, and he was at that time perhaps 65 years old. Fourteen years before, his own *guru* (his spiritual teacher) had enjoined upon him a vow of silence. He has never spoken since.

Little is known about his early life, but he was a student in Madras University in Madras, India, where he gained a master's degree in physics. All this is now lost and gone. Mouni knows as little of the modern science as the average villager. He has forgotten all that he learned, and he has lost the last vestige of sophistication. We know that he had a family, but who they are and where they are has also been forgotten.

Mouni is a man who has followed the path of God. He has not uttered a word for more than 14 years, but he is by no means uncommunicative. He has developed an intricate and vigorous sign language of his own. He has little or nothing to do with material comforts. He eats only when his disciples sit beside him to make sure that he does not give away his food to children or animals or any begger who might come along. His diet for an entire day is never more than two or three bananas and a few oranges, but this is enough to provide him with immense physical and mental energy. He owns nothing but the shawl and loin cloth which are his only garments. He never washes, but he doesn't smell. His hair is worn piled on the top of his head in a high coiffure in the 1,000-year-old style of Saivite saints.

Here is a man following a way of life as bizarre as any fantasy is to the Western mind. The most confirmed rationalist, the most hardened agnostic, would have difficulties resisting the man's charm, his sensitivity, and his powerful, unexampled serenity.

On top of a barren hill, in the interior of the hot plains of Madras State, Mouni lives the life of an ascetic. Indeed, many Indians feel and show the utmost respect for Mouni and those whom they consider holy.

This is withdrawal rather than service, spiritual achievement rather than abasement. Indeed, there are many difficulties in interpreting the finer points; but the main message of the basic Hindu orthodoxy is self-centered. A Hindu practices morality without any spontaneous creative relationship to other beings. The Easterner judges ethics in terms of spirit and intention; the Westerner judges ethics in terms of action and consequences. Dr. Albert Schweitzer, who searched for ethics in practice, pointed out: "Love and pity for one's fellow are still beyond the horizon of Hindu ethics."

I owe inexpressible gratitude to the "man in the street" of South India for the many insights explaining the heart and mind of his homeland. India is molded by four millennia of history and by powerful sources of modern environment. In spite of the contrasts of East and West, in the sphere of institutions and doctrines one can find a realm of values common to all humanity. People, then, are the focus of this journey of discovery. A concern for international medicine has left a permanent imprint on me.

Mental Health Directory Available Upon Request

The Texas Department of Mental Health and Mental Retardation is distributing without charge a current *Directory of Mental Health Services in Texas* for 1966-67.

The directory provides a comprehensive listing of professional services and facilities in Texas to which professional practitioners and agencies may have occasion to refer troubled persons. The directory was published for seven years by the Division of Mental Health in the Texas State Department of Health. The Division has now been transferred to the Texas Department of Mental Health and Mental Retardation.

Requests for complimentary copies should be made to Mr. Charles F. Mitchell, Acting Director, Division of Community Services Development, Texas Department of Mental Health and Mental Retardation, Box S, Capitol Station, Austin. If more than one copy is desired, a request letter should state for whom and for what purpose the copies are needed.

State Health Department Seeking Leprosy Control

The Texas State Department of Health began administering on Oct. 1 a program for the control of leprosy in Texas.

The program is being financed by a three-year US Public Health Service grant of $267,734. Dr. M. S. Dickerson, state epidemiologist, is serving as program director.

Leprosy is endemic in Texas. Cases are scattered throughout most areas of the state, but the greatest number of cases are concentrated in counties close to the Gulf of Mexico and the Rio Grande Valley.

A survey conducted during the past six years yielded two conclusions regarding the leprosy problem in Texas: (1) leprosy is much more prevalent in Texas than was previously known, and (2) leprosy is on the increase in Texas. Epidemiological methods and public education will be used in seeking to control and eradicate the disease.

UT Medical Branch Receives Public Health Service Grant

A US Public Health Service grant to establish a program project for research in hematology and rheology, amounting to more than $1.2 million over a seven-year period, has been awarded to the University of Texas Medical Branch.

The grant is the first such program project awarded to UTMB, said Dr. Spencer G. Thompson, assistant director and coordinator of sponsored research.

Named to carry out the investigative work were Dr. M. Mason Guest, professor and chairman of physiology, as program director, and Dr. W. C. Levin, professor of internal medicine who directs the school's Hematology Research Laboratory, as co-director.

Major objectives of this research involve clarification of the role of individual components of blood in determining its rheologic, immunologic and transport characteristics, and the determination of means by which blood flow, immunologic characteristics, and transport efficiency can be normalized when blood has been altered by disease or trauma.

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Medicine and the Law

Legal Provisions Concerning Autopsies Outlined in New Texas Criminal Code

It should come as no surprise to any well informed citizen of Texas that a new Code of Criminal Procedure has gone into effect in recent months. While lawyers, legislators, and law enforcement officials have alternately praised and condemned the new code, it has gone largely unnoticed that this new codification of the law also affects the members of the medical profession. Of prime interest in this regard and the subject of these comments is the code's provisions regarding authorization for autopsies as these provisions apply to physicians.

One of the most important things to remember is that in instances where the justice of the peace is required or authorized to hold an inquest, the attending physician should not perform an autopsy. This is true even when the consent has been given by those persons who may give legal consent to the physician for the autopsy as provided in Article 49.07, C. C. P.

This rule is necessary since the law requires that an inquest be held to determine the cause of death under certain circumstances. In many cases the primary evidence at such a hearing will be that given by the person who has, on the court's order, performed an autopsy. The law clearly gives the court the "priority" insofar as determining whether or not an autopsy will be necessary. If the court does not order an autopsy, then the physician may proceed to perform the autopsy if he has secured the consent of the surviving spouse. If no spouse survives, any child may give legal consent unless the child be a minor, in which case the authorization must come from the child's legal guardian or from the court having jurisdiction of the person of the minor child. If neither spouse nor child survive, then consent to an autopsy under Article 49.05 must come from the father or mother of the deceased, or from the next of kin. In the absence of any of the foregoing, consent may be granted by "any natural person assuming custody of and responsibility for burial of the body."

While the statutory law does not require it, the physician should secure the consent for autopsy by a written instrument which serves to hold the physician harmless for any legal claims arising out of the autopsy.

When does the justice of peace have the first claim on the body for purposes of an autopsy? Article 49.01 provides that the justice of the peace has a duty to hold an inquest whenever:

1. The person dies in prison or jail.
2. The person is killed or dies an unnatural death in absence of "one or more good witnesses."
3. The circumstances of death are unknown.
4. There is a suspicion that the person died of "unlawful means."
5. The person has committed suicide or suicide is suspected.
6. The person dies without having been attended by a practicing physician and the local health officer or registrar of vital statistics does not know the cause of death.
7. The attending physician is not certain of the cause of death and is unable to certify as to the cause, in which case the physician or hospital administrator should notify the justice of the peace of the death and request an inquest.

Thus, in the circumstances previously summarized, the decision on whether or not to order an autopsy is the responsibility of the justice of the peace holding the inquest. If he fails to order an autopsy and concludes the inquest, the physician is then free to proceed with a voluntary autopsy having once secured the consent from the proper party.

In rare instances, the physician or hospital administrator may attempt to notify the justice of the peace in his precinct of a death under circumstances which indicate the necessity for an inquest and may be unable to locate him; or the justice of the peace may refuse to hold an inquest. In such cases, the inquest may be conducted by the nearest available justice of the peace.

In those instances where the physician is able to certify with certainty as to the cause of death and no other circumstances surround the death which would make an inquest mandatory, then the physician may proceed to perform an autopsy immediately upon securing the consent from those persons authorized to grant it.

In other provisions of this law of interest to physicians, the justice of the peace may order any "duly licensed and practicing physician who is trained in pathology to make an autopsy "where the county health officer is unavailable or in those counties without a health officer." When ordered to perform an autopsy, the physician shall receive a fee of "not more than $100," the exact amount of which the commissioners court shall determine "after ascertaining the amount and nature of the work performed in making such autopsy." Also, the court is free to order laboratory tests or investigations which may be deemed necessary and to call in additional medical experts to aid in the investigation of the cause of death.

The new Code of Criminal Procedure has made no drastic change in the regulations relating to autopsies, yet it is an area of the law which may sooner or later affect almost any physician. Every physician would be well served to familiarize himself with the provisions on autopsies in the new code.

—Philip R. Overton, LL.B., and Sam V. Stone, Jr., LL. B., Austin.

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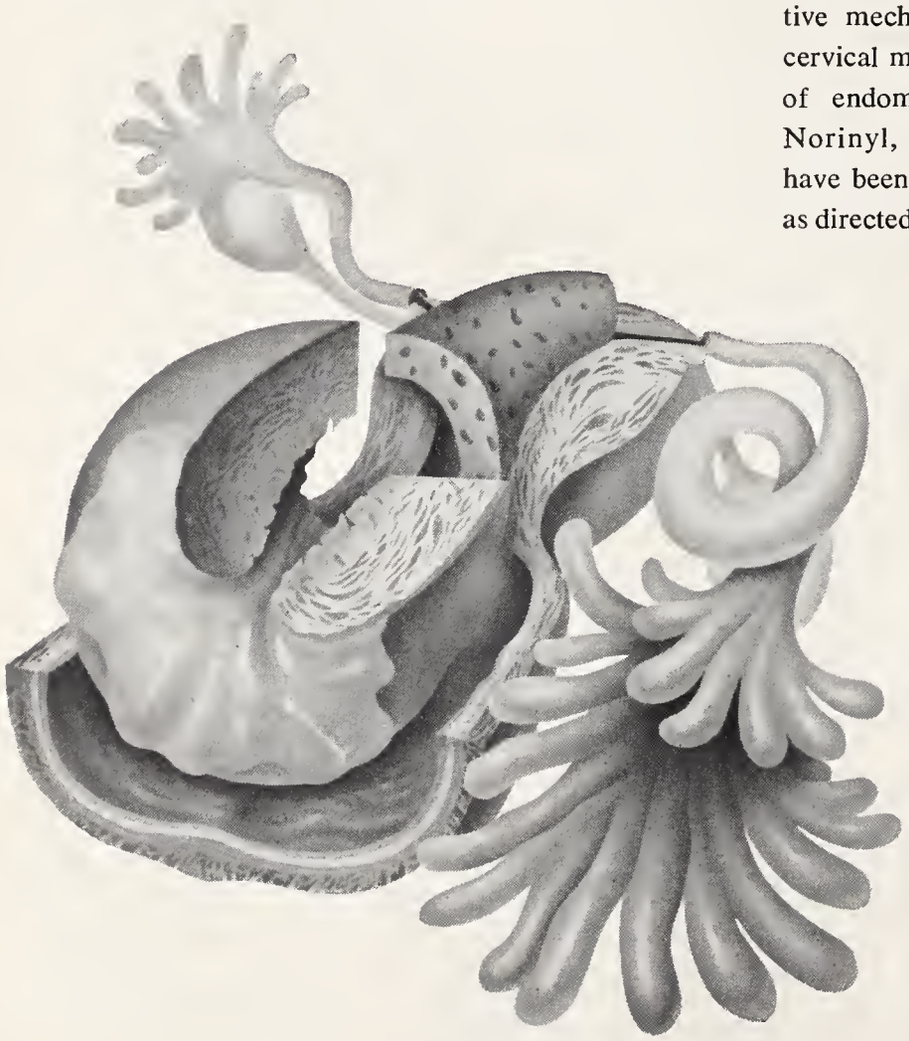

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Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb. 29) 1964. 2. Bryans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

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Scientific Exhibits, Refresher Courses Planned for Centennial Annual Session

Plans continue to progress toward the finalization of the program for Texas Medical Association's Centennial Annual Session to be held in Dallas May 4-7, 1967.

The 1967 Annual Session will be the celebration of the 100th TMA Annual Session. A commemorative painting is being prepared for the occasion, and other special features will call attention to the anniversary throughout the centennial year. Outstanding speakers for the centennial meeting will include Dr. Wernher Von Braun, director of the George C. Marshall Space Flight Center in Huntsville, Ala., and Dr. Charles Hudson, President of the American Medical Association.

In addition to the obtaining of 31 out-of-state speakers for the Session meetings, plans are well underway for scientific exhibits and refresher courses that will also be features of the Annual Session.

Scientific Exhibits

Information and application forms are now available for doctors wishing to display scientific exhibits at the Texas Medical Association Centennial Annual Session.

Application forms may be obtained by writing Mr. Dale Werner, Exhibit Manager, Texas Medical Association, 1801 North Lamar Blvd., Austin 78701.

Deadline for returning these applications is Jan. 14, 1967. All exhibit applications are reviewed for approval in January by the Committee on Scientific Exhibits. Members of TMA receive priority in space considerations over non-members and out-of-state physicians.

There is no charge for scientific exhibit space. Booths with either draped or plywood walls are constructed for exhibits according to the applicant's choice. The back wall length of the booth is determined by the space requirements of the individual exhibit. Any special requirements in booth construction are made at the exhibitor's expense. Costs involved in transportation, storage, and special electrical requirements must also be paid by the exhibitor.

Awards are given for the most outstanding exhibits. Exhibits are classified in four groups. Three awards are given in each group. In addition to these awards, Mead Johnson Laboratories in cooperation with TMA will present an Aesculapius Award for the outstanding exhibit presented by a Texas physician. The Aesculapius Award consists of an appropriate certificate and $200.

Group Classifications

The four group classifications are (1) exhibit without grant or assistance; (2) exhibit with grant or assistance from university, hospital, government agency, or nonprofit organizations; (3) exhibit with grant or assistance from a pharmaceutical or other commercial enterprise; and (4) exhibits primarily of educational-promotional nature which bring a message of interest and importance to the attention of the profession.

Exhibits are judged by an anonymous group of physicians from different specialties. Exhibits are judged for clarity, effectiveness, personal presentation, contribution to basic knowledge, practical value, originality, newness, etc.

Mr. Werner stressed the value of the scientific exhibits. He reiterated recent points made by the director of the AMA's Scientific Exhibit Section, "The scientific exhibits presented by the physicians are an important part of the Annual Session. The combined advantages of the spoken word, written word, and visual demonstration are all brought together into a unified and retainable form. The teaching value of a well presented scientific exhibit is probably higher than any other known media."

A book on planning, preparing, and showing a scientific exhibit can be obtained by writing the TMA Memorial Library. This book provides many helpful aids in preparation of a successful scientific exhibit.

Any further questions regarding exhibits can be answered by contacting members of the Committee on Scientific Exhibits or by getting in touch with Mr. Werner at the TMA Headquarters Building.

Committee members are Drs. Jasper H. Arnold of Houston, John A. Welty of Harlingen, Charles C. Jones, Jr., of Kerrville, J. Frank Beall of Nacogdoches, Walter Long of Austin, Nathan Cedars of Stephenville, W. L. Carrington of Mexia, Don C. Spencer of Texarkana, P. W. Voltz, Jr., of San Antonio, and John W. Stack of Mesquite (ex officio).

Refresher Courses

The schedule of refresher courses, for the most part, for the Centennial Annual Session has been completed. The program will include some 15 courses, each consisting of 45 minutes of lecture plus 30 minutes of questions and answers. Eleven refresher courses have been scheduled. Four more are still to be included on the program.

The courses will be presented by out-of-state medical leaders. They are primarily designed for the benefit of physicians in general practice, but they are also of value to specialists.

Courses will take place from

8:30 to 9:45 a.m., Friday and Saturday, May 5 and 6, in Dallas Memorial Auditorium. Each course will be completed in a single day.

Refresher courses and instructors scheduled to date include "Surgical Treatment of Diverticulitis," Dr. Oliver H. Beahrs of Rochester, Minn.; "The Treatment of Addiction," Dr. R. G. Bell of Ontario, Canada; "The Clinical Uses of Radioisotope Scanning," Dr. James H. Christie of Cleveland, Ohio; "The Surgical Treatment of Duodenal Ulcer," Dr. Robert J. Coffey of Washington, D. C.; and "Hemorrhoids," Dr. James A. Ferguson of Grand Rapids, Mich.

Additional courses scheduled are "Chronic Pelvic Pain—Its Diagnostic Significance and the Neurologic Pathways Involved," Dr. Zeph J. R. Hollenbeck of Columbus, Ohio; "Preventive Psychiatry in the First Years of Life," Dr. Reginald S. Lourie of Washington, D. C.; "Pain," Dr. Joseph H. Ransohoff of New York City; "Diagnosis and Management of Bursitis," Dr. Donald L. Rose of Kansas City, Kan.; "Management of Hearing Loss," Dr. William H. Saunders of Columbus, Ohio; and "Current Concepts of the Diagnosis and Treatment of Ligamentous Injuries of the Knee," Dr. Donald B. Slocum of Eugene, Ore.

More Active Nurses Is Goal Of Health Service Project

Texas Medical Association and the Texas State Department of Health are cooperating in an effort to increase the number of active nurses. Other professional associations are also cooperating in the effort.

The program is taking place as a special project under a US Public Health Service grant. The purpose of the project is to acquaint unemployed nurses in a given area by means of various media that an orientation course will be developed through a special grant. The orientation course will last approximately one month and will help nurses keep up to date on current practices and on job opportunities.

A list of inactive nurses in the Austin, San Antonio, and Beaumont-Orange-Port Arthur areas is being compiled. If time permits before May 31, 1967, other areas will also be selected. Local facilities will be encouraged to set up refresher courses, and assistance will be offered in securing training grants to defray expenses.

A study will be made to identify skilled nursing job needs within the nursing homes and home health programs in the selected areas. Those taking the refresher courses will be informed of job opportunities.

Further information is available from local nursing associations.

Hospital Association Receives Public Health Service Grant

A grant of $116,290 to the Texas Hospital Association has been awarded by the Public Health Service, Department of Health, Education and Welfare to finance continued research into "Planning of Community Health Resources for Disasters."

This is the second of a three-year grant received from the Research Grants Branch, Division of Community Health Services. The first grant was for $174,336. The Texas Hospital Association was selected as the grantee institution following development of a pilot study on emergency medical support by the Baylor-Medical Field Service School Hospital Administration Program and the hospital association.

The project, the first of its type in the United States, began Sept. 1, 1965, under the auspices of THA's San Antonio Health Resources Planning Unit with offices at the Chapman Graduate Center of Trinity University.

The objective of the program is to determine and maximize the optimum use of available health resources in case of disaster of any type, ranging from those caused by nature to those which could result from a nuclear attack. Following collection of extensive basic health data for computer exercises, a contract was awarded to Lockheed Missiles and Space Company of Sunnyvale, Calif., to provide a computerized disaster casualty management system.

The computerized exercise, scheduled to be operational on a training basis next March with completion in June, 1967, may be applied to any community for use in studying health resources. With a minimum of effort, the simulator can be programmed for use nationally.

Pamphlets

The facts about epilepsy, its treatment, and studies of the records of epileptics in such activities as working and driving, are summarized in Public Affairs Pamphlet No. 387, entitled "Epilepsy—Today's Encouraging Outlook." The pamphlet is available for 25 cents from the Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.

This pamphlet, which can be useful not only to epileptics but also to their families, teachers, employers, and to the general public, explains the nature and the causes of the three kinds of epileptic seizures and outlines what to do—and what not to do—for someone who is experiencing a seizure.

Three new Public Health Service pamphlets, based on nationwide studies and workshops, are (1) "Report: Surgeon General's Workshop on Prevention of Disability from Arthritis," (2) "Highlights of the Report," and (3) "Arthritis Source Book."

The 104-page "Report: Surgeon General's Workshop on Prevention of Disability from Arthritis" discusses the problem of arthritis disability, conference recommendations, and background papers presented at the May, 1965, meeting sponsored by the Diabetes and Arthritis Program of the Division of Chronic Diseases. The pamphlet is available from the Superintendent of Documents, US Government Printing Office, Washington, D.C. 20402, for 75 cents. The accompanying booklet, "Highlights of the Report," contains conclusions and recommendations of the seven expert workshop groups. This 17-page pamphlet is available for 20 cents also from the Superintendent of Documents. "Arthritis Source Book" contains a wide range of data on the many aspects of the arthritis problem, such as its prevalence, disability, longevity, and cost. This 73-page pamphlet may be ordered for 50 cents from the Superintendent of Documents.

Christmas Seal Symposium To Take Place in Houston

The Houston-Harris County Tuberculosis Association will present the third annual Tuberculosis Christmas Seal Symposium on Respiratory Diseases Dec. 9 at the Warwick Hotel, Houston.

Nine physicians in the field of respiratory diseases from over the United States will speak on the program.

Participants include Dr. Hurley L. Motley, University of Southern California, Los Angeles; Dr. David M. Spain, Brookdale Hospital Center, Brooklyn; Dr. Gunyon M. Harrison, Texas Children's Hospital and Baylor University College of Medicine, Houston; and Dr. Thomas W. Lester, Jr., National Jewish Hospital at Denver, Denver, Colo.

Other speakers are Dr. Charles A. Berry, Manned Spacecraft Center, Houston; Dr. Herbert L. Fred, Baylor University College of Medicine, Houston; Robert G. Loudon, M.B., University of Texas Southwestern Medical School, Dallas; Dr. Hylan A. Bickerman, College of Physicians and Surgeons, Columbia University, New York City; and Dr. James J. W. Raleigh, Baylor University College of Medicine, Houston.

Dr. Travis Smith Elected To AAMA Advisory Post

Dr. Travis Smith of Abilene was unanimously elected to the Advisory Board of the American Association of Medical Assistants at the Association's annual meeting Oct. 19-23 in St. Louis.

Mrs. Juanita Ainsworth of Dallas was elected as vice-speaker of the House of Delegates. Mrs. Ainsworth is a past treasurer of AAMA, was the first president of Texas Medical Assistants Association, and is a past president of the Dallas County Medical Assistants Society.

Texas delegates to the convention were Mrs. Gwen Martin of Amarillo, Mrs. Jan Mustonen of Dallas, Miss Josephine Estrada of San Antonio, Mrs. Phyllis Goodrich of El Paso. Alternates were Mrs. Bobbie Smith of Port Arthur, Mrs. Thelma Dahm of Fort Worth, Mrs. Jan Coffman of San Angelo, and Mrs. Yvonne Pace of Houston.

Mrs. Martin is the current president of TMAA; Mrs. Mustonen, the president-elect; Miss Estrada, past president; Mrs. Goodrich, treasurer; and Mrs. Smith, secretary.

Mrs. Judy Coleman of Dallas, past president of AAMA, Mrs. Mildred Crawford of San Antonio, AAMA Trustee, and other Texas members also attended the meeting.

Postgraduate Courses

Symposium on Adolescence, New Orleans, Dec. 1-3.—This three-day course will be sponsored by the Division of Psychiatry and Community Mental Health Center of Touro Infirmary and will be supported by a National Institute of Mental Health grant. It is approved for 15 hours credit by the American Academy of General Practice.

Topics to be discussed include The Physician's Role in Mental Retardation, Parents of Problem Children, Handling of Adolescents by General Practitioners, Sexual Morality—A College Dilemma, Drugs in the Treatment of Children and Adolescents, Learning Problems of the Adolescent, Adolescence and Social Mores, Talking About Sex with Adolescents, and Religious Conflicts.

Registration fee is $20. Check should be made payable to Touro Infirmary. For further information write Dr. Gene L. Usdin, Director of Psychiatric Services, Touro Infirmary, 1400 Foucher Street, New Orleans 70115.

Infectious Diseases, Pittsburgh, Dec. 14-17.—The American College of Physicians (ACP) will hold a course at the University of Pittsburgh School of Medicine and Medical Center. Dealing primarily with clinical problems related to adult infections, the course will incorporate information in the basic sciences that has a direct bearing on the management of human infections.

Tuition fee for ACP members is $60; for non-members, $100. Only ACP members will be accepted in courses immediately upon receipt of application. The names of non-members will be placed on waiting lists in the order in which they are received. Three weeks after the release of the course outline non-members will be notified whether sufficient room remains for them in the course.

For further information write Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine St., Philadelphia 19104.

Basic Mechanisms of Renal Disease, New York City, Jan. 16-20, 1967.—Another course to be presented by the American College of Physicians will take place at Cornell University Medical Center and New York Hospital.

The purpose of this course is to present new and significant advances in renology with emphasis on the basic mechanisms of renal disease. The major areas to be discussed include physiology, light and electron microscopy, histochemistry, experimental nephritis, and the diagnosis and treatment of renal disease.

Information concerning tuition fees and registration procedures is the same as in *Infectious Diseases* preceding.

General Practice Review, Denver, Jan. 15-21, 1967.—A thirteenth annual postgraduate course designed especially for the general practitioner will be sponsored by the University of Colorado School of Medicine.

The course will last a full week with each day devoted to an important area of practice. An applicant may register for the entire course or for the lectures on any selected days.

The entire course will include the following topics: sexual problems in clinical practice, medicine, pediatrics, surgery, trauma, obstetrics and gynecology, and dermatology.

For further information and detailed program write to the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver, Colo., 80220.

Vineristine Symposium, Jan. 27, 1967, Memphis, Tenn. — Current concepts of biological, pharmacological and biochemical action, and comprehensive summaries of therapeutic results obtained in treating solid tumors and the leukemias, will be presented at this symposium sponsored by the Southwest Cancer Chemotherapy Study Group. The course will take place at St. Jude Children's Research Hospital in Memphis.

There is no registration fee. Further information can be obtained by writing to Headquarters,

Southwest Cancer Chemotherapy Study Group, 6723 Bertner Drive, Houston 77025.

Microbiology, Atlanta, Ga.—The Laboratory Branch of the Communicable Disease Center of the US Public Health Center is presenting a series of laboratory refresher courses in special subdivisions of microbiology.

Courses with registration closing dates through December are Serologic Methods in Microbiology and Laboratory Methods in Medical Mycobacteriology, both to take place on Jan. 30-Feb. 10, 1967, and both with closing date on Dec. 19.

Information and application forms are available from the Training Office, Laboratory Branch, Communicable Disease Center, Atlanta, Ga. 30333.

Personals

Dr. Emerson Emory, Dallas internist, and **Dr. Robert Franken**, San Angelo surgeon, have recently returned from a two-month tour of duty as civilian physicians in South Vietnam. These doctors participated in the American Medical Association Volunteer Physicians for Viet Nam program.

Dr. Frederick C. Lowry, Austin orthopedic surgeon, and **Dr. Otto Heins**, Raymondville general practitioner, departed in October for similar two-month assignments in South Vietnam. This is the second such tour of duty for Dr. Heins who was honored, along with Dr. Emory and Dr. Franken, on Oct. 1 by Texas Medical Association for service in Vietnam. Dr. Heins' previous service was from Dec. 17, 1965, to Feb. 14, 1966.

Dr. Milford O. Rouse of Dallas President-elect of the American Medical Association, served as a panel moderator at the annual meeting of the Association of Life Insurance Medical Directors of America on Oct. 20 in New York. Dr. Rouse, who is also medical director of Rio Grande National Life Insurance Company, moderated the panel on public relations.

Dr. Raymond D. Pruitt has been named vice-president for medical affairs and chief executive officer of Baylor University College of Medicine in Houston. Dr. Pruitt has been professor and chairman of the Department of Medicine since 1959. Before coming to Baylor he was professor of medicine and chief of medicine for medical education at the Mayo Foundation graduate school at the University of Minnesota. He was associate director of the Mayo Foundation from 1954 to 1957.

Dr. Silas W. Grant of Hillsboro took over as president of the Texas Academy of General Practice September 13. Other new officers include **Dr. Durwood Neal** of Fort Worth, president-elect; **Dr. Harry A. Tubbs** of Post, vice-president; and **Dr. B. B. Westbrook, Jr.**, of Beaumont, treasurer. **Dr. Guy T. Denton, Jr.**, of Dallas retired as president.

Before leaving for two-months of civilian medical service in Vietnam, Dr. Otto Heins of Raymondville and Dr. Frederick C. Lowry of Austin receive briefing from Dr. Charles Moseley, director of the American Medical Association's Department of Governmental Medical Programs. This will be the second tour of duty in Vietnam for Dr. Heins, who served from December, 1965, to February, 1966.

New Books In TMA Library

Agranowitz, A., and McKeown, M. R.: *Aphasia Handbook*, Springfield, Ill., Charles C Thomas, Publisher, 1964.

Albert, D. M., and Scheie, H. G.: *A History of Ophthalmology at the University of Pennsylvania*, Springfield, Ill., Charles C Thomas, Publisher, 1965.

Armour, R.: *It All Started With Hippocrates*, New York, McGraw-Hill Book Co., 1966.

Bates, J. E., and Zawadzki, E. S.: *Criminal Abortion*, Springfield, Ill., Charles C Thomas, Publisher, 1964.

Boba, A.: *Death in the Operating Room*, Springfield, Ill., Charles C Thomas, Publisher, 1965.

Brooks, S.: *Civil War Medicine*, Springfield, Ill., Charles C Thomas, Publisher, 1966.

Brown, R. L.: *Pesticides in Clinical Practice*, Springfield, Ill., Charles C Thomas, Publisher, 1966.

Bryan, W. J., Jr.: *Religious Aspects of Hypnosis*, Springfield, Ill., Charles C Thomas, Publisher, 1962.

Calman, C. H.: *Atlas of Hernia Repair*, St. Louis, The C. V. Mosby Co., 1966.

Carter, C. H.: *Handbook of Mental Retardation Syndromes*, Springfield, Ill., Charles C Thomas, Publisher, 1966.

Cogan, D. G.: *Neurology of the Visual System*, Springfield, Ill., Charles C Thomas, Publisher, 1966.

Daves, M. L., and Loechel, W. E.: *The Interpretation of Tomograms of the Head*, Springfield, Ill., Charles C Thomas, Publisher, 1962.

Fishbein, M.: *Modern Home Remedies and How To Use Them*, New York, Doubleday and Co., Inc., 1966.

Goodman, F., and Schoediner, G. R., III: *Questions and Answers in Orthopaedics*, St. Louis, The C. V. Mosby Co., 1966.

Jablonska, S.: *Scleroderma and Pseudoscleroderma*, Warsaw,

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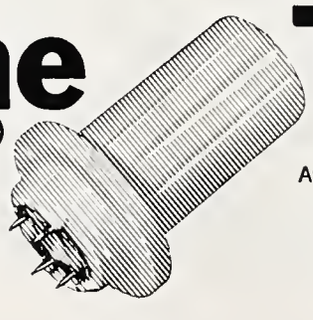

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Poland, Polish Medical Publishers, 1963.

Parke, Davis & Co.: *Great Moments in Medicine*, Detroit, Northwood Institute Press, 1965.

Parke, Davis & Co.: *Great Moments in Pharmacy*, Detroit, Northwood Institute Press, 1965.

Solomon, P.: *Psychiatric Drugs*, New York, Grune & Stratton, Inc., 1966.

Spedick, D. H.: *Chronic and Constrictive Pericarditis*, New York Grune & Stratton, Inc., 1964.

Weller, C., and Boylan, B. R.: *The New Way To Live With Diabetes*, New York, Doubleday and Co., Inc., 1966.

Book Notes

Community Psychiatry and the Clergyman. Granger E. Westberg and Edgar Draper, 110 pp., Springfield, Ill., Charles C Thomas, Publisher, 1966. $5.75.

THIS BOOK presents the efforts of religion and psychiatry to come together in a meaningful and workable relationship within the framework of community mental health. It is primarily written to point out the role of the minister in the community programs. Concepts of community preventive psychiatry are discussed with clarity, but to a limited degree.

The major portion of the work is devoted to a description of two experimental projects designed to develop the field of pastoral counseling. Case material from the projects is presented as a teaching medium. This material serves the purpose of educating both the minister and the physician to the benefits and pitfalls of having the minister in the front lines of community psychiatry.

For those interested in the area of community mental health, the book will provide several thought provoking problems. Problems of communication common to interdisciplinary projects, attrition rate of interested and involved people, and the potential of pastoral counseling are focused upon. Reflections from both the involved disciplines are encompassed in the discussions.

The physician involved in community mental health planning or work will find the book interesting. However, its greatest value will probably be to the clergyman involved in pastoral counseling.

—TRACY R. GORDY, M.D., Austin.

Diseases of the Heart. Charles K. Friedberg, ed. 3, 1787 pp., illus., Philadelphia, W. B. Saunders Co., 1966. $26.

THIS THIRD edition of *Diseases of the Heart* represents a great amount of work, effort, and thought on the part of the author in bringing the book up to date in all the fields of cardiology.

A vast amount of writing, research, new developments, and new drugs has been brought forth during the past ten years in the field of cardiology, probably more than during the preceding 50 to 100 years.

Dr. Friedberg has added 700 pages to the new edition in order to avoid sacrificing any of the successful qualities of the old edition and to add the latest material which brings it up to date. The chapters which are related to physical diagnosis, graphic methods, cardiac therapy, and angiography are particularly well presented.

—THOMAS R. JONES, M.D., Jasper.

New Film In TMA Library

Roentgen Anatomy of the Normal Alimentary Canal. Sound, black and white, 30 minutes, 1966, American College of Radiology, Dr. Theodore A. Tristan, Editor.

THE FILM consists of segments of cinefluorographic studies with narration and animation. It depicts the course of the entire alimentary system from swallowing to passage through the lower colon.

Recent Deaths

DR. J. R. SHAEFFER

Dr. Joseph R. Shaeffer, 63, director of medical education and medical director of Santa Rosa Medical Center in San Antonio, died of cancer at Brooke Army Hospital on Aug. 11.

Despite incurable cancer, Dr. Shaeffer was active almost until the end of his life warning others about cancer and instructing those who have it how to accept and fight the disease. A week before his death he was absent from Brooke Army Hospital for a few hours to accept in Austin the Annual National Division Award of the American Cancer Society for his outstanding contributions to cancer control.

Dr. Shaeffer was born in Ballston Spa, N.Y., on Oct. 15, 1903. He received his bachelor of arts degree from Cornell University in 1925. In 1928 he was graduated from the Cornell University Medical College. Following internship at the Hospital for Special Surgery, and residency at Bellevue General Hospital, both in New York City, he began practice in New York City and was a member of the faculty in surgery at the New York University School of Medicine. He was certified by the American Board of Surgery in 1942.

He entered the US Army in 1942, serving as chairman of the department of surgery at several major Army hospitals before retiring in January, 1959. He retired with the rank of colonel.

In addition to being medical director of Santa Rosa Medical Center, he was an assistant dean of the University of Texas South Texas Medical School and a member of the faculty of Baylor University Postgraduate School of Medicine. He also was a recurring consultant on disaster medicine and planning to the American Medical Association, the American Hospital Association, the American College

of Surgeons, the Department of Health, Education and Welfare, the US Public Health Service, and the US Office of Civil Defense.

Known as "Mr. Disaster Medicine," he edited the official disaster medicine manual of the North Atlantic Treaty Organization. During the past 15 years, Dr. Shaeffer had traveled an estimated 750,000 miles giving lectures to physicians, armed forces personnel of several nations, health officials, community leaders and educators. During Hurricane Carla, Dr. Shaeffer was responsible for the care of 20,000 residents evacuated from the Texas gulf coast.

For his work in the field of cancer detection and research, and civil defense disaster planning, the San Antonio Council of Presidents named Dr. Shaeffer the "Outstanding Citizen of San Antonio" for 1961. In 1964, he was named "Texas Volunteer of the Year" by the Texas Division of the American Cancer Society.

Active in the American Cancer Society, Dr. Shaeffer served on the national board and served two terms as president of the Bexar County unit.

He was a member of Bexar County Medical Society, Texas Medical Association, American Medical Association, American Association for the Surgery of Trauma, Association of Military Surgeons of the United States, Texas Surgical Society, and International Medical Assembly of Southwest Texas. He was a past president of the Second District, Texas Chapter of the American College of Surgeons, and a member of the board of directors and medical director for disaster of the Bexar County Chapter of the American Red Cross. He was chairman of the Subcommittee on Disaster Surgery of the AMA, a member of Postgraduate Surgical Training Committee of the AMA, a member of the Trauma Committee of the American College of Surgeons, and a member of the Committee on Disaster Medical Care of the Texas Medical Association.

He attended St. David's Episcopal Church. He was a member of the Fort Sam Houston Officers' Club and was a Mason.

On Sept. 9, 1928, he married Frances H. Burns in Charles Town, W. Va. She survives as do two sons, Dr. Joseph R. Shaeffer, III, of Houston, and Ronald E. Shaeffer of Boston, Mass., and two grandchildren.

DR. SHAEFFER

DR. R. B. PADDOCK

Dr. Richard Bolles Paddock died Aug. 14, 1966. He was 51. He was born March 31, 1915, in New York City, the son of Richard B. and Anne Loughran Cunningham Paddock. His preliminary education was obtained at Canterbury School in New Milford, Conn. He received both a bachelor and a master of arts degree from Stanford University in Palo Alto, Calif. He was graduated from Jefferson Medical College of Philadelphia in 1957. After interning at Methodist Episcopal Hospital in Philadelphia, he served a residency in anesthesiology at the Hospital of the University of Pennsylvania. He was chief of anesthesiology at the Veterans Administration Hospital in San Francisco before moving to Houston in 1965.

He served in the US Army during World War II.

A diplomate of the American Board of Anesthesiology, he belonged to the Harris County Medical Society, Texas Medical Association, American Medical Association, American Society of Anesthesiologists, and the New York Academy of Sciences.

Dr. Paddock was married to the former Rita Caroline Ludwig of Little Falls, N.J. Mrs. Paddock survives as do a son, Stephen Paddock, also of Houston; and a daughter, Miss Margot Paddock of New York City.

DR. PADDOCK

DR. H. QUIRARTE

Dr. Humberto Quirarte, 44, was killed Aug. 26 in a three-car collision in El Paso. He had practiced there since 1954.

Born in Juarez, Chihuahua, Mexico, on October 1, 1921, he attended Texas Western College in El Paso, and received his bachelor's degree from Tulane University in 1942. He received his medical degree from Tulane University School of Medicine in 1945. In 1945 and 1946 he interned at Charity Hospital of Louisiana at New Orleans and was enrolled in the University of Pennsylvania Graduate School of Medicine in 1946-47. He was a precepté of Dr. Eduardo Castro at the National University of Mexico school of medicine 1947-1950. He had practiced in Juarez until 1954 before moving to El Paso.

He belonged to the El Paso County Medical Society, Texas Medical Association, and the American Medical Association. He also was a member of the Mexican Urological Association, Mexican Association for the Study of Sterility, the International Fertility Association, the International Urological Association, the Texas Urological Society, and the South Central Section of the American Urological Association.

Dr. Quirarte is survived by his wife, Matilde Quirarte, three daughters, Anna, Gabriela, and Claudia Quirarte, four sons, Estaban, Humberto, Adrian, and Jaime Quirarte, all of El Paso; his mother, Mrs. Antonina Quirarte of Juarez; and one sister, Mrs. Yolanda Sanchez of Juarez.

DR. C. D. JOYCE, JR

A Palestine physician, Dr. C. D. Joyce, Jr., 55, died Aug. 5, in Hermann Hospital in Houston after a long illness.

The son of Claude Dalton and Minnie Haynie Joyce, he was born in Palestine on Jan. 17, 1911. He attended schools there, Sam Houston State College, and the University of Texas. In 1937, he was graduated from Baylor University College of Medicine, then in Dallas. He remained in Dallas to serve an internship and residency at Parkland Memorial Hospital. He returned to Palestine in 1939 where he was house surgeon with the

DR. JOYCE

DR. KNIGHT

DR. CASTNER

Missouri Pacific Lines Employees Hospital from 1939 until 1946, when he began private practice.

A founder-member of the American Board of Abdominal Surgery, he was a fellow of the American College of Surgeons, International College of Surgeons, and International Academy of Proctology; and a diplomate of the International Board of Surgeons and the International Board of Abdominal Surgery. He was a past president of the Anderson-Leon Counties Medical Society, and a member of the Texas Medical Association and the American Medical Association.

Dr. Joyce married the former Dorothy Mae Wright on July 22, 1928, in Palestine. She died on Sept. 5, 1958. On Jan. 15, 1964, he married Winna Willy Earle, who survives. Dr. Joyce is also survived by three sons, Claude D. Joyce, III, of Bryan, Jack W. Joyce of Houston, and Joe Bob Joyce of Palestine; a daughter, Dyna Joyce of Palestine; a brother, Gerald Joyce of Palestine; a sister, Mrs. Carolyn Rea of Palestine; and six grandchildren.

DR. M. J. KNIGHT

A Port Arthur general surgeon, Dr. Max John Knight, 62, died July 6 in St. Mary's Hospital there. He had been confined for six months.

The son of John A. and Susie Welborn Knight, he was born March 12, 1904, in Memphis, Tenn. He attended schools in Merryville, La., attended Tulane University, and was graduated from Tulane University School of Medicine in 1928. He interned at Charity Hospital of Louisiana in New Orleans for two years. He began practice in Port Arthur in 1930 and remained there until his death. He was a co-owner of the Knight Painton Clinic in Port Arthur.

Dr. Knight had served as a lieutenant commander in the US Navy Medical Corps during World War II.

He belonged to the Jefferson County Medical Society, Texas Medical Association, American Medical Association, Southern Medical Association, and was a fellow of the American College of Surgeons. He belonged to the First Baptist Church of Port Arthur, Cosmopolitan Masonic Lodge No. 872, AF and AM, and was a Shriner. He was a director at the First National Bank of Port Arthur.

Several years ago Dr. Knight deeded his home to the First Baptist Church to be used as a parsonage.

On Nov. 25, 1932, he married Irene H. Williams of Port Arthur. She died Jan. 8, 1966. Survivors include a sister, Mrs. Lucille Floyd of Columbus, Ga.; and a brother, Leo Knight of Lafayette, La.

DR. C. W. CASTNER

Former superintendent of the Rusk State Hospital, Dr. Charles W. Castner, 77, died Aug. 14 of cancer in Worcester Memorial Hospital, Worcester, Mass. He retired as superintendent of the hospital in June, 1965, bringing an end to a 50-year career of serving Texas in state hospitals.

A native of Cumby, Tex., he was born Nov. 28, 1888, the son of Whitfield and Janie Orlena Jones Castner. He attended schools in Cumby and White Sulphur Springs, and also attended Independent Normal College of Cumby. In 1909, he received his medical degree from the College of Physicians and Surgeons in Dallas. He interned at Dr. White's Hospital in Dallas.

On June 4, 1911, he married Grace Faye Tollett of Lavon.

He started his practice in Lavon, Tex., as a general practitioner. In 1915 he began his career with the state as a staff physician at Terrell State Hospital. He interrupted his career to serve with the US Army during World War I. In 1923, he first went to Rusk State Hospital as superintendent, only to leave in 1925 to become superintendent of the San Antonio State Hospital. After a brief stay in San Antonio, he moved to Wichita Falls to serve in the same capacity in the state hospital there. In 1937 he moved to Austin to serve as chief of the Division of Institutions, Board of Control. After nine years in that office, he was superintendent of the Austin State School for nine years. In 1954 he returned to Rusk and remained superintendent of the hospital until his retirement.

In 1955 the state legislature paid tribute to him in a resolution praising his valor during a riot in the maximum security unit of the hospital in Rusk. Dr. Castner offered himself as a hostage to free another physician who had been injured during the riot.

During World War II, Dr. Castner was a lieutenant colonel in the Texas National Guard.

A member of Collin County Medical Society, he belonged to the Texas Medical Association, American Medical Association, American Psychiatric Association, and the American Association on Mental Deficiency. A diplomate of the American Board of Psychiatry and Neurology, he was a co-founder and past president of the Texas Neuropsychiatric Association.

Dr. Castner had served two terms as president of the Rusk Kiwanis Club, and was a member of the American Legion and was a Mason. He belonged to the First Baptist Church of Rusk.

Mrs. Castner survives, as do a daughter, Mrs. Edahjane Svenson of Worcester, Mass.; two brothers, Morris Castner and Edwin Castner, both of Lubbock; a sister, Mrs. Pearl Fletcher of Naples, Tex., three grandchildren; and a great-granddaughter.

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COMING MEETINGS

American Medical Association, Clinical Meeting, Las Vegas, Nov. 27-Dec. 1, 1966. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 60610, Exec. Vice Pres.

Texas Medical Association, Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

NOVEMBER

American College of Chest Physicians, Las Vegas, Nev., Nov. 26-27, 1966. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

Radiological Society of North America, Chicago, Ill., Nov. 27-Dec. 2, 1966. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb. 68104, Sec.

Southern Medical Association, Washington, D. C., Nov. 14-17, 1966. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

DECEMBER

American Academy of Dermatology, Inc., Bal Harbour, Fla., Dec. 3-8, 1966. Dr. Stanley E. Huff, 636 Church St., Evanston, Ill., Sec.-Treas.

Private Clinics and Hospitals, Austin, Dec. 9-10, 1966. A. A. Hubbard, Alvarado Community Hospital, Alvarado 76009, Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 6-8, 1966. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore 21201, Sec.

Texas Academy of Internal Medicine, Galveston, Dec. 3, 1966. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth 76104, Sec.

Texas Rheumatism Association, Galveston, Dec. 9, 1966. Dr. Homer R. Goehrs, 801 W. 34th St., Austin 78705, Sec.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 2-3, 1966. Dr. Jack B. Lee, 409 Camden St., San Antonio 78215, Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 5-7, 1966. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 76102, Sec.

Twelfth District Society, Waco, Dec. 11, 1966. Dr. Walker Lea, Scott and White Clinic, Temple 76501, Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of General Practice, Dallas, Sept. 15-23, 1967. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 64112, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn. 55901, Exec. Sec.

American Academy of Pediatrics. Dr. E. H. Christopherson 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 7-9, 1967. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill. 60301, Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American Cancer Society, Inc. Mr. Charles R. Ebersol, 219 E. 42nd St., New York 10017, Sec.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie. 2141 14th St., Boulder, Colo. 80302, Treas.

American College of Gastroenterology. Mr. D. Weiss, 33 W. 60th, New York 10023, Exec. Dir.

American College of Obstetricians and Gynecologists. Mr. D. F. Richardson, 79 W. Monroe, Chicago 60603, Exec. Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Radiology, Los Angeles, Jan. 31-Feb. 4, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

American College of Surgeons. Dr. Samuel P. Harbison, 55 E. Erie St., Chicago, Ill. 60611.

American Congress of Physical Medicine and Rehabilitation, Miami Beach, Fla., Aug. 27-Sept. 1, 1967. Mr. Creston C. Herold, 30 N. Michigan, Chicago 60602, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn. 55901, Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., 27103, Sec.

American Gynecological Society, Phoenix, May 4-6, 1967. Dr. G. W. Douglas, 550 First Ave., New York 10016, Sec.

American Heart Association. Mr. Rome A. Betts, 44 E. 23rd, New York 10010, Exec. Dir.

American Hospital Association, Chicago, Aug. 21-24, 1967. Mr. James E. Hague, 840 N. Lake Shore Dr., Chicago 60611, Sec.

American Medical Women's Association. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 10032, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Public Health Association, Inc. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Las Vegas, Sept. 30-Oct. 4, 1967. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., 60608, Exec. Sec.

American Society of Clinical Pathologists, Las Vegas, Feb. 26-March 3, 1967 (Interim). Dr. William D. Dolan, Arlington Hospital, Arlington, Va., 22203, Sec.

American Surgical Association, Colorado Springs, May 11-14, 1967. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis 46207, Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Association of American Physicians and Surgeons, Inc. Mr. H. E. Northam, 185 N. Wabash, Chicago 60601, Exec. Dir.

Gastrointestinal Research Forum. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104, Sec.

Gastrointestinal Research Group. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn. 55901, Sec.

International College of Surgeons, April 30-May 5, 1967. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago 60610, Sec.

Medical Society of United States & Mexico, Guadalajara, Mexico, Jan. 25, 1968. Dr. Robert Witzemann, 222 W. Osborn Rd., Phoenix, Ariz., 85013, Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender. 3600 Florida St., Baton Rouge, La. 70806, Sec.

Southern Psychiatric Association. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

South Texas District, American College of Surgeons. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio 78205, Sec.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston 77550, Pres.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas 75219, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla., 73103, Sec.

State

Texas Academy of General Practice, Austin, June 12-14, 1967. Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin 78756, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas 75246, Sec.

Texas Association of Public Health Physicians, Dallas, May 4, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics. R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dallas, May 7, 1967. Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, Dallas, May 6-7, 1967. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio 78205, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, 4500 S. Lancaster, Dallas 75216, Sec.

Texas Division, American Cancer Society, Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Heart Association, Houston. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 US Highway 290 E., Austin 78723, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615, Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dallas, May 5-6, 1967. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio 78205, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, May 5-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Houston, Sept. 28-30, 1967. Dr. C. E. Gilmore, 811 Bonham, Paris 75460, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston 77025, Sec.

Texas Proctologic Society, Dallas, May 4, 1967. Dr. T. Melton, 636 Hermann Professional Bldg., Houston 77025, Sec.

Texas Public Health Association, San Antonio, Feb. 26-March 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 78731, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967. Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth 76102, Sec.

Texas Rehabilitation Association, Dallas, May 4, 1967. D. Preston, 3700 Worth, Dallas 75210, Sec.

Texas Society of Anesthesiologists, Dallas, May 6-7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society on Aging. Mrs. Edith DeBusk, Rio Grande Bldg., 251 N. Field, Dallas 75202, Sec.

Texas Society of Athletic Team Physicians, Dallas, May 5, 1967. Dr. W. Cone Johnson, Scott and White Clinic, Temple 76501, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont 77701, Sec.

Texas Society of Plastic Surgeons, Dallas, May 6, 1967. Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 201 E. 14th St., Austin 78701, Exec. Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Surgical Society, Fort Worth, April 2-4, 1967. Dr. Jesse E. Thompson, 1008 N. Washington, Dallas 75204, Sec.

Texas Traumatic Surgical Society. Dr. J. C. Long, 805 W. 8th, Plainview 79072, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Rd., Austin 78722, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview 75601.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso 79901, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullan, El Paso 77905, Sec.

Third District Society. Dr. Louis Devanney, 2209 W. 7th, Amarillo 79106, Sec.

Fifth District Society, San Antonio, Jan. 25, 1967. Dr. Walter Walthall, 1114 South Texas Bldg., San Antonio 78205, Councilor.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin 78701, Sec.

Eighth District Society. Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City 77414, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston 77002, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont 77701, Sec.

Eleventh District Society, Tyler, Spring, 1967. Dr. Lucy Mathis, 107 Redland Lane, Palestine 75801, Sec.

Thirteenth District Society, Fort Worth. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth 76104, Sec.

Clinics

Blackford Memorial Lectures. Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas 75201, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Pl., San Antonio 78212, Exec. Sec.

Oklahoma City Clinical Conference. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 73102, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 70112, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 17-19, 1967. Dr. W. Tom Arnold, 6448 Fannin, Houston 77025, Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 Fifth Ave., Fort Worth 76104, Sec.

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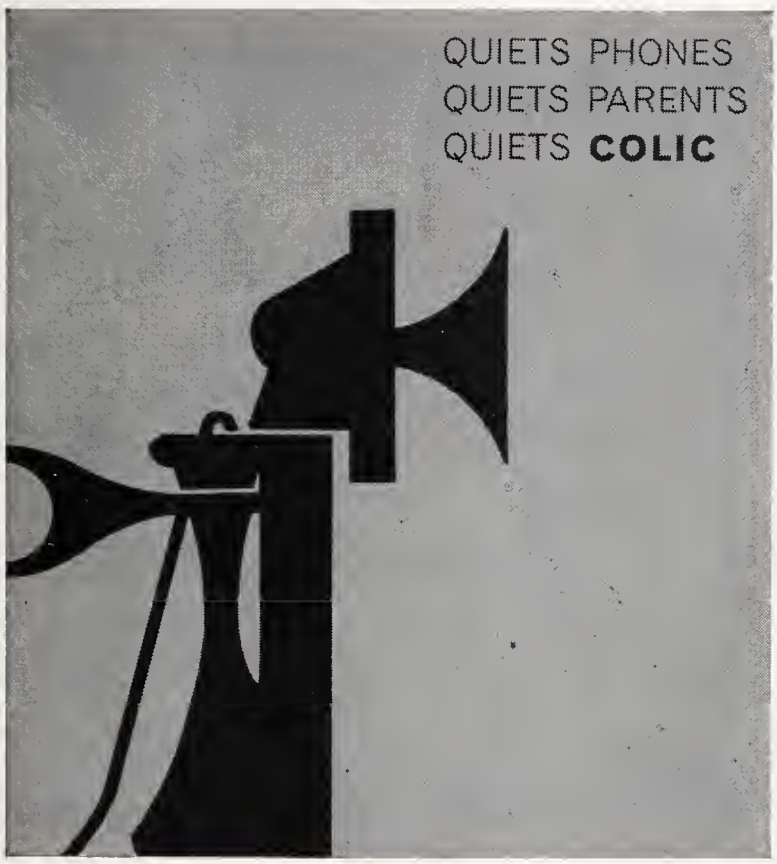

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More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.

Contraindications: Severe renal impairment; previous hypersensitivity.

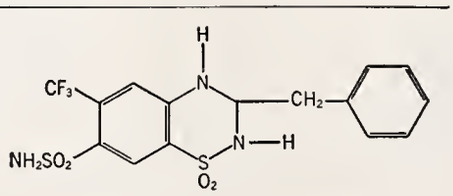
Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: **1.** Southworth, H.: Proc. Soc. Exper. Biol. & Med. *36*:58, 1937. **2.** Mann, T. and Keilin, D.: Nature *146*:164, 1940. **3.** Pitts, R. F., and Alexander, R. S.: Am. J. Physiol. *144*:239, 1945. **4.** Schwartz, W. B.: New England J. Med. *240*:173, 1949. **5.** Friedberg, C. K., in Moyer, J. H., and Fuchs, M.: Edema Mechanisms and Management, Philadelphia, W. B. Saunders Co., 1960, p. 259. **6.** Cumming, J. R.; Tabachnick, E., and Seelig, M., in Moyer, J. H., and Fuchs, M.: op. cit., p. 254. **7.** Werko, L., in Moyer, J. H., and Fuchs, M.: op. cit., p. 188. **8.** Beyer, K. H., Jr., in Moyer, J. H., and Fuchs, M.: op. cit., p. 274. **9.** Maren, T. H., and Wiley, C. E.: J. Pharmacol. & Exper. Therap. *143*:230, 1964. **10.** Earley, L. E., and Orloff, J.: Ann. Rev. Med. *15*:149, 1964. **11.** Fuchs, M., and Mallin, S. R., in Moyer, J. H., and Fuchs, M.: op. cit., p. 276. **12.** Ford, R. V., in Moyer, J. H., and Fuchs, M.: op. cit., p. 290. **13.** cited in Fuchs, M., and Mallin, S. R. (ref. 11): op. cit., p. 283.



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1-2 yr. . . . ½ tsp. 5 times daily (5 mg.)
2-5 yr. . . . 1 tsp. t.i.d. (6 mg.)
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8-12 yr. . . 1 tsp. 5 times daily (10 mg.)

Adults: 2 tsp. 5 times daily (20 mg.)
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*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

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Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

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PHARMACOLOGY: Triaminic decongests and promotes drainage of nasal and paranasal passages, and prevents any further histamine-induced damage; the triple sulfonamides inhibit susceptible bacterial invaders. INDICATIONS: For congestion and infection of the upper respiratory tract caused by sulfa-susceptible organisms. DOSAGE: Adults: 2 to 4 tablets initially, followed by 2 tablets every 6 hours. Medication should be continued until patient has been afebrile for 3 days. ADVANTAGES: The advantages of Trisulfaminic in upper respiratory infections are: freedom from narcotics or alcohol; therapeutic reliability; safety; economy; ease of administration; freedom from potential sensitization to broad-spectrum antibiotics which may be reserved for lower respiratory or other infections caused by susceptible organisms. CONTRAINDICATIONS: Contraindicated in sulfonamide and antihistamine sensitivity, impaired renal function, pregnancy approaching term, and in premature infants and newborn infants during the first month of life. Do not use in patients with glaucoma, prostatic hypertrophy, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction. WARNING: Use only after careful evaluation in patients with liver or renal damage, urinary obstruction, or blood dyscrasias. Deaths have been reported from hypersensitivity reactions with administration of sulfonamides. In intermittent or prolonged therapy, blood counts and liver and kidney function tests should be performed periodically. Sulfonamide therapy may potentiate the hypoglycemic action of sulfonylureas. PRECAUTIONS: Use with caution in patients with histories of significant allergy or asthma. Assure an adequate fluid intake. Because the antihistamines may cause drowsiness of varying degree, warn patients about activities requiring alertness such as driving a car or operating dangerous machinery. Use with caution in the presence of hypertension, hyperthyroidism, cardiovascular disease and diabetes. ADVERSE REACTIONS: As in all sulfonamide therapy, the following reactions may occur: headache, nausea, vomiting, diarrhea, icterus, hepatitis, pancreatitis, urticaria, rash, fever, cyanosis, hematuria, crystalluria, proteinuria, blood dyscrasias, petechiae, purpura, neuropathy and injection of the conjunctiva and sclera. If one or more of these reactions occur, the drug should be discontinued. With antihistaminic therapy there have been reports of sedation varying from mild drowsiness to deep sleep, dizziness, lassitude, inability to concentrate, fatigue, incoordination, tinnitus, blurred vision, diplopia, euphoria, nervousness, insomnia, tremors, palpitation, hypotension, headache, chest tightness, urinary frequency, dysuria, tingling of the hands, dryness of the mouth, throat, and nose, gastrointestinal disturbances such as epigastric distress, anorexia, nausea, vomiting, constipation and diarrhea and very rarely, leukopenia and agranulocytosis. Adverse reactions reported with the use of sympathomimetic amines include anxiety, tension, restlessness, nervousness, tremor, weakness, insomnia, headache, palpitation, tachycardia, angina, elevation of blood pressure, sweating, mydriasis, anorexia, nausea, vomiting, dizziness, constipation, and dysuria due to vesicle sphincter spasm. PACKAGE INFORMATION: Trisulfaminic Tablets: Supplied in bottles of 100 tablets. CAUTION: Federal law prohibits dispensing without prescription.

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1. McCabe, W. R., and Jackson, G. G.: New England J. Med. 272:1037, 1965.

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Adverse Reactions: Occasional reactions such as circumoral paresthesias, tingling of the extremities, pruritus, vertigo or dizziness may occur. Reduction of dosage may alleviate symptoms. Therapy need not be discontinued, but such patients should be observed with extra care.

Warning: Patients should be cautioned not to drive vehicles or use hazardous machinery while on therapy.

Precautions: In cases of impaired or suspected renal impairment, use with greater caution and reduce dosage in proportion to extent of impairment. Transient elevations of BUN have been reported. As a routine precaution, appropriate blood studies should, therefore, be made during prolonged therapy.

As with all polypeptides, the possibility of muscular weakness, including apnea, due to inadvertent overdosage or normal dosage in the presence of impaired renal function, should not be overlooked. In cases of apnea, medication should be promptly discontinued and assisted respiration given until serum levels fall and normal breathing is restored.

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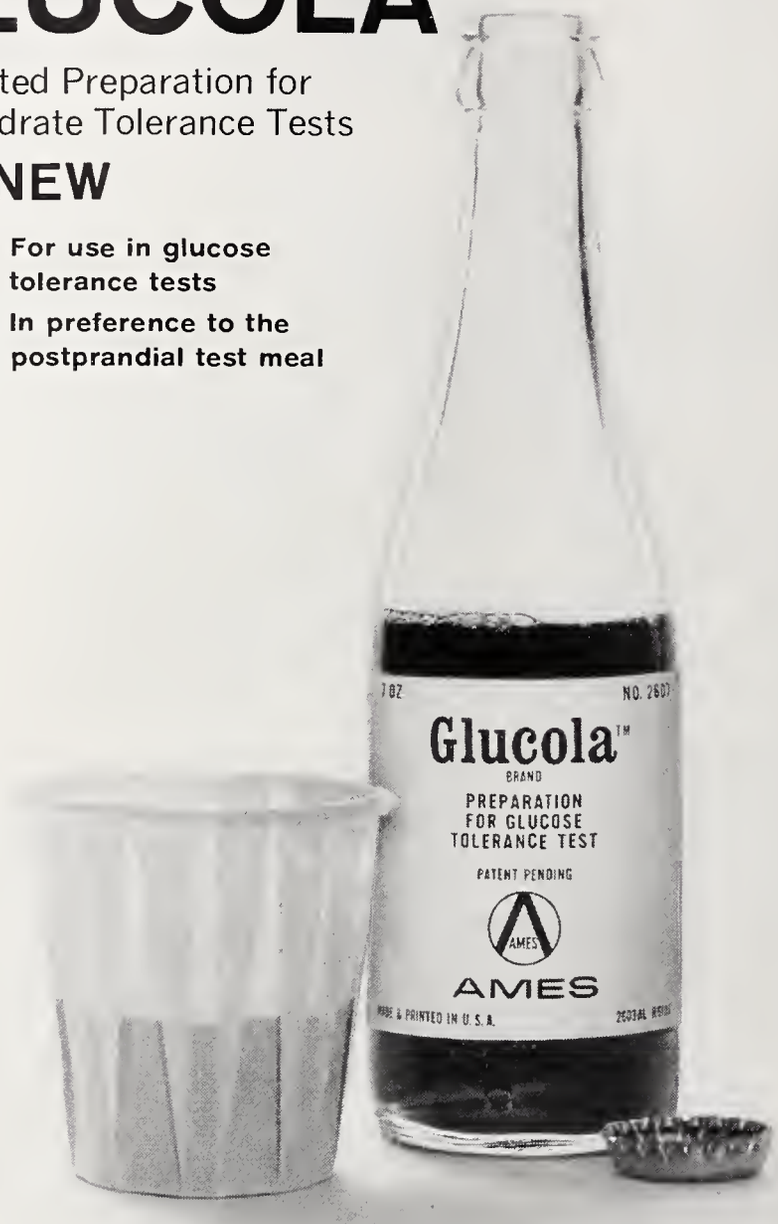

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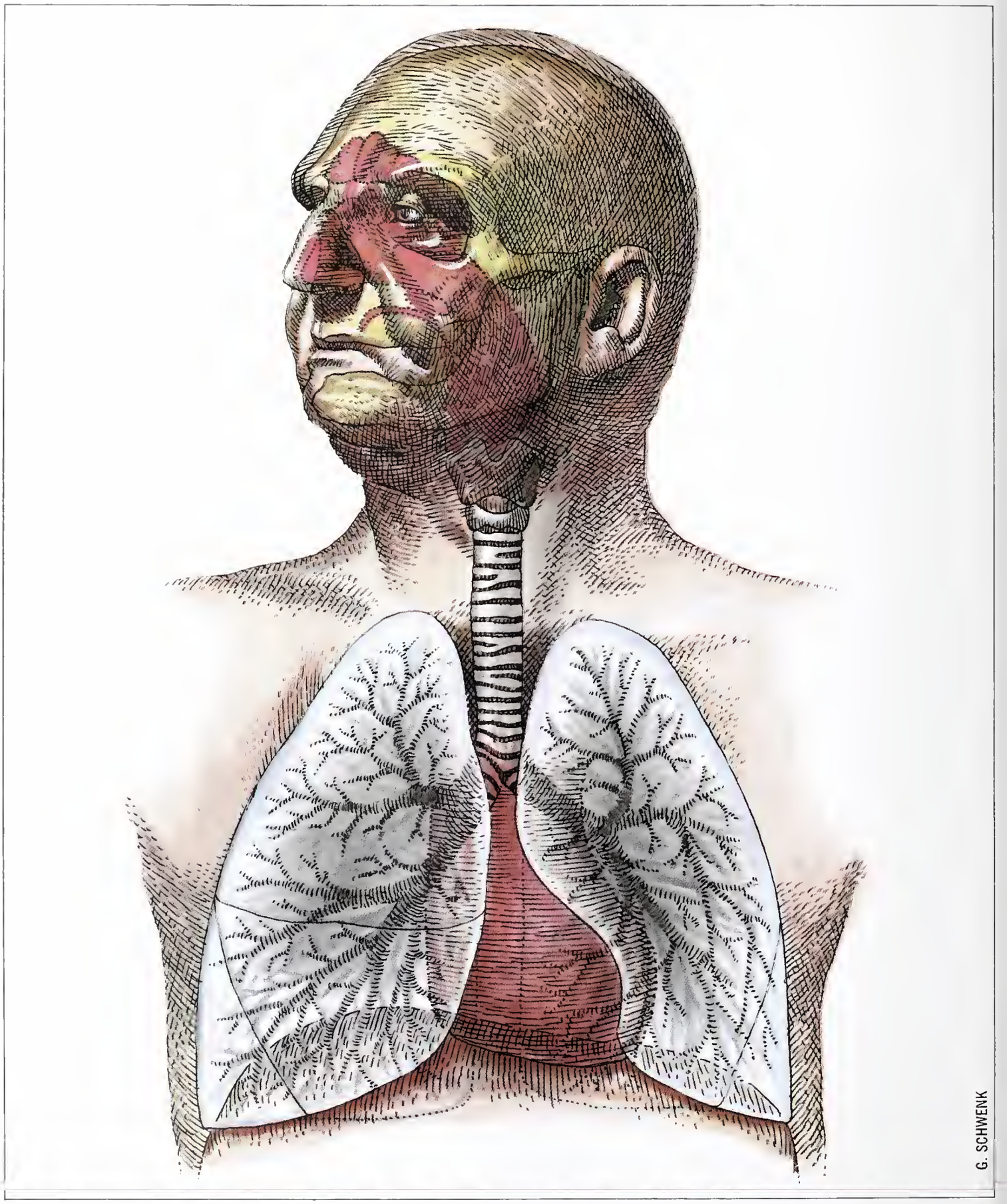
this issue: the common cold and the aging patient

the common cold and the aging patient

Louis J. Vorhaus, II, M.D., F.A.C.P.



G. SCHWENK

Effects of aging on the anatomic and physiologic aspects of the respiratory apparatus.

The chest becomes more fixed, less mobile and less elastic as the bronchial walls and thoracic ligaments lose elasticity. The diaphragm and intercostal muscles atrophy and weaken. The lungs become smaller, flabbier and weigh less, decreasing vital and total lung capacity, increasing residual volume and the alveolar dead space.

Sir William Osler described pneumonia as the welcome friend of the aged patient, because the patient with pneumonia usually died quietly. But today, the well-informed physician is an even better friend of the aging patient, since it is better to live than to die, no matter how quietly.

One of the first avenues of approach in the control of the hazards of respiratory disease in the aging patient is prompt and proper attention to the common cold or upper respiratory infection. The common cold may be the first step in the relatively short path to lower respiratory infection, broncho-pneumonia and death. This train of events occurs frequently among older persons. Indeed, pneumonia is one of the most common causes of their admission to hospitals and ranks high on the list of geriatric killers. Colds are more debilitating in elderly people and the aged are more likely candidates for secondary infections such as sinusitis and bronchitis. These infections, in turn, are more prone to lead to broncho-pneumonia, because of lowered resistance and anatomic and physiologic changes in the lungs of the elderly.

What is different about the respiratory tree of an aged person and that of an otherwise healthy younger adult? Aging certainly takes its toll on all parts of the body, affecting both anatomic and physiologic aspects of the respiratory apparatus. These changes are in part due to the wear and tear that occurs over the years; the repeated bouts of respiratory infection, long exposure to atmospheric pollutants, to occupational inhalants, smoking, malnutrition, obesity, inactivity and the development of other diseases which may affect the lungs.

With the passage of years, the lungs change. They become scarred and emphysematous and lose their compliance. The whole chest becomes more fixed, less mobile and less elastic.

The anatomic changes that occur in aging render the lungs less efficient. Tests of pulmonary function in senescence show a deterioration characterized by a decrease in vital capacity and total lung capacity, an increase in residual volume and alveolar dead space. Maximum breathing capacity is reduced and uniformity of ventilation deteriorates. These problems are often aggravated by the obstructed breathing, fever and secondary infection associated with the common cold, placing an additional stress on the entire cardiopulmonary reserve.

In addition, the efficiency of a cough is below par in older persons even though they are in good health. This is partly due to the decreased respiratory excursions and distensibility of the chest wall, and partly from loss of elasticity of bronchial walls which tend to make them collapse in a cough.

The elderly patient's resistance to infection is often reduced. Nutritional deficiencies are more common in aged people. There is some evidence to indicate that their capacity to respond to stress is less efficient. Finally, there is often relatively meager symptomatic response to acute disease. The absence of obvious or dramatic clinical signs and symptoms of severe illness is particularly dangerous because, coming as it

(concluded on following page)

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Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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does in a person whose defenses are weakened both locally and systemically, pulmonary disease may progress rapidly to irreversible stages before medical attention is sought. Respiratory infection is especially hazardous because the aged patient responds badly to hypoxia. Not only is his response to oxygen lack impaired, but the work of breathing, due to decreased compliance of the lung and increased stiffness of the thorax, is markedly augmented.
Many patients late in life are in a precarious and delicate cardiopulmonary balance which is easily decompensated from relatively minor insults such as colds and upper respiratory infections.

For all of these reasons, geriatricians long have stressed the importance of preventing respiratory insults. Today we have better ways of treating respiratory infection, improved techniques for clearing the lungs and bronchial tubes of secretions and better understanding of ways of improving ventilation. We possess a broader spectrum of antimicrobial agents including newer ones to deal with previously resistant organisms. Even so, the death rate from pneumonia is high in older people, and it is preferable to avoid the disease than to treat it. To do so, attention must be paid to the general maintenance of good health and all that implies, as well as to the prevention, elimination and treatment of associated conditions that predispose to or cause pneumonia such as chronic upper or lower respiratory infection, respiratory allergy, chronic sinusitis and exposure to inspired irritants.

Colds and other minor respiratory infections, which favor the development of broncho-pneumonia, should be treated vigorously and promptly, particularly those patients whose aging process has been accompanied by the development of chronic pulmonary disease. Upper respiratory passages should be cleared with decongestants. Sinuses should be drained adequately. And, when indicated, appropriate antimicrobial therapy should be instituted before serious infection of the lower respiratory tree supervenes.

In decades past it was understandable that physicians welcomed pneumonia for the aged patients because it offered them a quiet and peaceful demise. Today we recognize that in many cases, peaceful as it may have been, such deaths were often avoidable. With the current knowledge and understanding of the problems that respiratory infections impose on aging people, vigilant medical attention can often restore them to a vigorous, rewarding and productive life so that the many opportunities that exist today for people to enjoy their golden years are realized and not stolen by untimely death.

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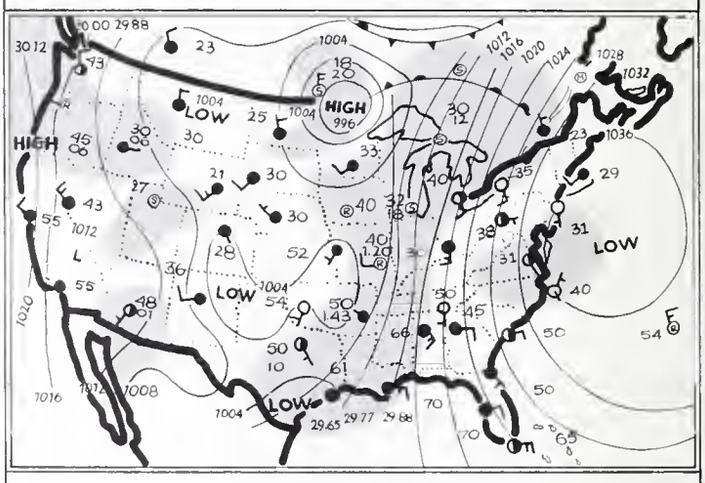

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Tussagesic® provides up to 24-hour coverage of the tough cold with a single timed-release tablet dosed morning, midafternoon and at bedtime. Coughs are broken up, runny and stuffed noses are cleared and pain is relieved.

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Acetaminophen	325 mg.

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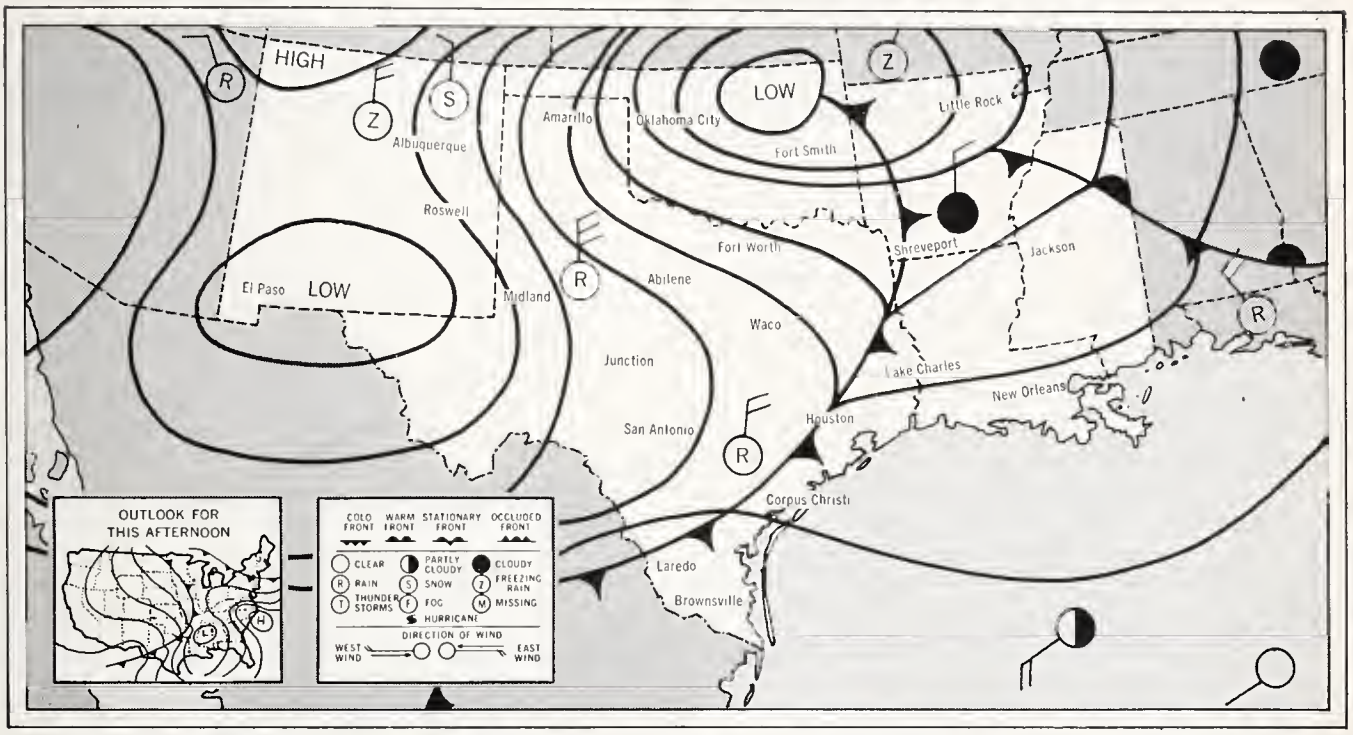

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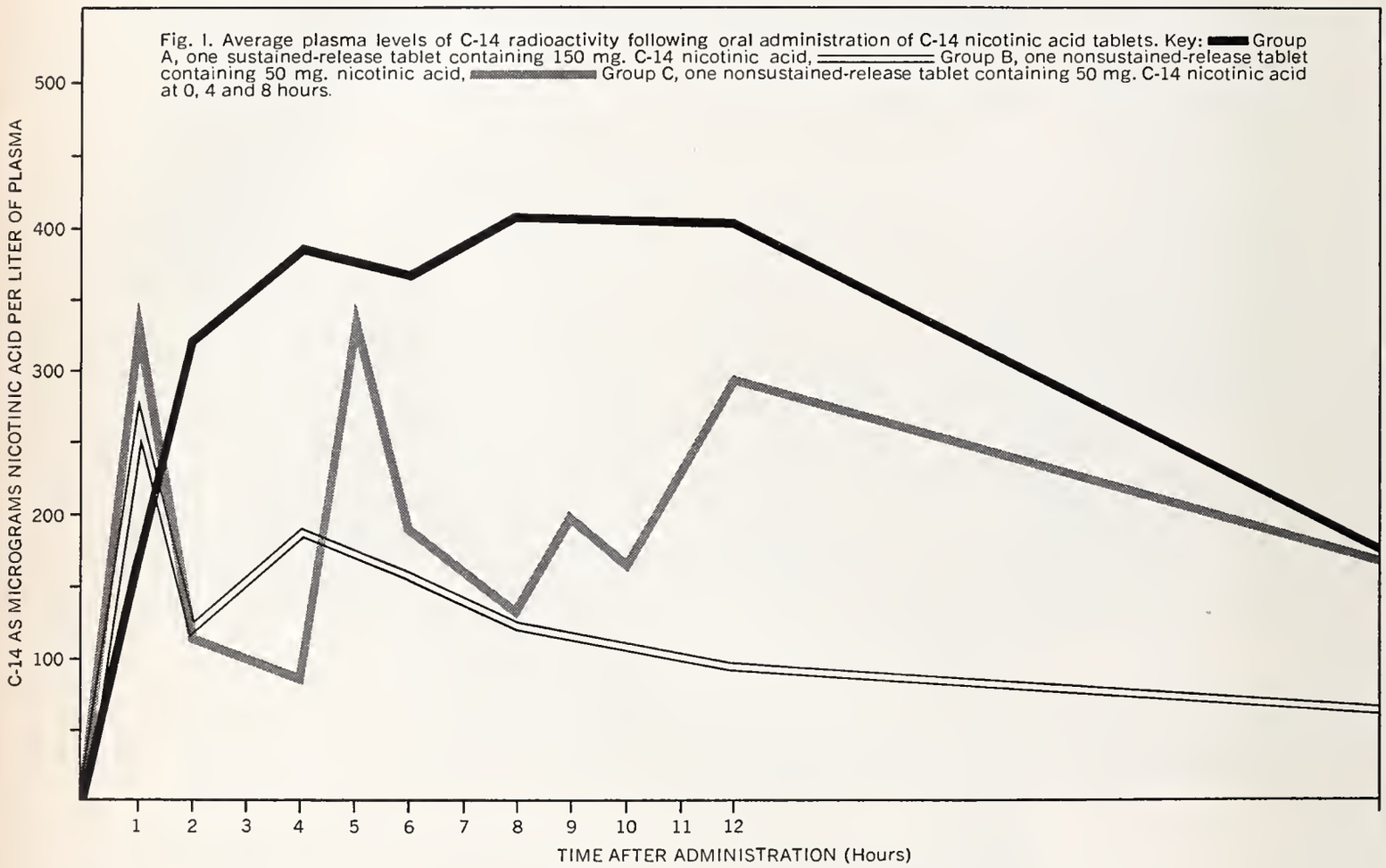
Fig. I. Average plasma levels of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MICROGRAMS NICOTINIC ACID PER LITER OF PLASMA
500
400
300
200
100
1
2
3
4
5
6
7
8
9
10
11
12
TIME AFTER ADMINISTRATION (Hours)

(fewer absent doses by absent-minded patients)

Human volunteer subjects were administered Geroniazol TT tablets with the nicotinic acid component made radioactive with C-14. Plasma and urine samples were analyzed. (See Figures I and II) The radioactive tracer study substantiated the previous clinical evidence that the release of nicotinic acid from the Geroniazol TT tablet produced a gradual rise in plasma levels to a plateau for a total of 12 hours and more.

Such proven sustained activity makes the management of geriatric patients much easier by minimizing the possibility of neglected doses through absent-mindedness or senile confusion. Therapy *can* be continuous on a daily dose of only one Geroniazol TT tablet every 12 hours.

The gradual release of nicotinic acid in Geroniazol TT will provide the well-known peripheral vasodilatation needed in patients with deficient circulation and with a minimum amount (if any) of "flushing." Also, cerebrovascular circulation is complemented by pentylenetetrazol, long-established as a cerebral and respiratory stimulant.

Geroniazol TT improves the typical, unfortunate, signs of senile confusion. Patients become more alert,

ged and debilitated

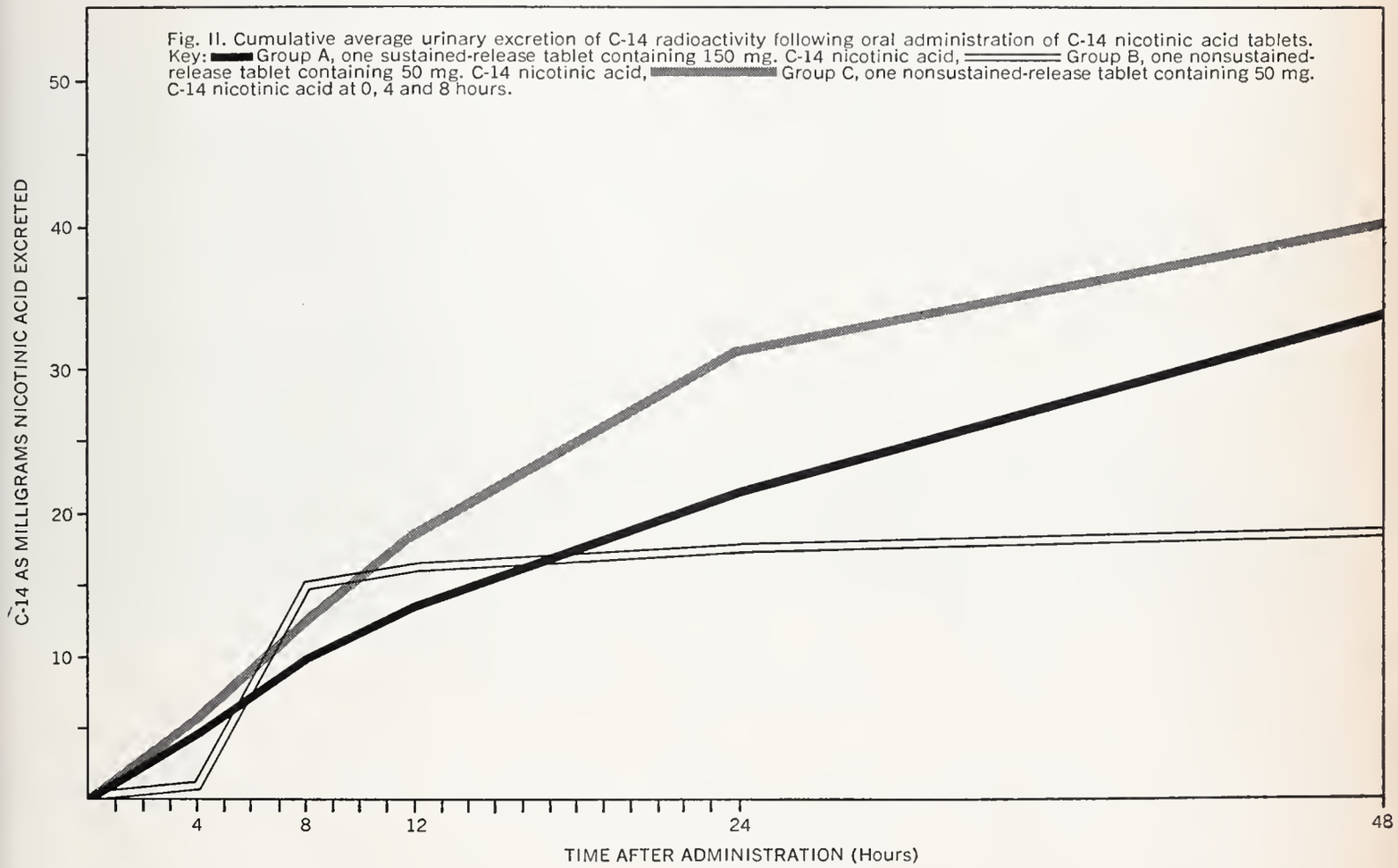
Fig. II. Cumulative average urinary excretion of C-14 radioactivity following oral administration of C-14 nicotinic acid tablets. Key: Group A, one sustained-release tablet containing 150 mg. C-14 nicotinic acid, Group B, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid, Group C, one nonsustained-release tablet containing 50 mg. C-14 nicotinic acid at 0, 4 and 8 hours.
C-14 AS MILLIGRAMS NICOTINIC ACID EXCRETED
50
40
30
20
10
4
8
12
24
48
TIME AFTER ADMINISTRATION (Hours)

less confused and moody. Personal care, memory, emotional stability, social attention improve. Fatigue, apathy and irritability are reduced.

A prescription for 100 tablets of Geroniazol TT will permit your patients to enjoy the benefits of time-prolonged nicotinic acid/pentylenetetrazol therapy, at an economical price. Dosage is only one tablet every 12 hours.

Contraindications: There are no known contraindications.

Precautions: Exercise caution when treating patients with a low convulsive threshold.

Side Effects: Side effects are rarely encountered, however due to the vasodilatation effect of nicotinic acid, transitory mild nausea, flushing, tingling and pruritus are possible.

Dosage: One tablet every 12 hours.

Supplied: Prescribe bottles of 100 tablets, to take advantage of recent price reduction.

References: 1. Report by Nuclear Science & Engineering Corp., Pittsburgh, Pa., in files of Philips Roxane Laboratories. 2. Connolly, R.: W. Virginia Med. J. *56*:263 (Aug.) 1960. 3. Curran, T. R., and Phelps, D. K.: Am. Pract. & Digest Treat. *11*:617 (July) 1960.

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Contraindications
Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. Because of the increased possibility of toxic reactions, the drug should be used with greater care in the elderly and should not be given when the patient is senile or when other potent chemotherapeutic agents are given concurrently. Large doses of Butazolidin alka are contraindicated in patients with glaucoma.

Warning
If coumarin-type anticoagulants are given simultaneously, the physician should watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Patients receiving such concomitant therapy should be carefully observed for this effect.

Use with caution in the first trimester of pregnancy.

Precautions
Before prescribing, the physician should obtain a detailed history and perform a complete physical and laboratory examination, including a blood count. The patient should be kept under close supervision and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Regular blood counts should be made to guard against blood dyscrasias.

Adverse Reactions
The most common adverse reactions are nausea, edema and drug rash. Moderately lowered red cell count may sometimes occur due to hemodilution. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vertigo or languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia are also possible side effects.

Confusional states, hyperglycemia, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hepatitis, jaundice and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently.

Dosage
The initial daily dosage in adults is 300-600 mg. daily in divided doses. In most instances, 400 mg. daily is sufficient. When improvement occurs, dosage should be decreased to the minimum effective level: this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

For complete details, please refer to full prescribing information.
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Behind continued high blood pressure readings lies the possibility of organic damage[1-13]

MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.

"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

Reduce the blood pressure with Rautrax-N

Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

References: 1. Page, I. H., and Dustan, H. P.: The Usefulness of Drugs in the Treatment of Hypertension, in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 95. **2.** Hollander, W.: The Evaluation of Antihypertensive Therapy of Essential Hypertension in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 97. **3.** Nickerson, M.: Antihypertensive Agents and the Drug Therapy of Hypertension, in Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 3, New York, The Macmillan Co., 1965, p. 727. **4.** Berkson, D. M.: Indust. Med. & Surg. **32**:371, 1963. **5.** Cohen, B. M.: M. Times **91**:645, 1963. **6.** Lee, R. E., et al.: Am. J. Cardiol. **11**:738, 1963. **7.** Moyer, J. H.: Am. J. Cardiol. **9**:821, 1962. **8.** Moser, M.: New York J. Med. **62**:1177, 1962. **9.** Wood, J. E., and Battey, L. L.: Am. J. Cardiol. **9**:675, 1962. **10.** Moyer, J. H., and Heider, C.: Am. J. Cardiol. **9**:920, 1962. **11.** Moser, M., and Macaulay, A. I.: New York State J. Med. **60**:2679, 1960. **12.** Judson, W. E.: Nebraska M. J. **44**:305, 1959. **13.** Hodge, J. V.; McQueen, E. G., and Smirk, H.: Brit. M. J. **1**:5218, 1961. **14.** Moyer, J. H., and Brest, A. N.: Hypertension Recent Advances, Philadelphia, Lea & Febiger, 1961, p. 633. **15.** Berry, R. L., and Bray, H. P.: J. Am. Geriatrics Soc. **10**:516, 1962. **16.** Reid, W. J.: J. Am. Geriatrics Soc. **13**:365, 1965. **17.** Feldman, L. H.: North Carolina M. J. **23**:248, 1962.

Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

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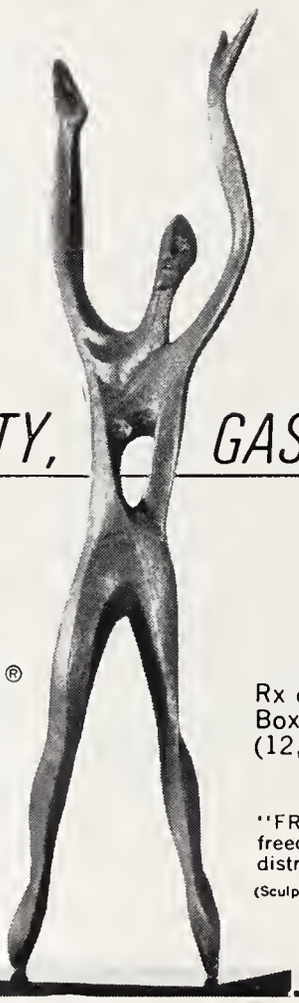

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
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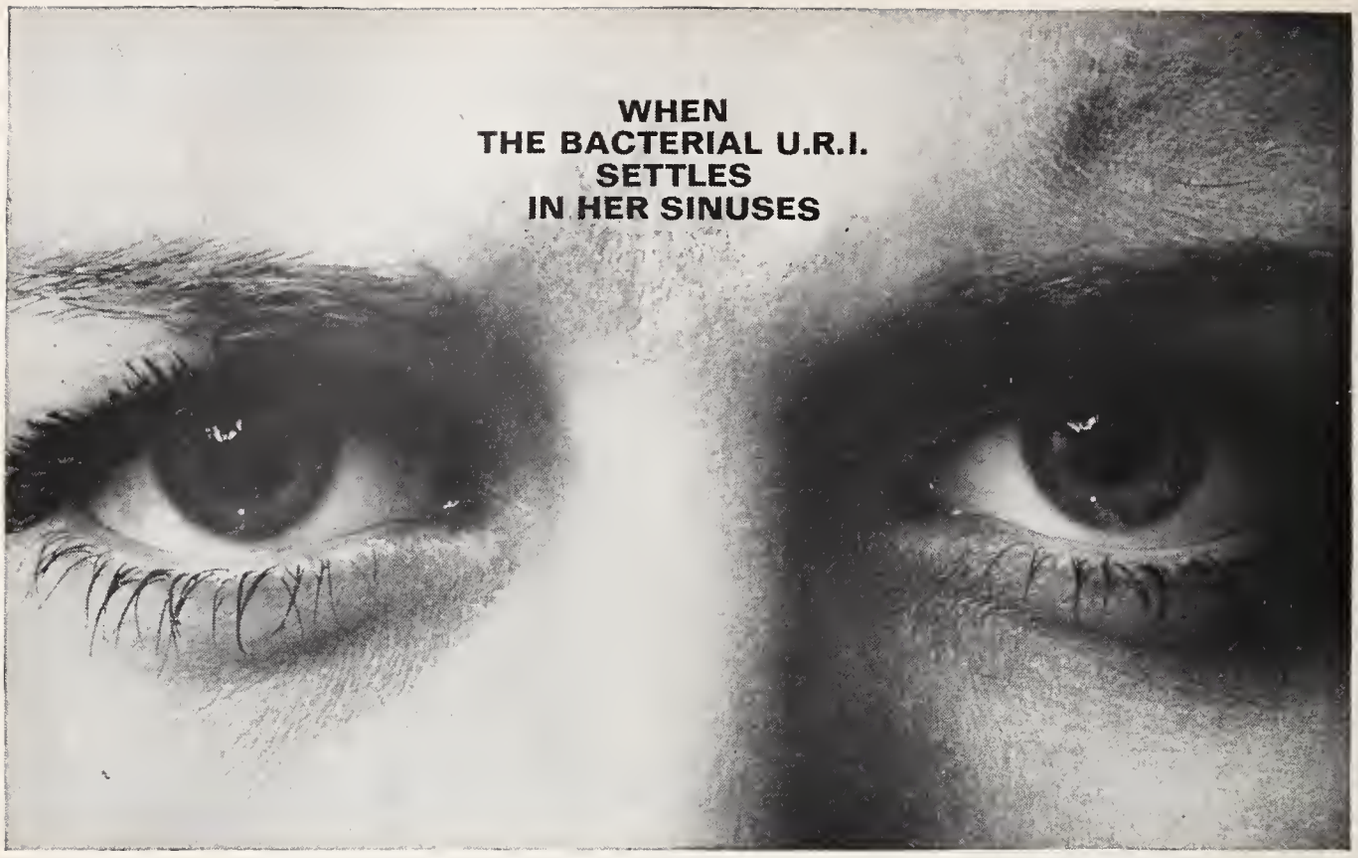

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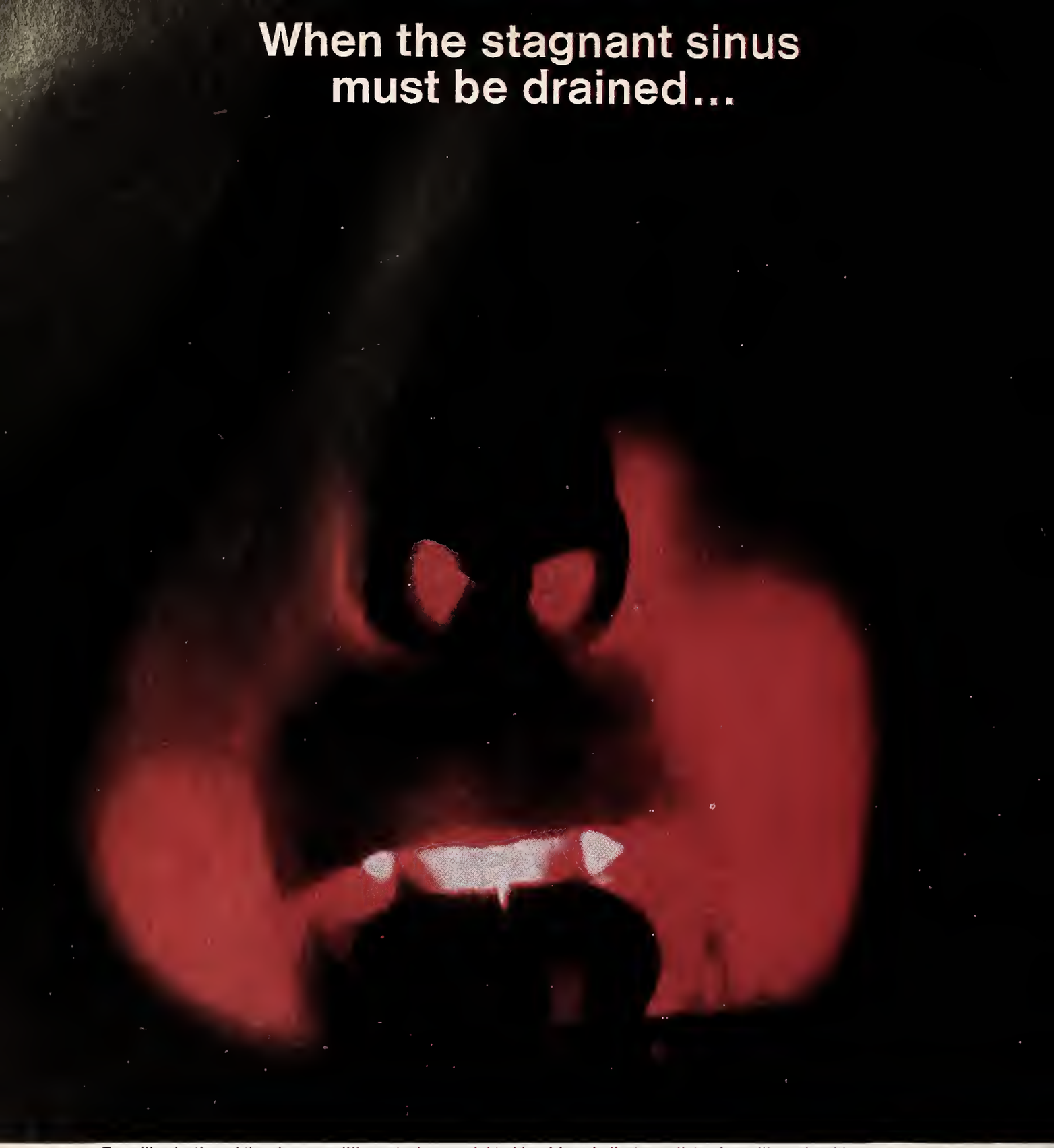

Transillumination of the sinuses—diffuse shadow on right side of face indicates unilateral maxillary sinusitis.

In the common cold, Neo-Synephrine is unsurpassed for reducing nasal turgescence. It stops the stuffy feeling at once. It opens sinus ostia to re-establish drainage and lessen the chance of sinusitis. With Neo-Synephrine, in the concentrations most commonly used, decongestion lasts long enough for extended breathing comfort, without endangering delicate respiratory tissue. Systemic side effects are virtually unknown. There is little rebound tendency.

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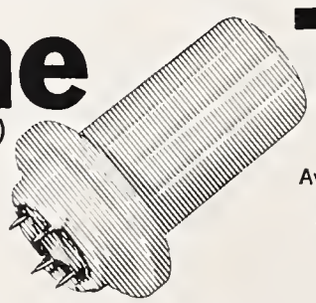

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"'Tranquilizer' is not a good word"[1]

"THIS classification is psychologically too seductive, pharmacologically too unspecific, and in terms of results not infrequently untrue."[2]

What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there *are* differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1] This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Triflupromazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

Prolixin is therapeutically effective without excessive sedation

When used as a 'tranquilizer' in general medical practice, in many patients Prolixin (Squibb Fluphenazine Hydrochloride) suppresses anxiety, but not normal activity. These two features are of particular importance to patients who must be able to live and work without their normal daily activities being restricted.

Because of its longer duration of action, Prolixin, in doses of as little as one to three milligrams in adults, generally taken once a day, is effective in maintaining many patients free of their symptoms of anxiety and tension.

Contraindications: Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use with caution in patients with a history of convulsive disorders. Severe reactions may occur in patients with idiosyncrasy to other centrally-acting drugs, and severe hypotension may occur in patients with special medical disorders, e.g. mitral insufficiency and pheochromocytoma.

Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

Available: 1 mg. tablets. Bottles of 50 and 500.

For full prescribing information, see package insert.

References: 1. Simpson, G.M.: Postgrad. Med. 39:557, 1966. 2. Freyhan, F.A.: Am. J. Psychiat. 115:577, 1959. 3. Dorland's Illustrated Medical Dictionary, ed. 24, Philadelphia, W. B. Saunders Co., 1965, p. 1603. 4. Physicians' Desk Reference, 1966, Oradell, N.J., 1965, p. 310. 5. Cohen, S.: Northwest Med.: 65:197, 1966. 6. Detre, T., and Jarecki, H.: Connecticut Med. 25:553, 1961. 7. Sainz, A.: Psychosomatics 5:167, 1964.

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atropine sulfate .....0.025 mg.

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• Functional hypermotility • Acute infections • Malabsorption syndrome • Drug therapy • Gastroenteritis and colitis • Irritable bowel • Regional enteritis • Ileostomy • Ulcerative colitis • Food poisoning

For correct therapeutic effect
Rx correct therapeutic dosage

Dosage: The recommended initial daily dosages, given in divided doses until diarrhea is controlled, are:

Children: Total Daily Dosage

3-6 mo. . . ½ tsp.* t.i.d. (3 mg.)
6-12 mo. . ½ tsp. q.i.d. (4 mg.)
1-2 yr. . . . ½ tsp. 5 times daily (5 mg.)
2-5 yr. . . . 1 tsp. t.i.d. (6 mg.)
5-8 yr. . . . 1 tsp. q.i.d. (8 mg.)
8-12 yr. . . 1 tsp. 5 times daily (10 mg.)

Adults: 2 tsp. 5 times daily (20 mg.)
(or 2 tablets q.i.d.)

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Lomotil should be kept out of reach of children since accidental overdosage may cause severe respiratory depression. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

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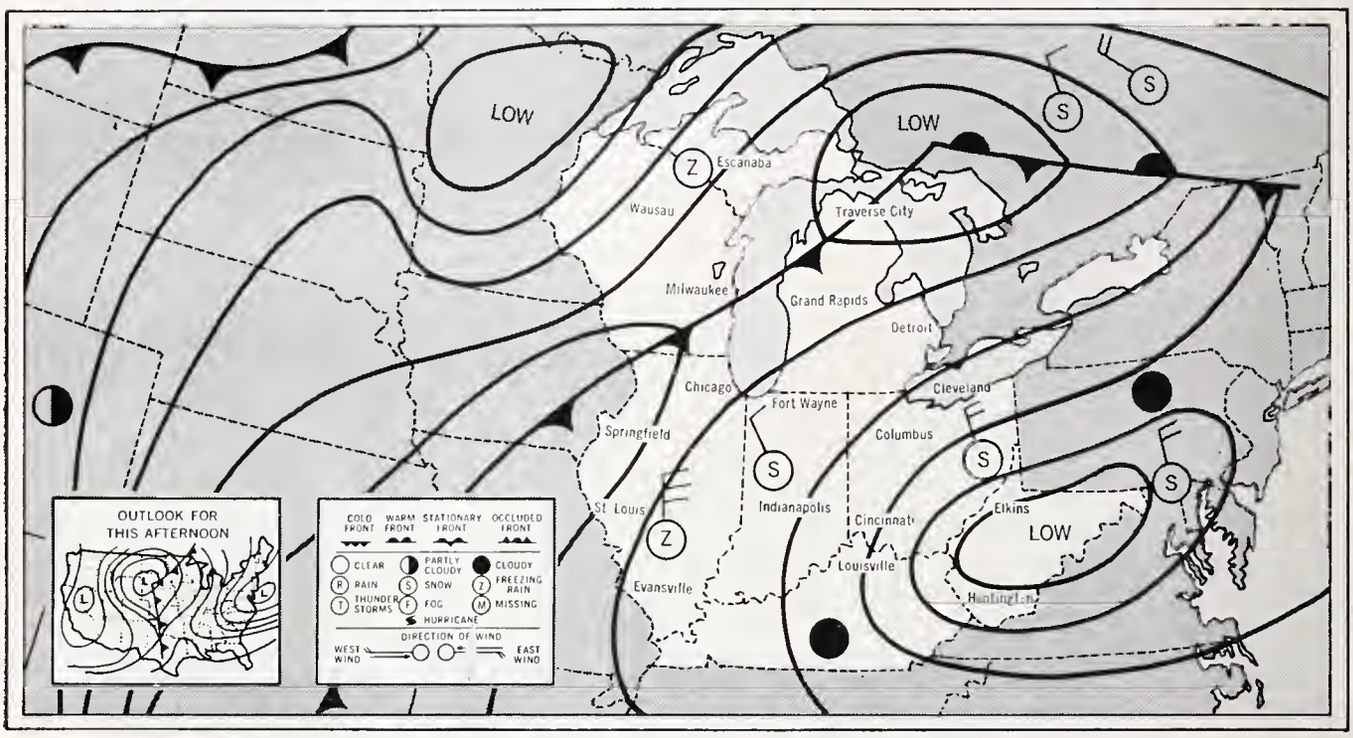

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Triaminic® (phenylpropanolamine hydrochloride 25 mg., pheniramine maleate 12.5 mg., pyrilamine maleate 12.5 mg.)	50 mg.
Dextromethorphan hydrobromide	30 mg.
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Acetaminophen	325 mg.

Dosage: Adults—1 tablet, swallowed whole to preserve timed-release feature, in morning, midafternoon and at bedtime. *Side effects:* Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. *Precautions:* The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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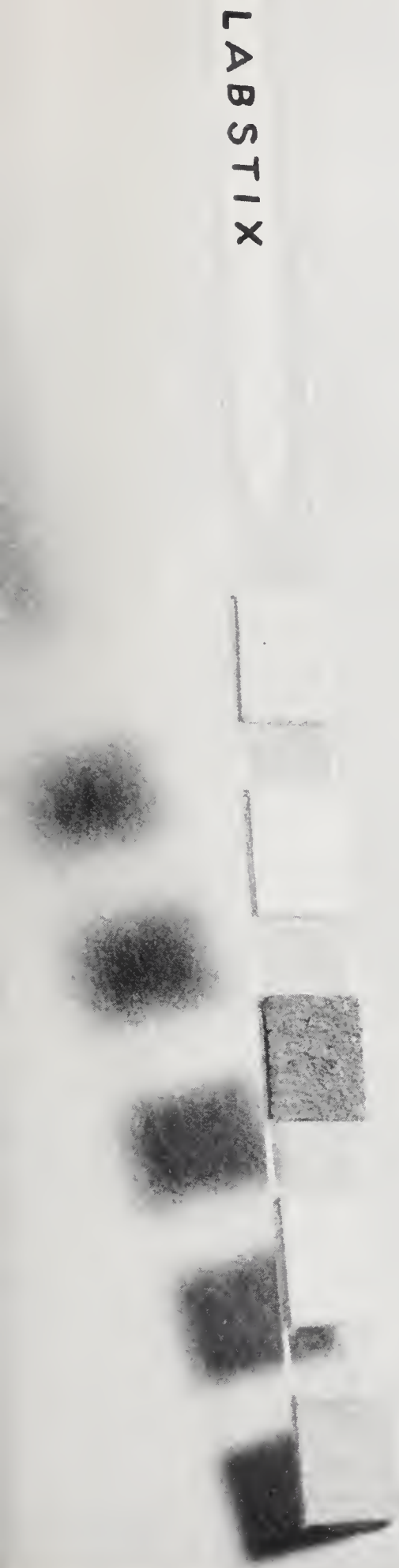

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Dosage: Adults, 1 tablespoonful; children (over 15 years old), 1 to 2 teaspoonfuls; children (4 to 15 years old), 1 teaspoonful. To be taken three times daily 30 minutes before meals.

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Corcoran, A. C.: The choice of drugs in the treatment of hypertension. In: Drugs of Choice 1966-67, W. Modell, Ed., St. Louis, C. V. Mosby Company, 1966, p. 417.

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Composition: Furoxone Liquid contains, per 15 cc. tablespoonful, furazolidone 50 mg., pectin 225 mg., and kaolin 3.0 Gm. Furoxone Tablets each contain 100 mg. of furazolidone.

References: 1. Editorial: J.A.M.A. *189*:691 (Aug. 31) 1964. 2. Foertsch, J. H.: J. Oklahoma Med. Assn. *57*:449 (Oct.) 1964. 3. Paul, H. E., and Paul, M. F.: The Nitrofurans—Chemotherapeutic Properties, in Schnitzer, R. J., and Hawking, F. (Eds.) Experimental Chemotherapy, Vol. 2, New York, Academic Press, 1964.

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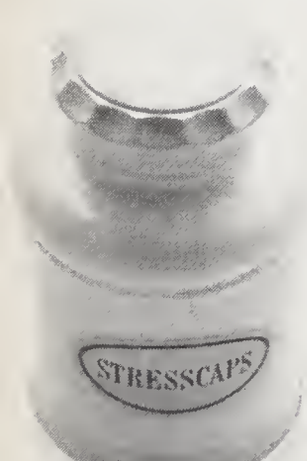

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Brief Summary

Indications: Use Erythrocin-Sulfas in infections more susceptible to the combination than to either agent alone. These are usually found in urinary, lower respiratory tract, and chronic ear infections.

Contraindications: Known sensitivity to erythromycin or sulfonamides. Because of the possibility of kernicterus with sulfonamides, do not use in pregnancy at term, premature or new born infants.

Warnings: As with other forms of sulfonamide therapy, carefully evaluate patients with liver or kidney damage, urinary obstruction, or blood dyscrasia. Deaths have been reported from hypersensitivity reactions and blood dyscrasias following use of sulfonamides. Perform blood counts and liver and kidney function tests if used repeatedly at close intervals or for long periods.

Precautions: Use sulfonamides with caution in patients with a history of allergy. Assure adequate fluid intake to prevent crystalluria and institute alkali therapy if indicated.

Adverse Reactions: Sulfonamide therapy may be associated with headache, nausea, vomiting, urticaria, diarrhea, hepatitis, pancreatitis, blood dyscrasias, neuropathy, drug fever, skin rash, injection of the conjunctiva and sclera, petechiae, purpura, hematuria and crystalluria.

Side effects due to erythromycin are infrequent, but occasional abdominal discomfort, nausea, or vomiting, urticaria and other skin rashes may occur.

If a reaction or overgrowth of nonsusceptible organisms occurs, withdraw the drug.

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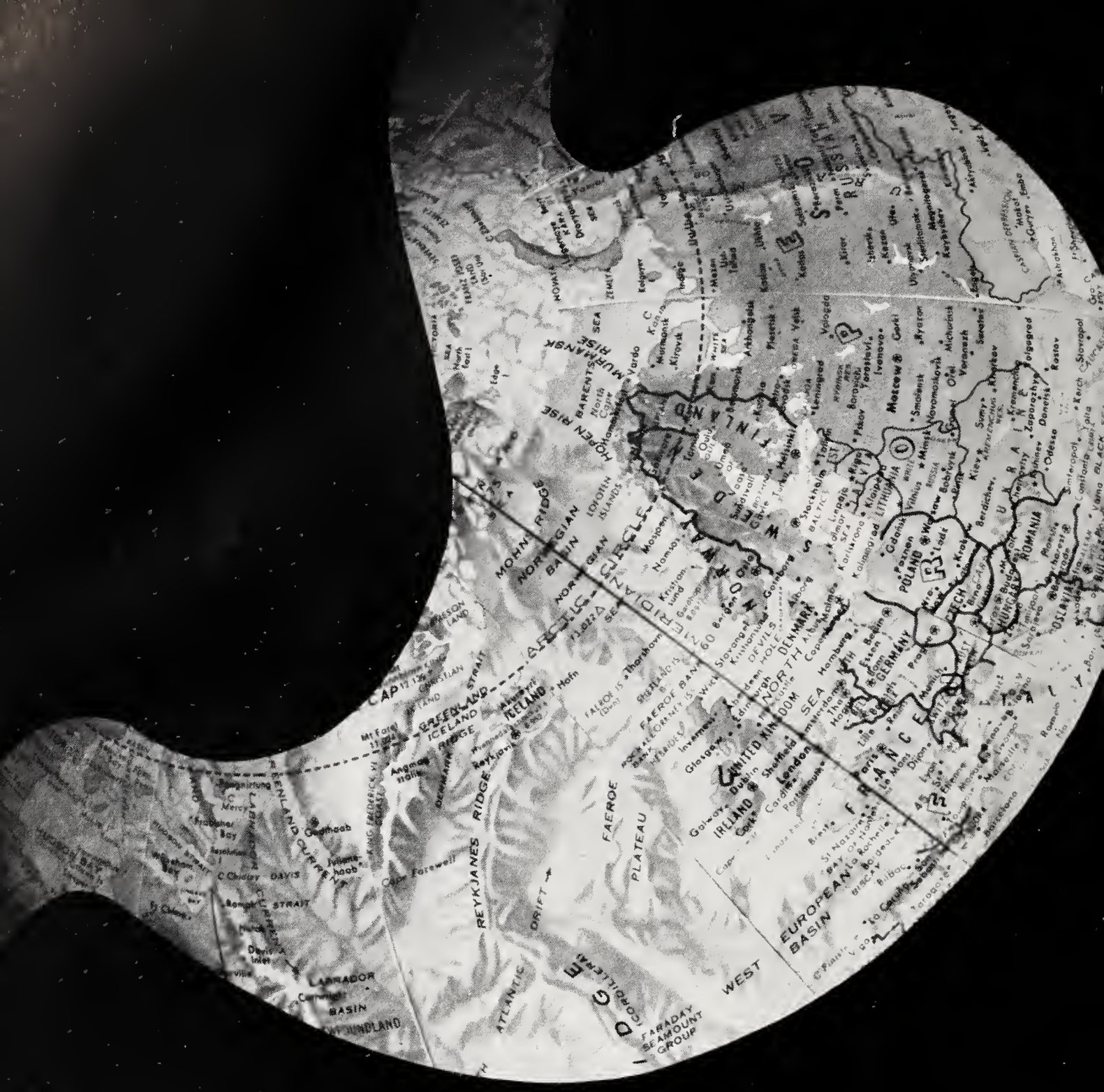

The "Socio-geographic" mystery

Why is one man's gastric ulcer another man's duodenal?

Geographic variation in the *incidence* of peptic ulcer is a familiar fact. But the proclivity of certain *kinds* of ulcer for certain geographic areas is a recently recognized phenomenon.[1,2]

For example, in one particular Norwegian fishing village there is a tendency for patients to develop a gastric ulcer; anywhere else in Norway, ulcers are usually duodenal. Peruvians high in the Andes have more gastric ulcers than their compatriots in the lowlands. Why? Nobody knows.

Social variations, too. Even in the same geographic areas there are interesting variations. An Englishman's ulcer depends on his social standing—professional men suffer with duodenal ulcers, while workingmen have more of the gastric variety. In southern India the pattern is reversed. Here, duodenal ulcers are common among laborers and agricultural workers and rare among the upper classes.

Investigators are exploring every possible theoretical avenue in their search for the cause of peptic ulcer. Of all the factors implicated in ulcerogenesis, the one that is generally acknowledged to be of primary importance is hypersecretion of gastric acid.[3-8] Or, as one author states it: "The medical management of peptic ulcer pharmacologically is, in the final analysis, concerned largely with the effective inhibition of peptic activity."[3]

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Contraindications: Glaucoma, urinary bladder neck obstruction, pyloric obstruction, stenosis with significant gastric retention, prostatic hypertrophy, duodenal obstruction, cardiospasm (megaesophagus), and achalasia of the esophagus, and in the case of Robinul-PH Forte, sensitivity to phenobarbital.
Precautions: Administer with caution in the presence of incipient glaucoma.
Adverse Reactions: Dryness of the mouth, blurred vision, urinary difficulties, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.
Dosage: Dosage should be adjusted according to individual patient response. Average and maximum recommended dose is 1 tablet 3 times a day: in the a.m., early p.m., and at bedtime. *See product literature for full prescribing information.*
Supply: Robinul (glycopyrrolate 1 mg.); Robinul Forte (glycopyrrolate 2 mg.); Robinul-PH (glycopyrrolate 1 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming); Robinul-PH Forte (glycopyrrolate 2 mg.) with phenobarbital 16.2 mg. (Warning: May be habit-forming.) In bottles of 100 and 500 scored tablets.
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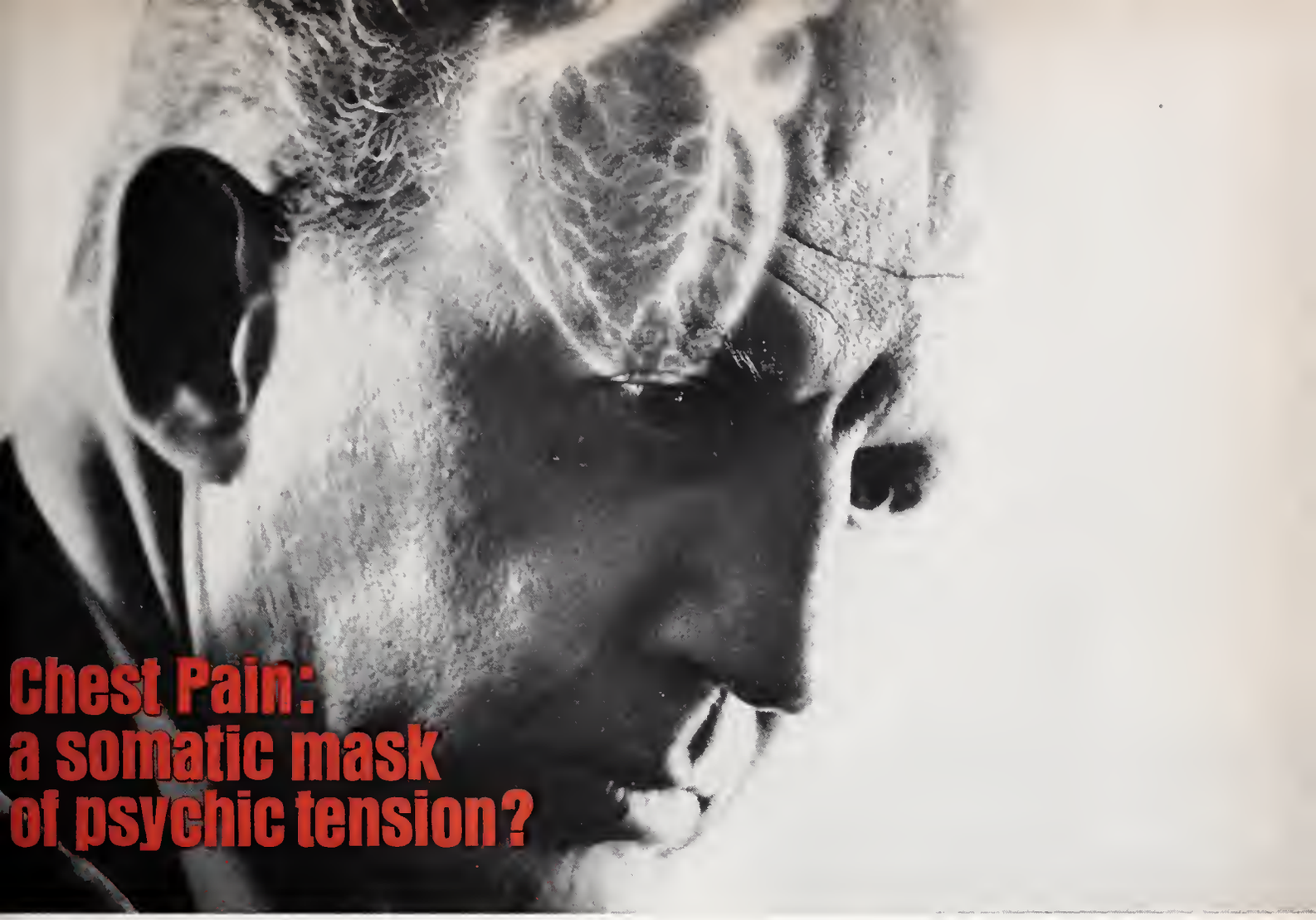

Chest Pain: a somatic mask of psychic tension?

"Heart symptoms"—chest pain, tachycardia, arrhythmia—invariably alarm and preoccupy the patient, though they may be completely without organic basis. Such symptoms often are somatic masks of psychic tension, arising from constant encounters with stressful situations.

When the problem is diagnosed as emotionally produced, consider Valium (diazepam) as adjunctive therapy. Valium (diazepam) acts rapidly to calm the patient, to reduce his psychic tension and relieve associated cardiovascular complaints.

NEUROTIC FATIGUE—the chronic tiredness resulting from emotional strain which so often accompanies psychogenic "heart" symptoms—also can be alleviated by this highly useful agent. Valium (diazepam) often achieves results where other psychotherapeutic agents have failed.

Valium (diazepam) is generally well tolerated, and usually does not impair mental acuity or ability to function. If side effects such as ataxia and drowsiness occur, they usually disappear with dosage adjustment.

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

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Physician-Patient Relations— Your Campaign and Mine

THROUGH THE combined efforts of the American Medical Association and the various state and local medical societies, there is underway a nationwide effort to emphasize and publicize the subject of physician-patient relations.

The key goal of the campaign is to teach the individual physician and impress upon him the need for positive action. Nationally, the physician's image among his patients has slipped because of the pressing demands being placed upon his medical practice. If each physician can be encouraged to devote a few minutes each day toward practicing a few simple public relations techniques that show his patients that he has a personal—as well as professional—interest in their welfare, the campaign is certain to succeed. Both the physician and his patient will gain.

It is easy and too often fashionable to complain that the poor image of medicine is the fault of the AMA. We tend to forget—or refuse to acknowledge—that medicine's public relations start with the doctor and his patient. We are inclined to overlook the basic truth that, in the last analysis, an individual's opinion of the medical profession usually is colored by his experiences with an individual doctor.

You have heard it said many times, and have probably made the statement yourself, that people like their individual doctors, but they generally dislike doctors as a group, or the medical profession as a whole, particularly the AMA.

I think that it is time that we, as individual physicians, reevaluate this statement. In fact, it is possible that just the opposite may be true. It is difficult for me to believe that people dislike General Motors because it is a large, powerful corporation which exerts a great influence over the daily lives of millions of people. It is more likely that people dislike this or any other company because of an unpleasant experience with an individual dealer who represents the parent organization. It is just as likely that patients become disenchanted with medical organizations because of an unhappy experience with some member of the profession.

Today, I think that we can safely say that most doctors are busy and that some are truly overworked. The full appointment sheets reflect the public's demand for more health care, rather than a popularity vote. Many physicians, however, using this as a guidepost, may honestly believe that they have excellent rapport with their patients. It might come as a surprise that many of their patients do not share this opinion.

Some of the items about which patients complained most in a recent survey were (1) that we are too busy to give personal attention, (2) we frequently make people wait unnecessarily for service, (3) we are too difficult to see, (4) we tend to conceal some fact we shouldn't, (5) we often charge more than the patient expected, and (6) we fail to make home calls. These are public relations problems which belong fundamentally to the individual doctor and not to the county and state societies or to the AMA. When, however, the individual performs poorly in one of these categories, it reflects on the entire structure of medical society organization.

How, then, can we as practicing physicians improve this vital relationship with our patients? We should begin when the patient first calls or walks into the reception room. It has been said many times that a physician's public relations depends to a great extent upon the impression his receptionist and other personnel in the office make upon the patient. In this regard, it is urged that your assistant become a member of Texas Medical Assistants Association and the American Association of Medical Assistants. These organizations have achieved considerable professional status for their members during the past several years. They provide an opportunity for your assistant to improve and broaden her services both to the profession and to the public.

We may also strive to improve our public relations with the patient by working on an appointment basis when possible and by reducing the waiting time of the patient, discussing fees more openly and more freely, arranging time off so that our practices are adequately covered at all times, taking more time to explain to the patient what his medical problem is and what we expect from the treatment we prescribe, and taking part in local civic, church, and school activities.

In essence, this reflects one thing—more concern for the patient as an individual. I have no doubt that if we as practicing physicians will present a strong public relations program in our daily contact with patients, the leadership and trained staffs at our headquarters offices can improve and protect our image at the national and state levels.

—JOSEPH T. AINSWORTH, M.D.,
Houston, Chairman,
Council on Public Relations
and Public Service.

Pancreatic Neoplasms Call For Early Diagnosis

THE INCIDENCE of carcinoma of the pancreas seems to be rising and it is being discovered more often in younger persons. A vigorous diagnostic approach and a generalized revision of current attitudes toward therapy have become obligatory.

Only 10 percent of the patients who are operated upon for carcinoma of the head of the pancreas are suitable candidates for radical operations; of these, only 13 percent survive for five years. In an attempt to improve these statistics, a few persons have cautiously suggested that total pancreatectomy may be a better surgical procedure than the standardized Whipple's operation. With total pancreatectomy, there is apparently minimal risk and morbidity actually is lessened.[1] Replacement of insulin with artificial preparations has not proved to be the problem it once was considered, and the newer oral enzymes have served adequately to correct maldigestion due to lack of pancreatic enzymes. Such vigorous treatment demands early diagnosis.

Points to be emphasized in obtaining the medical history of a patient with possible pancreatic disease:

1. The presence of pain which radiates into the back and which often may be relieved by flexing the body.

2. The recent onset of diabetes mellitus, or a positive response to the glucose tolerance test. In the absence of a family history of diabetes, these findings should cause the physician to suspect pancreatic disease and should aid early diagnosis.

3. The physical examination usually is unrewarding in establishing an early diagnosis.

4. Recently, a systolic bruit in the left hypochondrium has been described in a series

of patients with carcinoma of the body or tail of the pancreas, or in both areas.[2] Apparently, the bruit results from compression of the splenic artery by the tumor.

At present, pancreatic cancer may remain undiagnosed at a potentially curable stage. However, since Elman's[3] correlation of an elevated level of serum amylase with pancreatitis in 1937, the diagnosis of acute inflammatory disease of the pancreas has been facilitated. The simple expedient of measuring urinary amylase in units excreted per hour may detect elevation of amylase over a longer period of time and in about twice as many patients. The course of serum amylase would appear to parallel that of serum lipase. To differentiate chronic inflammation from carcinoma of the pancreas remains the *sine qua non* of the diagnostic problem.

The clinical laboratory thus becomes important in the evaluation of chronic pancreatic disease. Isolated serum enzyme tests probably are worthless as well as misleading and evocative tests for serum enzymes are not specific enough for a definite diagnosis culminating in an exploratory operation.

As inconvenient and unpleasant as duodenal intubation may be, we believe this procedure must be advanced as the single best method for determining early pancreatic dysfunction and disease. The pancreatic juice must be assessed after stimulation, and the parameters of usefulness appear to be:

1. The flow rate and the maximum bicarbonate concentration attainable after intravenous injection of secretin.
2. The concentration of lipase in the duodenal juice after pancreozymin.
3. The careful cytologic evaluation of pancreatic sediment. The latter procedure provides the only pathognomonic findings (when positive) of carcinoma. However, the normal pancreas does not exfoliate recognizable cells, and only a few cells are exfoliated during malignant invasion. About one-half of known pancreatic carcinomas will allow diagnostic (positive) cytologic examinations. Special training in the techniques of collecting, staining, and evaluating the final product are essential. Add to these criteria a minimum scanning for each examination, and it is a small wonder that, in general, cytopathologists are hardly enthusiastic over the procedure. Tetracycline fluorescence, proposed as a simpler method for identifying malignant cells, has the disadvantage of providing a significant number of false-positive findings.

Selective abdominal angiography, using a two-artery technique, has been reported capable of demonstrating tumors in the pancreas.[4] This procedure has been considered safe, although not simple; however it is apparently of no value in early diagnosis. Likewise, transverse laminal roentgenographic studies after retroperitoneal injection of carbon dioxide are of value only in advanced lesions.

Radioisotope photoscanning, using selenium methionine Se 75, has been discussed in recent months. The procedure is costly; the methodology is involved; and, again, the results seems to reveal only the presence of advanced disease. Eventually, other techniques of visualization with radioactive isotopes or with contrast dyes may replace this technique.

Even when definitely malignant cells can be washed out of the pancreas, our experience and that of others has been that the surgeon has difficulty identifying the lesion when it is at an early stage. Gross findings at operation (such as a mass in the head of the pancreas, distention of the common duct or the duct of Wirsung, or sclerosis of the distal pancreas) have been claimed to provide evidence for radical surgical treatment without histologic proof; yet, that this is an early stage of pancreatic cancer is hardly obvious. During a surgical procedure, pancreatic biopsy (searching for carcinoma) is only accurate, at best, 66 percent of the time. The biopsy carries a morbidity value of greater than 9 percent, and a mortality rate of more than 4 percent.[5] This evidence pleads for physicians to search more diligently for a diagnosis that may be elusive and often may be misleading until the time for hope has passed.

—J. Arnold Bargen, M.D., and
Dennis K. Wentz, M.D.,
Temple.

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Disaster Medical Care Depends On Local Practicing Physicians

THE TEXAS State Department of Health has been made responsible for providing emergency medical and health services for the people of Texas *in the event of a large scale disaster*. The public health problems brought on by a major disaster would be dealt with primarily by public health personnel; however, disaster medical care—the actual care and treatment of casualties—can only be provided by private physicians.

For this reason, the state health department's Division of Disaster Health and Medical Services is working in close cooperation with the Texas Medical Association's Committee on Disaster Medical Care by assisting private physicians in creating a statewide organization of medical and paramedical personnel and facilities so that they might function as effectively as possible in their most vital role in the total disaster relief effort.

This statewide medical organization is consistent with the official State Civil Defense Organization, and is legally recognized as a part of it.

Upon the declaration of an emergency the Commissioner of Health will automatically become the Director of Emergency Medical and Health Services. In that capacity he will be legally responsible for medical care and health services in Texas during the emergency.

The medical care aspects will be delegated to the Associate Director who, as the chairman of the Texas Medical Association's Committee on Disaster Medical Care, is providing guidance and assistance in the development of a statewide plan for disaster medical care. For purposes of administration, the statewide emergency organization is divided into 17 district areas, these being the same districts now utilized by the Department of Public Safety. This provides needed legal structure and basic communications facilities.

In each of these districts, a physician engaged in the private practice of medicine has accepted the position of district director of disaster medical care. These official appointments are made by the Commissioner of Health upon the recommendation of the TMA Committee on Disaster Medical Care. Alternate appointments have also been made.

The broad responsibility of these district directors is to develop the disaster medical care program within their districts. To accomplish this, physicians at the local level are appointed to work with the district director, and they are assisted by field personnel from the state health department.

The goal of this organization is a practical, workable plan to provide the best possible medical care for the people of Texas in disaster situations, be they natural or man-made. Success depends upon the willingness of the individual private physician to accept his role in planning for, and participating in, the medical care of disaster's casualties.

—BEN A. MERRICK, M.D., Dallas,
Chairman, Committee on
Disaster Medical Care.

In English: Where Does It Hurt?

Travelers who become ill in a country where the language is unfamiliar may obtain valuable and comforting help by phoning one of the 140 offices of the International Association for Medical Assistance to Travelers. This organization offers the tourist a choice of English-speaking physicians, most of whom were trained in the United States, Canada, or Great Britain.

IAMAT operates 24 hours a day in more than 50 countries, and membership cards, listing the addresses and telephone numbers of all centers, are available free of charge. Prospective travelers may address inquiries to the main office, 11 Via Parigi, Rome, or to a branch office at 11 Temperance Street, Toronto, Canada.—*Schering Science Bulletin.*

Family Practice—A New Specialty

The House of Delegates at the American Medical Association meeting late in November considered two important reports on medical education: The Citizens Commission on Graduate Medical Education (Millis Report) and the Ad Hoc Committee on Education for Family Practice.

Both these reports view with alarm the decreasing number of general practitioners in the medical census. Both reports also stress the need for a well trained "primary physician," "comprehensive-care physician," "first-contact physician," or "family physician." Such a physician would complete three to four years of postgraduate training in medicine, pediatrics, psychiatry, minor surgery, and obstetrics and gynecology. Upon finishing the program, he would be board eligible for the specialty of family practice.

These reports echo the feelings of many leaders of the American Academy of General Practice that a board of family practice would encourage many medical students to enter family practice.

Programs of course must be developed, curriculums in medical schools changed, educators made available, and the content of the residency agreed upon before this new specialist will arrive on the medical scene.

The Texas Medical Association recognizes our need for more family doctors and is presently working with the medical schools to encourage programs which will expose students to family practice.

And for you and your family, may I wish a Merry Christmas and a Happy New Year.

Sincerely,

James D Murphy M.D.

J. D. Murphy, M.D., President

When the road back to good health is long and expensive...

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meet the authors

WILLIAM J. NELSON, JR., is a postgraduate fellow at the National Hospital for Neurological Disease, London. Next year, he will begin private practice in El Paso. **Cryosurgical Destruction of Intracranial Neoplasms, p. 44.**

C. T. ASHWORTH is professor, Department of Pathology, the University of Texas Southwestern Medical School, Dallas. **Reappraisal of Whipple's Disease, p. 48.**

JAMES W. McBEE is chief of the gastroenterology section at the Houston VA Hospital and is assistant professor of medicine at Baylor University College of Medicine. **The Sprue Syndrome, p. 54.**

ROBERT S. NELSON is with the Department of Medicine, M. D. Anderson Hospital and Tumor Institute, Houston. **Evaluation of Gastrointestinal Malignant Neoplasia, p. 57.**

JOHN M. EIBAND and his coauthors are with the Department of Internal Medicine, Baylor University College of Medicine, Houston. **Some Newer Aspects of the Carcinoid Syndrome, p. 62.**

MAURICE C. ARCHER practices radiology in Fort Worth. **Old and New Methods of Therapy For Carcinoma of the Cervix; Report of 170 Cases, p. 72.**

JOHN A. WALL, ALAN L. KAPLAN, and **GAYNOR JANES,** Houston gynecologists associated with Baylor University College of Medicine, present a thorough review of the role of **Surgery in the Management of Carcinoma of the Cervix, p. 66.**

JAMES NEIL GILLIAM, former resident in internal medicine at Parkland Memorial Hospital, now practices in Goldthwaite. He gives the case presentation of this month's **Clinicopathologic Conference, p. 82.**

Breast-feeding and the "modern mother"

Despite a mild resurgence of interest in the importance of breast-feeding a few years ago, many women today do not choose to nurse their young. This is for a variety of reasons—social, economic, cultural and sometimes medical. In such cases the physician's task is to find the most suitable means of preventing lactation and easing the pain of breast engorgement.

The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

However, factors other than effectiveness must also be considered. The agent selected should not interfere in any way with parturition, subsequent uterine involution and the restoration of normal ovarian cyclic function. Furthermore, it should not cause rebound breast engorgement or other manifestations of hormonal imbalance.

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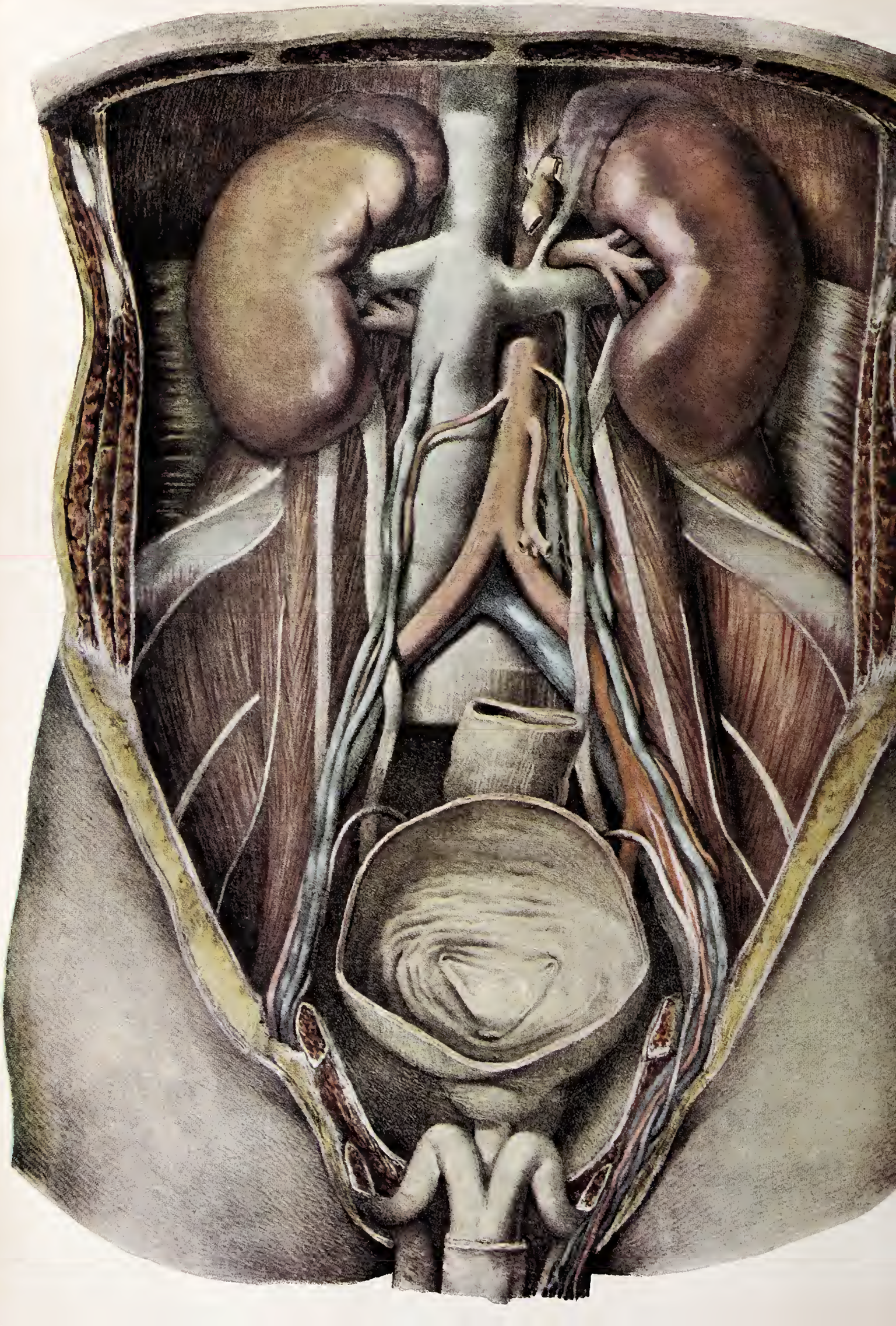

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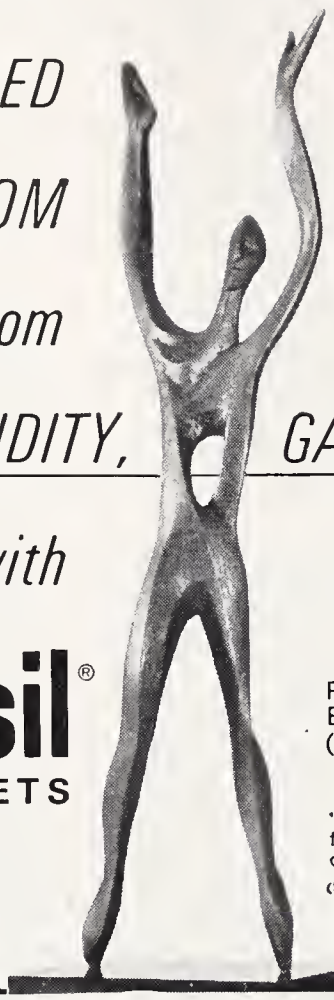

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1. Schleif, R. H.: J.A.PH.A., 46:179, 1957
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Cryosurgical Destruction Of Intracranial Neoplasms

W. J. Nelson, Jr., M.D.; R. B. Marshall, M.D.; and John E. Coe, M.D.

By inserting a cryoprobe through a burr hole into an intracranial tumor, the neoplasm can be frozen in situ and decompressed by the subsequent process of liquefaction necrosis. With refinement, the technique may be useful in cases that resist conventional therapy.

RESEARCH & INVESTIGATION

THE CURRENT state of the art of treatment of astrocytomas leaves much to be desired. Conventional surgical therapy, which often is difficult and time consuming, offers temporary palliation. Radiation therapy may or may not add to the results of surgical resection.

Intracranial neoplasms have been treated by cryosurgical destruction with surgical decompression and excision of the lesion by Rowbotham and associates.[1] Cooper[2, 3] also described techniques for freezing and excising the tumor mass. In extending the techniques described by these and other workers, we utilized a method of destroying the tumor by freezing in situ through a small burr hole. The frozen tissue was left in place and the process of liquefaction necrosis was allowed to decompress the tumor. It was recognized that postoperative edema might present a problem and we planned to attempt medical rather than surgical control of the resultant edema while the absorption process was going on.

In addition to this technique, we have performed cryodestruction in one patient through a conventional craniotomy flap using a specially designed four-pronged probe (Fig. 1) to produce a larger lesion than could be obtained with a single cannula.

"Closed" Cryodestruction of Tumor

This surgical technique has been used in two cases. In each case, an occipitoparietal astrocytoma had been removed previously by conventional craniotomy; case 1 was an astrocytoma grade 2, while case 2 was an astrocytoma grade 4. Each patient had a strongly positive radioactive brain scan (Fig. 2) and each showed clinical evidence of recurrence of the lesion. Arteriography also demonstrated the lesion (Fig. 3).

Initially, a burr hole was placed slightly eccentrically over the lesion, and a pressure transducer was placed between the brain and dura. The leads were brought out through the incision and attached to a Grass recorder for continuous operative and postoperative intracranial pressure recordings. Details of the continuous pressure recordings will be reported in another article.[4]

A second burr hole was then placed over the center of the lesion as determined by the

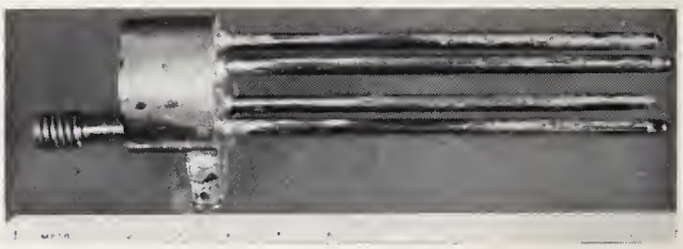

Fig. 1. Four-prong cryoprobe for destruction of large lesions.

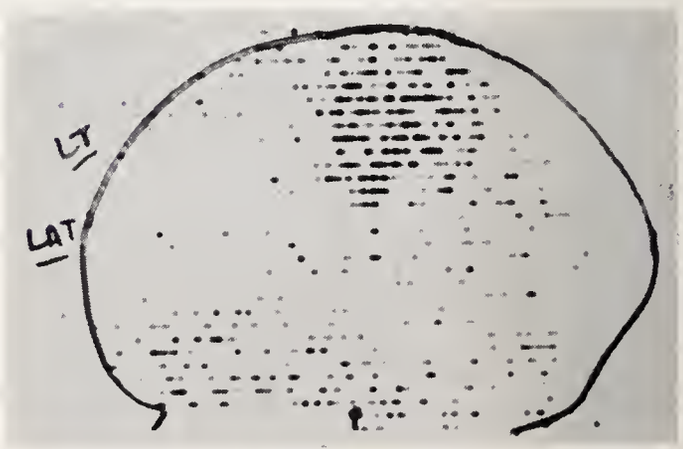

Fig. 2. Radioactive brain scan shows "hot" area of tumor.

radioactive scan and arteriography. The conventional 5 mm. cryoprobe (Fig. 4) was then inserted into the center of the lesion as judged from the scan and the arteriogram. Operative roentgenograms were then made to verify the placement of the probe. Liquid nitrogen at −195.8 C. was then passed through the probe for seven minutes in case 1 and five minutes in case 2. Tip temperature was monitored by thermistor (Fig. 5). After freezing, the area was allowed to thaw and the probe was removed. No attempt was made to remove any tissue or do any type of decompression, and the scalp was closed. While removing the transducer, one week postoperatively (case 2), a needle was inserted into the center of the lesion and liquid necrotic tissue was aspirated. This verified the fact that liquefaction necrosis was indeed taking place.

Results of Therapy

Prior to cryosurgery each patient was showing definite neurological signs of recurrence of the tumor, and each had positive arteriographic and radioactive scan evidence of tumor. It had been 54 months since previous surgery in case 1 and six months since surgery in case 2. This is sufficient time to assure that the preoperative positive scans were not the result of postsurgical changes.

In case 1 there was no evidence of increased intracranial pressure before cryodestruction, while in case 2 there was evidence of increased pressure and the arteriographic study indicated a shift across the midline.

The cryosurgical procedure produced alteration in the physiologic state of each patient as evidenced by immediate postopera-

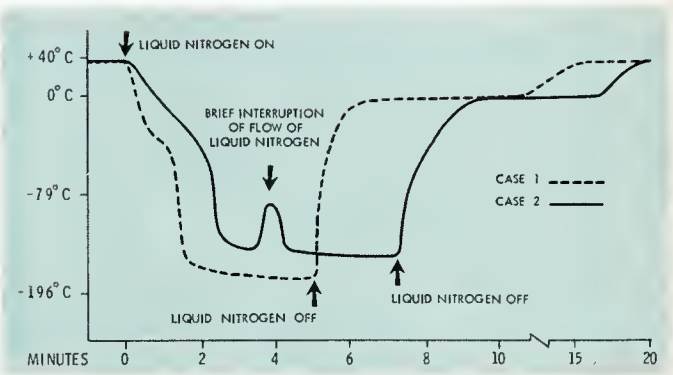

Fig. 5. Tip temperatures during cryodestruction.

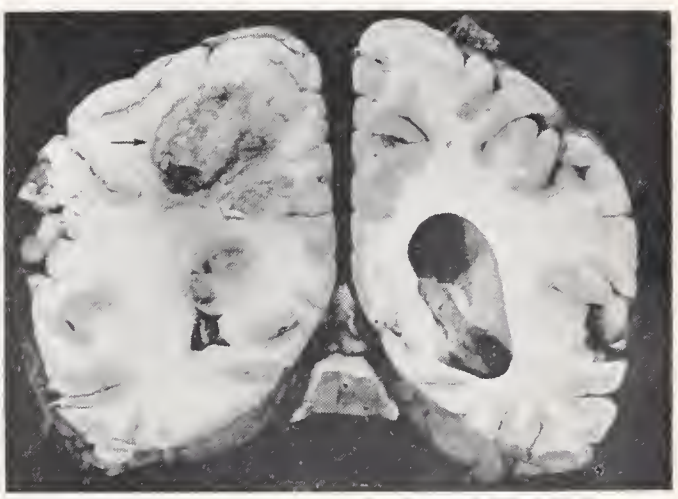

Fig. 6. Coronal section of brain, case 2. Arrow indicates area of cryodestruction in center of tumor.

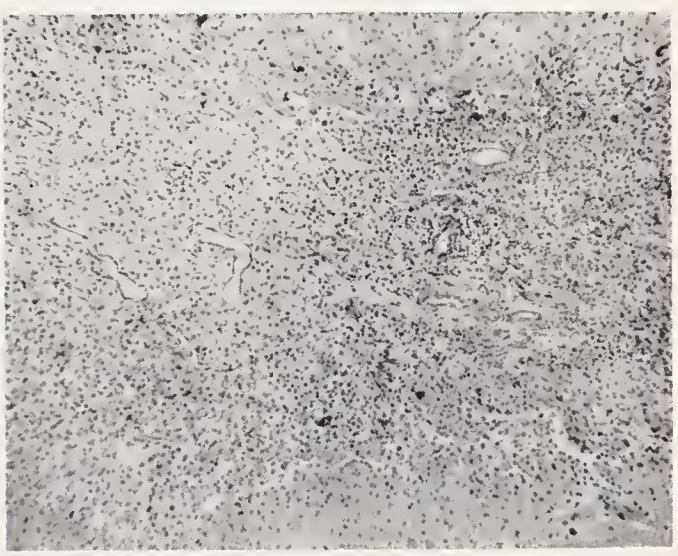

Fig. 7. Photomicrograph of tissue at the periphery of the area of cryodestruction. The necrotic area is at the bottom, and the reactive zone is above the area of necrosis.

Fig. 3. Arteriogram showing abnormal vessels in tumor area and evidence of previous craniotomy.

Fig. 4. Insulated 5 mm. cryoprobe with thermistor tip.

tive decrease in their state of responsiveness. Both required intravenous support immediately after surgery, and nasogastric tube feeding until their level of consciousness returned to a preoperative level in approximately three weeks.

This alteration of physiologic state was not unexpected, since it has been shown by other investigators, including Klatzo and associates,[5] that edema will occur in the brain around areas of cold injury.

Large doses of steroids (Decadron, 4 mg. every six hours) were administered during this two- to three-week period and mild total body hypothermia (94 to 96 F.) was maintained in an attempt to diminish edema. In addition, administration of urea during the first 28 hours after surgery was necessary in case 2, and at four and five days postoperatively in case 1.

Both patients gradually improved. In case 1, the patient surpassed his presurgical state in that he was more alert and responsive and oriented.

The second patient improved progressively for about four weeks postoperatively. His condition then began to deteriorate and he died six weeks after operation. The brain contained a sharply demarcated area of necrosis (2½ by 2½ by 3½ cm.) in the center of the tumor (Fig. 6). The tumor, however, was extensive and only about one-fourth of the total mass had been destroyed. Microscopically the frozen area and the surrounding tissue resembled a six-week-old infarct (Fig. 7).

Case 3 was a 56-year-old white man who was in a deep coma when he was first seen. Radioactive scan and arteriography indicated an extensive parietal lobe lesion with extension of tumor across the midline. A flap was turned over the tumor and the four-pronged cannula was inserted under direct vision into the center of the tumor. The lesion was frozen and was then excised, using the electrosurgical cutting scalpel to hollow out the frozen area, leaving a frozen crust at the periphery. The patient died 24 hours after surgery without regaining consciousness.

Discussion

The open cryosurgical technique used in case 3 offers certain clinical advantages in control of bleeding during surgical removal of tumors. In addition, the freezing process may be used to destroy an additional margin around the tumor which cannot safely be removed.

The technique has certain disadvantages at its present stage of development. The equipment is bulky and difficult to handle. The liquid nitrogen is expensive and may not be readily available in some areas.

We believe that our "closed" cryosurgical destruction is a more promising technique and deserves further development and evaluation.

The exact localization of the destructive lesion needs to be further refined, and it is thought that this can be done by adapting the technique of stereotactic biopsy described by Coe and others[6] to the placement of the lesion.

As pointed out by Coe and Ommaya,[7] the size of the freezing lesion is difficult to predict and control. We believe it is possible to construct a cooling cannula with side extruding thermistor probes to measure temperature at a distance from the center of the cannula. This would be structurally similar to the side extruding electrodes now commonly used in basal ganglia surgery. With the ability to measure temperature at a distance, the size of the lesion could be accurately controlled. It might also be feasible to produce multiple small lesions at two- to four-week intervals to destroy tumors too large for ablation by a single cryodestructive procedure.

Our two cases indicate that with hypothermia, urea, and steroid administration we can control the edema associated with cryodestructive procedures without surgical decompression.

Continuous monitoring of intracranial pressure transducer placed in the subdural space contributes to the accuracy with which intracranial pressure can be measured and controlled.

Conclusions

Cryodestruction of intracranial tumors by insertion of a cannula through a burr hole offers a technique which, with refinement, may eventually give good palliation and possible cure of neoplasms which are resistant to surgical and radiation therapy techniques.

Postoperative edema can be controlled without surgical decompression.

Much work is necessary to refine the accuracy of lesion placement and size.

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This study is supported by the Mrs. Francis I. Zoch Fellowship Grant of the American Cancer Society.

Quack's Giftbox: Emptiness Wrapped with Art

"Why do people go to quacks? They go seeking that which they have not found elsewhere." Although they may say that they are searching for a cure, they are perhaps looking for a kind, sympathetic, and concerned friend.

"The quack has nothing to sell but himself, but he sells himself well. Could the ethical practitioner of medicine learn a lesson from this?"

Dr. William H. Gordon, Lubbock, in examining the reasons why people go to quacks, posed this question during the Third National Congress on Medical Quackery sponsored by the AMA and the National Health Council. His comments and those of other participants were published in the December issue of *Today's Health*.

The miracle seeker, the uninformed, the restless, and the straw-graspers are four types of patients who may easily be detoured from conventional to nonconventional care, he said, citing a study by Dr. Beatrice Cobb, Lubbock, former research psychologist at M.D. Anderson Hospital. Miracle seekers hope for "something good" to happen quickly; the uninformed, tragically, are just that. The restless may act on their attitude of "if you can't do it now, I'll go somewhere else," or they may be professional nonconformists who despise respectability and reason and feel honor-bound to defend and patronize the minority view. Pathetic are the straw-graspers; when told that there is no cure for them, they frantically seek, and find, someone who will say it isn't so.

The physician of yesteryear had little to offer except his personal sympathy and understanding, Dr. Gordon said, but now the science of medicine tends to overshadow the art.

"I continue to believe," said Dr. Gordon, "that one can never practice good medicine without employing both the art and the science. It is essential to have a good rapport between patient and physician. When welded over years these strong bonds are seldom broken for the benefit of quackery. The physician has a good product to sell, but first he must sell himself . . . He should still employ the 'art' of medicine.

"What is the 'art of medicine?' I like to think that it is the milk of human kindness prescribed in such amounts as to allow the science of medicine to be swallowed painlessly. In my opinion, we need more of it."

Reappraisal of Whipple's Disease

C. T. Ashworth, M.D.

Small bacilli which are regularly present in lesions of Whipple's disease undergo phagocytosis, but leave within the macrophages an undigestible cell wall which imparts a foamy appearance. With antibiotic therapy, continued for an undetermined time, the bacilli and disease symptoms disappear.

REVIEW ARTICLES

WHIPPLE'S DISEASE is an uncommon disorder, but a reappraisal is appropriate because recent studies have provided evidence indicating a bacterial etiology, and because an effective means of treatment has now been adduced. In its untreated form the disease is usually stormy and progressive, and is generally fatal; but if adequately treated there is reason to believe that this usually relentless course can abate, and the disease can be cured or held in check.

Whipple described the disease in 1907[1] and suggested the name of intestinal lipodystrophy. His report was a thorough study of a theretofore completely unknown entity, and his observations of the clinical and morphological features can hardly be improved upon. Using a Levaditi stain he even described bacillus-like bodies in the lesion. This observation was not duplicated by subsequent observers until the advent of electron microscopic study of intestinal mucosa around 1960. In this interim, the disease known as intestinal lipodystrophy or Whipple's disease remained an enigma insofar as its etiology and pathogenesis were concerned, and randomly applied treatment appeared to be wholly ineffective.

Paulley,[2] Jorgensen,[3] and Chears and others[4] reported on the effectiveness of antibiotic and adrenal corticosteroid therapy in Whipple's disease and showed that patients could survive for many years with this form of therapy. These results led to suggestions of a possible infectious etiology for the disease. Beginning in 1961, reports of electron microscopic studies of the intestinal lesion of Whipple's disease began to appear, and bacilliform bodies were found consistently.

Clinical Findings

The clinical features have been described fully in several recent reports.[4-6] The disease occurs almost exclusively in men. It is more common in the fourth, fifth and sixth decades. Fleeting multiple joint pains usually precede the main onset of the disease by several months. The active stage is ushered in by fever, malaise, mild abdominal pain with postprandial distress, anorexia, and nausea and vomiting. In the majority of patients, diarrhea occurs during or shortly after the onset of these symptoms. In most cases the temperature does not exceed 102 F., but chills with temperature up to 105 F. may occur in a few. These symptoms persist with partial regressions for weeks or months. Weight loss, weakness, cramping abdominal pain, generalized lymphadenopathy, migratory joint pains, and malabsorption with steatorrhea and foul-smelling stools occur. Brown pigmentation of the skin in exposed areas is present in some cases. Fever generally continues but may be less severe than earlier in the course. Untreated, the patient usually becomes progressively weaker with inanition, persistent fever, and diarrhea, and he may die. In some cases, evidence of systemic dissemination is seen. Aside from involvement of upper abdominal lymph nodes, which are affected in practically all cases,

specific lesions may also be found in the heart, serous membranes, lungs, liver, spleen, and other organs.[7]

The distinctiveness of the combination of clinical findings enables one to suspect the diagnosis in about 50 percent of the cases before examining a biopsy specimen.[4] The availability of small biopsy specimens of intestinal mucosa by a safe peroral procedure[8] makes it possible to establish the diagnosis in practically all cases.

The Lesion

The ability to obtain biopsy specimens of the intestinal mucosa has enabled us to study the disease in earlier stages than was possible previously. Information derived from this source and from autopsy material has led to a clearer understanding of the nature of the changes. We have examined the intestinal mucosa in three cases[9, 10] and have studied autopsy material from four other cases of Whipple's disease. The following observations were made.

Tissues from the small intestine reveal the lesions in almost every case. This is because the involvement begins in the duodenum and affects a major portion of the small intestine. Lesions may also be present in the stomach. The intestinal villi are present, but are thickened and clubbed. The lamina propria contains numerous, closely packed, large, pale macrophages (Fig. 1). These macrophages are diagnostic. They have foamy, pale cytoplasm which stains strongly with PAS (periodic acid, Schiff)[11] (Fig. 2), alcian blue, and colloidal iron, indicating their mucopolysaccharide nature. Lipid droplets are often present in the lamina propria, and the lymphatics may be dilated and contain lipid droplets, but, as Whipple originally observed, the pale foaminess of the macrophages is not due to lipid. Instead, as first shown clearly by Sieracki,[12] it is due to individual sickle-shaped mucopolysaccharide-containing bodies. Regional lymph nodes contain numerous lipid droplets, and their lymph spaces are distended. The lymph nodes also contain the foamy PAS-positive macrophages. While PAS-positive macrophages may be found in small numbers in the intestinal mucosa in other conditions, their presence in large numbers and content of the typical sickle-shaped PAS-positive bodies (pale bodies in hematoxylin and eosin stains) are characteristic of Whipple's disease. In active stages of the disease neutrophils may be present in moderate numbers among the macrophages. No localized granulomatous areas, typical granulation tissue, ulceration, fibrosis, or distinct areas of necrosis or severe tissue injury occur. The inflammatory response is a mild one resembling that which occurs in inflammation caused by comparatively mildly acting agents of tissue injury.

The first description of the lesion as seen with an electron microscope, in which rod-shaped bodies were noted, was in 1960 by Cohen and others.[13] However, these observers did not interpret the bodies as bacilli. Since

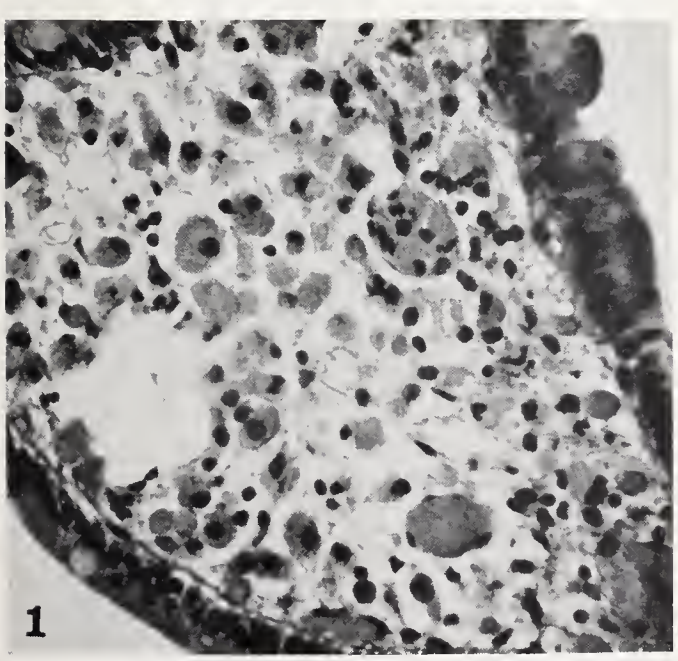


Fig. 1. Photamicrograph of intestinal mucosal biopsy specimen in Whipple's disease. Shown is an enlarged villus, in the lamina propria of which are numerous large, foamy, pole macrophages. Hematoxylin-eosin stain. X 430

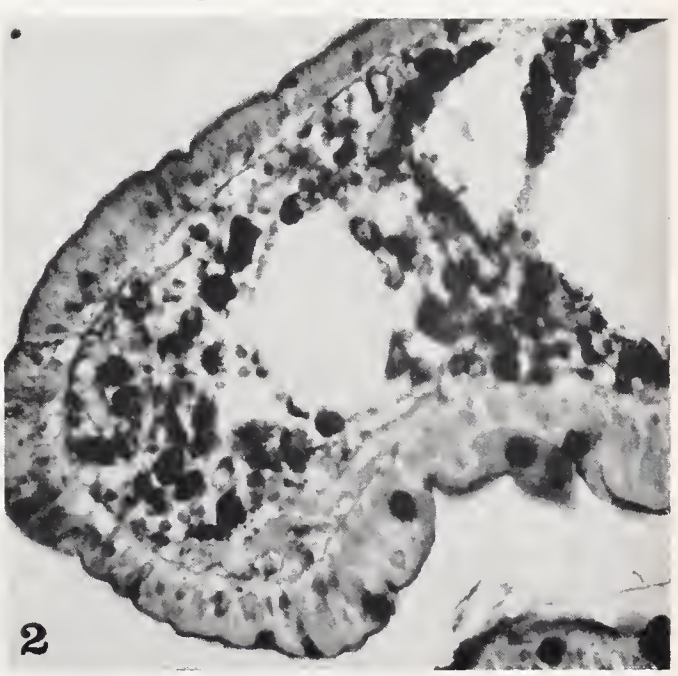
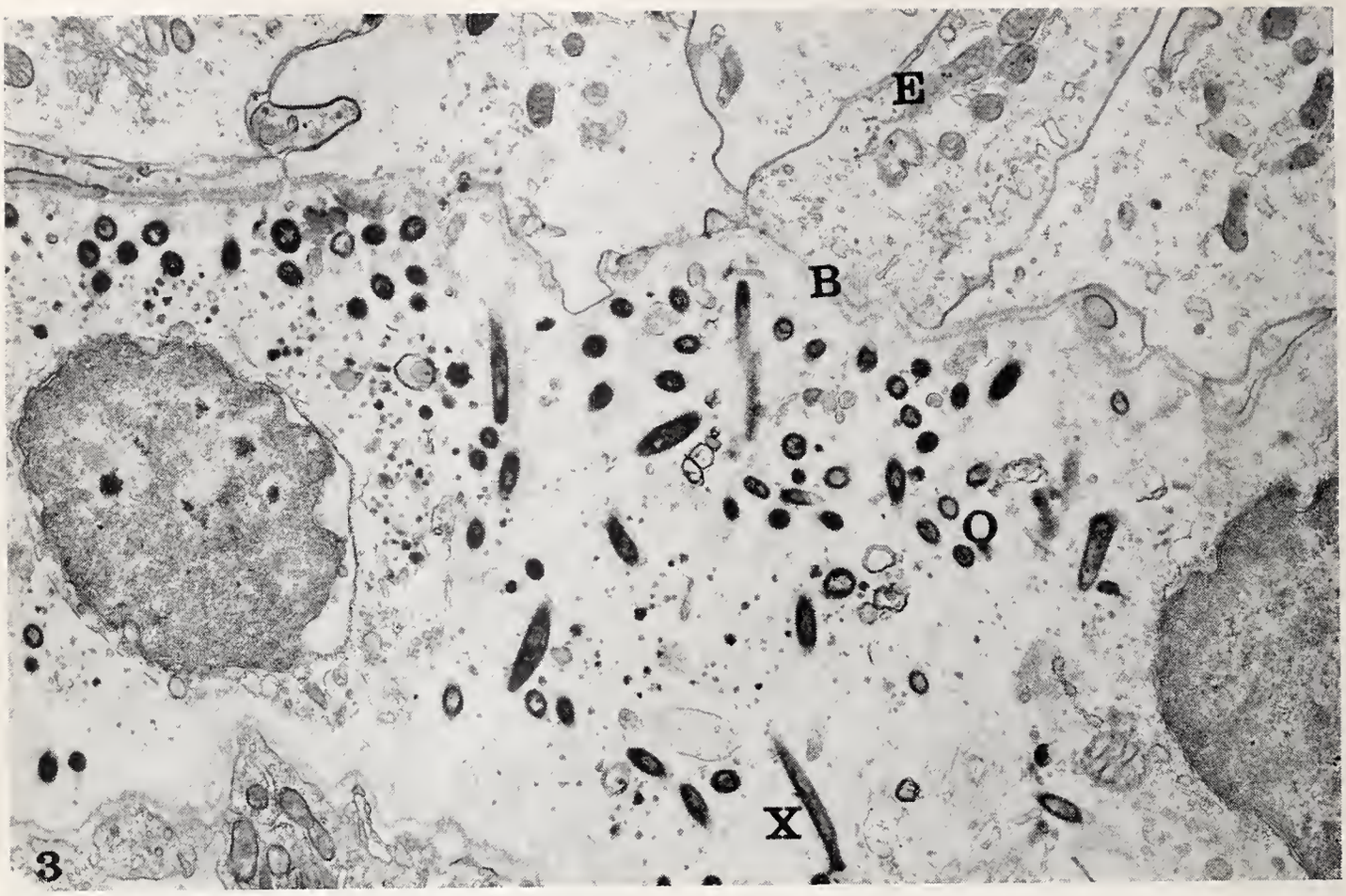


Fig. 2. In this section of intestinal mucosa (periodic acid-Schiff stain), the large macrophages contain strongly PAS-positive material, which in some areas can be seen as separate oval or sickle-shaped particles. X 430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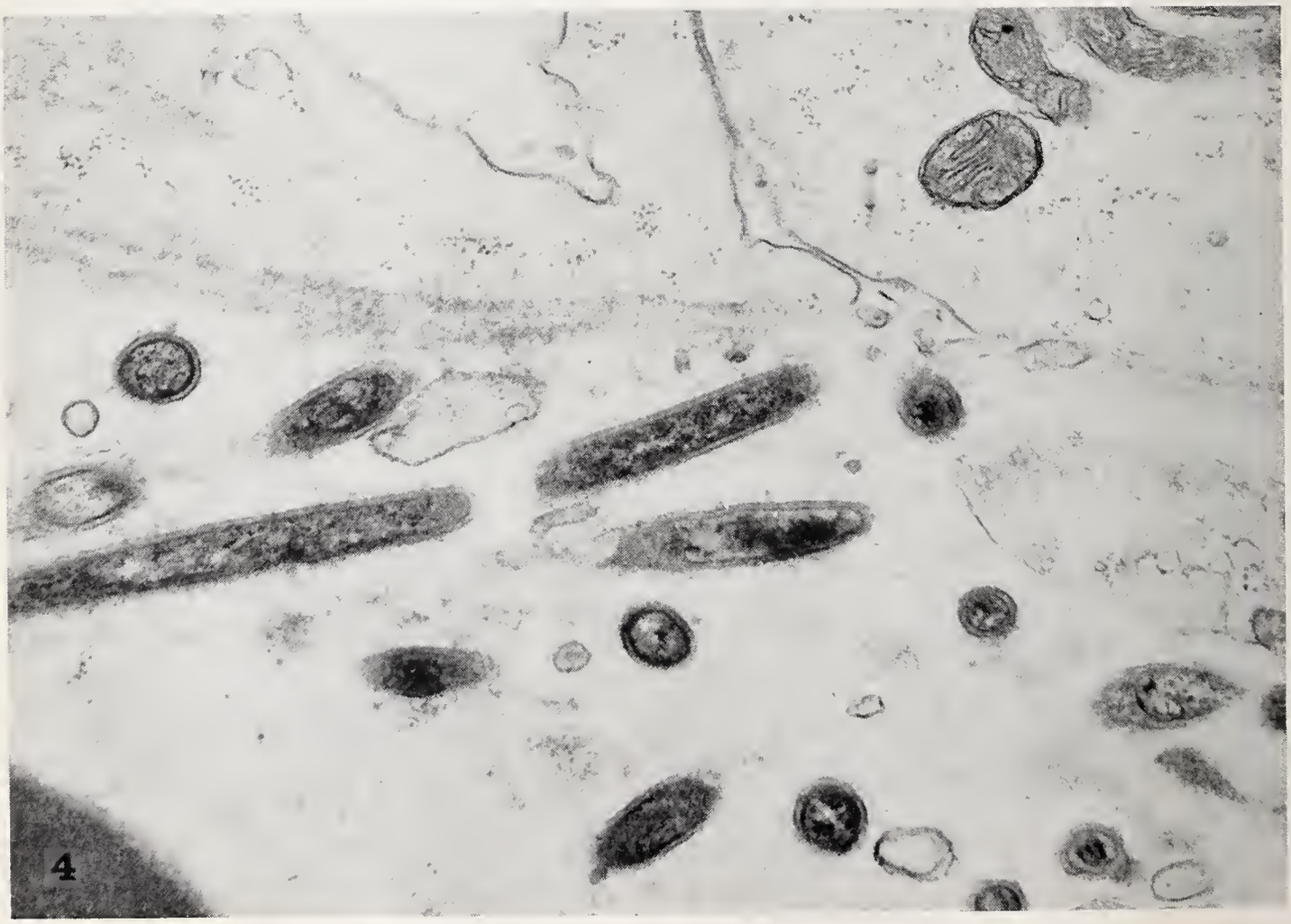
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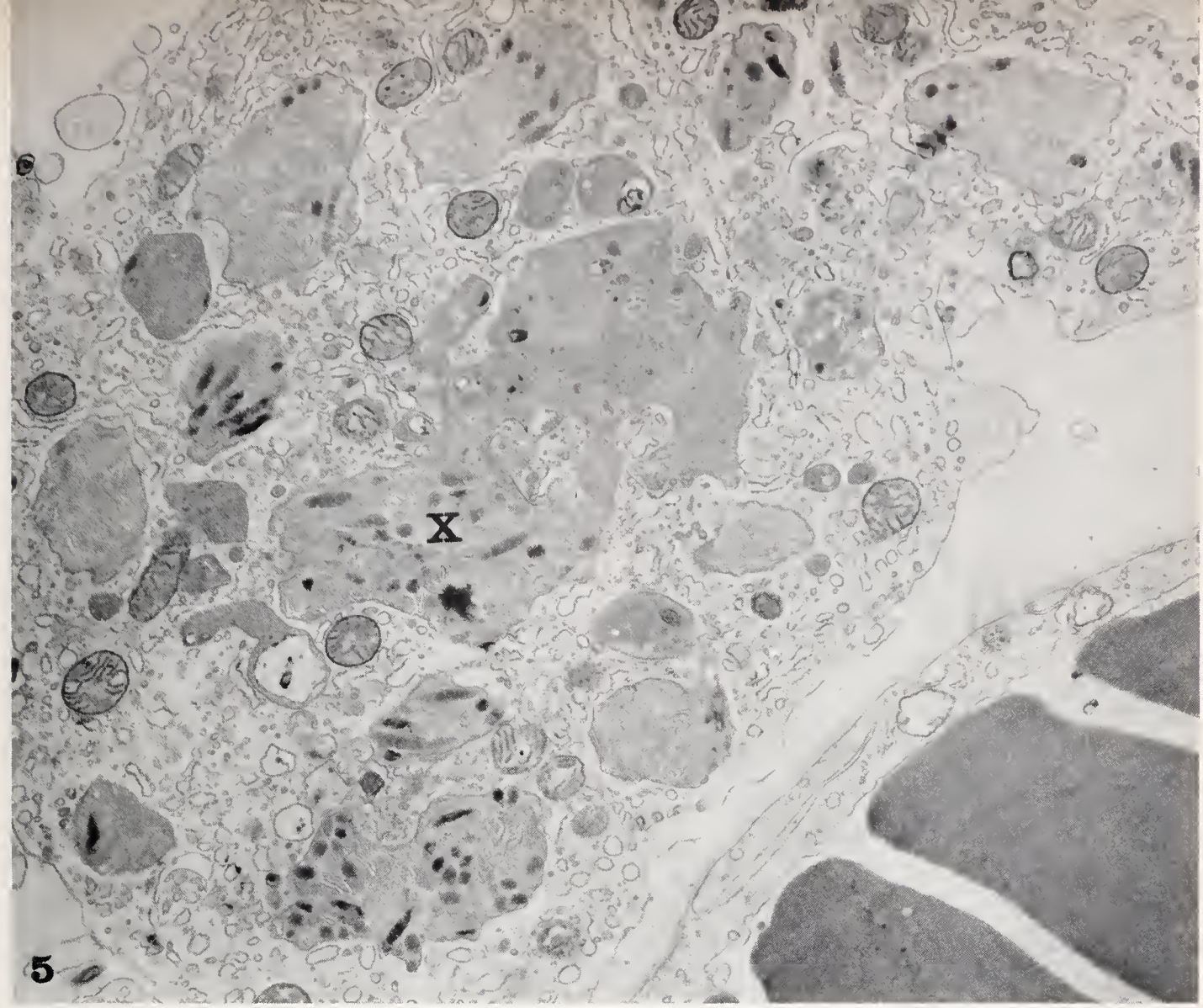

Fig. 3. Electron micrograph of a section of intestinal mucosa shows portions of intestinal columnar epithelial cells (E) and the basement membrane (B). In the underlying lamina propria are numerous minute bacilli; some are sectioned longitudinally (X) and others are sectioned transversely (0). X 14,000

Fig. 4. This electron micrograph shows the bacilli at higher magnification. The outer cell walls and internal filamentous structure of these bacteria can be seen. X 38,000

Fig. 5. Electron micrograph showing one of the PAS-positive macrophages in the lamina propria. Throughout the cell are numerous dense bodies (X) containing partially digested bacilli and residual bacterial cell walls from previously digested bacilli. X 14,000

1961, when our first case was reported and the bodies were found and interpreted as small bacilli,[9] at least eight additional untreated cases have been reported in which the distinctive rod-shaped bodies, about 0.2μ wide and up to 2μ long, were demonstrated by electron microscopy and interpreted as minute bacilli[10, 14-18] (Fig. 3). The bacilliform agents present a cell wall, which stains with periodic acid-methenamine silver,[10] a plasma membrane, and dense and filamentous material arranged within the body in a manner entirely characteristic of bacteria (Fig. 4). The bodies have been seen in the process of binary fission and in the stages of phagocytosis by macrophages and neutrophils with intracellular digestion. Retrospective studies of Gram-stained sections have disclosed minute bacilli corresponding to the bodies more readily visible with the electron microscope.

With electron microscopy the large, pale macrophages can be seen to have phagocytized vast numbers of the minute bacilli (Fig. 5). In active cases intact bacilli are present in the foam cells, but even in these cases and especially in the inactive stages, the protoplasm of most of the bacteria has been digested, leaving only the cell walls of the bacilli. These bacterial cell walls apparently are indigestible by macrophages, and they collect in sickle-shaped packets, which impart the pale foaminess to the macrophages as seen with light microscopy.

Etiology

The bacillus-like bodies present all of the morphological features of bacilli. When first observed, microscopists were less familiar with the fine structure of bacteria, and there

was considerable reluctance to accept their bacillary nature. Comparison with known bacteria[10] shows their identity, and practically all recent observers have accepted the rod-shaped bodies as minute bacilli. Their stainability with Gram and Giemsa stains aids in this identification. Several concerted attempts have been made to culture the organisms,[10, 19] but to date most of these have been unsuccessful, and animal inoculations have likewise given negative results. Kok and others[20] isolated *Hemophilus* organisms and diphtheroids from the intestine of one patient, and Caroli and others[21] have isolated a diphtheroid from a lymph node of a patient with Whipple's disease. What relationship these may have to the etiologic agent remains to be determined.

There is considerable secondary evidence favoring the bacterial nature of the rod-shaped bodies. They disappear after antibiotic therapy, and during the time of their absence the patient is free from symptoms of the disease. With recurrence of symptoms after discontinuance of therapy they return in large numbers. Additional evidence is that the clinical nature of the disease suggests an infectious process. Thus, the onset with fever can best be explained on this basis.

Morphologic uniformity of the organisms has been a notable feature in all cases adequately studied. In the untreated stage these bodies have been invariably present and are of similar size and morphology. Even in cases reported from foreign countries[19, 21] these identical morphologic properties prevail. Thus, it appears that a highly specific organism type is involved. Smaller than common pathogens,[10] they are less than one-half the size of *Escherichia coli, Mycobacterium tuberculosis,* or *Proteus* organisms. In size and internal structure, we found these organisms to be more similar to *Listeria* than to any other bacterial species studied. It must be conceded, however, that the species has not been identified.

A relatively low degree of pathogenicity is suggested by the nature and progress of the lesions; although large numbers of organisms are present, there is no evidence of severe tissue injury or of marked inflammatory exudation. There is a striking similarity between the lesion of Whipple's disease and the human tissue response to leprosy. The large number of organisms, local accumulation of histiocytes, phagocytosis of bacteria, lack of tissue necrosis, and formation of foam cells as a result of retention of undigestible remnants of phagocytized organisms in macrophages are all features shared by the two diseases. These similarities extend only to the inflammatory response since the organisms in leprosy and in Whipple's disease are overtly different.

Treatment

Even before the discovery of bacilli in the lesions, Paulley,[2] Jorgensen,[3] and Chears and others[4] reported on the effectiveness of antibiotic therapy. They found that patients treated with antibiotics and adrenal corticosteroids could survive for many years. The discovery of bacilli in the lesions has strengthened this position, and has led to many therapeutic trials which have been generally successful.[9, 10, 15, 17, 18, 21] Tetracycline, 250 mg. four times daily, has almost uniformly produced prompt remission of symptoms. Streptomycin and Chloromycetin have also been effective in some cases. Corticosteroids administered concurrently appear to be beneficial and are usually employed. With such treatment, fever and diarrhea disappear and the patient gains weight. Bacilli disappear from the lesions within a few weeks after antibiotic therapy begins. When treatment stops or the dose is reduced to an ineffective level, fever and diarrhea usually return and the patient again loses weight. In several cases we determined that concurrent with this relapse, organisms return in the intestinal mucosa. However, in at least one case[16] the patient remained symptom-free for two years without antibiotics.

In most cases bacterial resistance to the antibiotic does not develop, and when symptoms recur they respond rapidly to re-institution of the therapy.

The duration of therapy is still uncertain. We do not yet know whether a patient can be permanently cured by antibiotics although a few patients have remained free of symptoms up to two years after discontinuing antibiotics. Since symptoms abate with therapy and may recur without it, one might presume that the body may not be able to develop an effective immunologic response. Eventually an antibiotic may be found which will have more prompt and permanent effects.

Summary

In all recently reported untreated cases of Whipple's disease in which electron microscopy has been used for tissue study, rod-

shaped bodies have been found in large numbers. These bodies measure 0.2μ wide and up to 2μ long, and have been identified as bacteria. They are phagocytized, but remaining within and causing the characteristic foaminess of the macrophages are undigestible, PAS-positive, mucopolysaccharide cell walls.

Tetracycline or certain other antibiotic therapy causes remission of the disease, and adrenal corticosteroids appear to enhance the effects of therapy. However, continued remission requires antibiotic therapy for an undetermined but prolonged period of time, and if medication is discontinued too soon the symptoms recur. The patient with Whipple's disease, previously doomed to a progressive and fatal course, can now be cured or his condition can be held in remission by appropriate antibiotic therapy.

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It's Science, but What Branch?

While using a scientific instrument to test for gas before opening a manhole, telephone repairmen overheard a youngster remark, "Look at them taking that sewer's blood pressure."

The Sprue Syndrome

James W. McBee, M.D.

Small bowel disorders grouped together as the sprue syndrome appear to be two distinct diseases. Gluten-sensitive sprue involves a patient's response to a diet containing gluten but the cause of tropical sprue has not been determined although it appears to be endemic in well-localized geographic areas. Treatment is different for the two types of sprue.

REVIEW ARTICLES

THE DEVELOPMENT and widespread use of peroral bowel biopsy, electron microscopy, and improved methods for studying absorption have greatly stimulated interest in small bowel diseases and related disorders of digestion and absorption. In the past few years there has been renewed interest in one group of small bowel disorders whose clinical features are so similar and histological appearances so identical that they are usually grouped together as the sprue syndrome. This syndrome may be conveniently classified into two varieties: (1) *gluten-sensitive sprue* in which the disease is related to ingestion of gluten, a protein fraction of certain flour, and (2) *tropical sprue*, which, while occurring endemically in well-localized geographic areas, has not been linked with any consistent causative factor. Further, gluten-sensitive sprue may be limited either to childhood or adult life and this has led to some confusion of terminology (celiac disease in children, nontropical sprue or idiopathic sprue in adults).

Laboratory Findings

The mucosal lesions of gluten-sensitive sprue and of tropical sprue are almost identical grossly and, when severe, the lesions may be readily recognized in fresh biopsy specimens viewed under the dissecting microscope. Examined with the dissecting microscope, the mucosa of a fresh biopsy specimen is characterized by a variety of abnormal changes ranging from shortening and blunting of villi in the milder forms of the disease to a flat mucosa with mosaic pattern and complete absence of villi when the disease is more severe. It has been demonstrated that all degrees of abnormal patterns may be present in one patient with the most severe changes occurring in the duodenum and upper jejunum and progressively less severe changes occurring distally.[1, 2] In sectioned and stained preparations, the villi are severely blunted or may be completely absent and there are far fewer epithelial cells than normally present. Flattening of the surface epithelium and increased round cell infiltration of the lamina propria are frequently observed but are less specific diagnostically. The milder lesions are most difficult to recognize and reliance must be placed upon the histologic evidence of reduction in epithelial surface.[3] Special staining techniques have allowed detection of other abnormalities. In fasting control patients, lipid droplets are found in the lamina propria and not in the superficial epithelial cells, but in sprue patients the droplets are limited to the supranuclear region of the epithelial cell and to a thin band in the region of the basement membrane.[4] Adenosine triphosphatase enzyme activity, normally present in the brush border and supranuclear area of epithelial cells, is greatly decreased or absent in gluten-sensitive sprue but is present in tropical sprue, a possible diagnostic criterion for histological differentiation of these two diseases.[5] In cases of sprue studied with electron microscopy, the brush

borders of epithelial cells are composed of microvilli which are deficient in size or number or both.[6]

Clinical Signs

Clinically, tropical sprue and gluten-sensitive sprue are similar and may occur at any age. While the frequency of occurrence of diarrhea and steatorrhea may vary with the degree of severity and duration of illness, lassitude and weight loss (or decline in growth rate in children) occur in almost every case. Other gastrointestinal symptoms, such as nausea, vomiting, flatulence, and abdominal discomfort may occur with variable frequency. Anemia is usually found in both types of sprue. In gluten-sensitive sprue, the anemia may be macrocytic with megaloblastic bone marrow or hypochromic microcytic anemia reflecting iron deficiency. Gastrointestinal absorption of iron may be defective even in the absence of steatorrhea.[7] The anemia in tropical sprue is almost always megaloblastic. Hypocalcemia, sometimes severe enough to cause tetany and osteomalacia, is not an uncommon finding in gluten-sensitive sprue, but is rarely observed in tropical sprue. Patients with gluten-sensitive sprue as well as patients with tropical sprue may complain of milk intolerance, and acquired disaccharidase deficiency has been demonstrated in patients with either disease.[8, 9] Roentgenological abnormalities of the bowel are similar in the two diseases and include such features as dilatation, segmentation, fragmentation, thickening of folds, hypersecretion and moulage. Dilatation of the small bowel is perhaps the most common and most specific sign for gluten-sensitive sprue and is best correlated with clinical and histological findings.[10] The jejunum is the best site for measuring the degree of dilatation and, in patients with severe sprue, the diameter may be increased to 35 mm. or more (normal, less than 25 mm.). Good correlation also has been shown among histological, radiological, and clinical parameters of tropical sprue.[11] Malabsorption of d-xylose, fat, radioisotope-labeled lipids, and a variety of other substances can usually be demonstrated by appropriate laboratory methods.

Treatment

While most of the morphological, clinical, and laboratory features of gluten-sensitive sprue and tropical sprue are similar, they apparently represent two distinct diseases, at least from the standpoint of etiology and pathogenesis. One prime differential factor is the response of the patient to a diet containing gluten, the protein fraction of wheat and rye flour. Investigators in Holland in the early 1950's observed that removal of wheat from the diet caused disappearance of signs and symptoms of celiac disease in children. More recently it has been possible to produce the mucosal lesion locally in gluten-sensitive sprue patients by instilling wheat through a tube into a previously normal appearing ileum,[12] and it has been demonstrated that great improvement both clinically and histologically occurs in such patients while they are on a gluten-free diet.[2] The mechanism for the clinical response to gluten in these patients is not known. The possibility that the disease may be immunologic has not been supported by antibody studies.[13, 14] There are data to suggest that gluten-sensitive sprue is familial and is inherited through a dominant gene of incomplete penetrance.[15] The striking feature of tropical sprue is its geographical distribution. The disease is found principally in the Far East, India, and in the Caribbean area, but is rare in tropical Africa. Curiously, patients having tropical sprue may not develop overt symptoms of the disease until after many years of residence in a temperate climate.[16] Although nutritional deficiency, infection, and gluten sensitivity have been suggested as causative factors, the etiology of tropical sprue remains as obscure as ever.

The most important step in the treatment of gluten-sensitive sprue is the elimination of wheat, barley, rye, and buckwheat from the diet. Oats may be given without harmful effects. The degree of improvement is related to the effectiveness in eliminating gluten from the diet. On a gluten-free diet, clinical improvement, with decrease in diarrhea and steatorrhea, weight gain, and decrease in abdominal distention may occur within a period of days to weeks. Histologically, improvement in the condition of the surface epithelial cells occurs earliest (six-ten days) and correlates best with the clinical improvement.[17] Gradual restoration of villi usually occurs after several months, but does not correlate well with the clinical course.[17] Adding gluten to the diet of gluten-sensitive patients whose disease is in remission may precipitate a clinical and histological relapse which can be recognized within 24 hours.[18] A good response to dietary treat-

ment can be expected in 80 percent to 100 percent of patients with gluten-sensitive sprue.[19] In some cases elimination of milk from the diet has effected further improvement since these patients may have acquired disaccharidase deficiency.[9, 20] Folic acid is considered the easiest and best therapy for tropical sprue. The clinical state responds rapidly but histological and biochemical recovery may not occur for a period of one to three years.[21] Oral antibiotics may also be beneficial, but require an adequate dietary supply of folic acid to produce a hematologic remission.[22]

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The Bigger They Are . . .

Discounting the cases in which death by accident or suicide has been a direct result of the effects of LSD, the only known victim of a lethal overdose of the drug was an elephant at the Oklahoma City Zoo. Though the case is mentioned in an article in the September issue of *MD*, the elephant's medical history is not presented.

Evaluation of Gastrointestinal Malignant Neoplasia

Robert S. Nelson, M.D.

Cancer of the colon and rectum can be diagnosed by present methods, whereas primary lesions of the liver and pancreas defy early diagnosis. Earlier and more accurate diagnosis of lesions of the esophagus and stomach would be possible if wider use were made of available methods.

REVIEW ARTICLES

PATIENTS WITH gastrointestinal malignant neoplasia can be classified in two main categories: (1) those in whom the referring physician has found clinical signs of cancer, but has not made a firm diagnosis, and (2) those in whom the diagnosis has been confirmed, with or without treatment, and who require evaluation regarding further care. When the diagnosis is only suspected, a careful and thorough routine of examination is necessary for confirmation in view of the radical surgery which must be proposed as the only hope for cure. When the clinical suspicion is strong and symptoms persist, close follow-up and extensive testing is in order even when results of the preliminary examination are negative or equivocal. Management of the patient with cancer involves determining the extent of metastases, the general condition of the patient, and his ability to withstand further surgery, radiotherapy, or chemotherapy.

Measures to be employed in the diagnosis and treatment of these two categories of gastrointestinal cancer, as encountered at The University of Texas M. D. Anderson Hospital and Tumor Institute, are considered in this article.

Diagnosis of Suspected Neoplasia

Esophagus.—In reviewing case studies of patients, it was found that a most striking feature in the poor results obtained was the long interval of continued and increasingly severe dysphagia undergone by the patient before he received adequate evaluation. Undoubtedly there are many instances in which the patient simply does not go to his physician, but there are others in which various medical remedies are given unsuccessfully before any diagnostic studies are carried out.[4] The importance of dysphagia, any sign of substernal pain, or slight delay in passage of food after eating, cannot be overestimated.[12] The extremely poor prognosis in cancer of the esophagus is, in many instances, directly related to the long interval between the beginning of symptoms and definitive attempts at diagnosis. When the diagnosis is made early, adequate and curative resection is possible.

Another factor involved in early diagnosis of carcinoma of the esophagus is that roentgenologic findings are frequently equivocal when the patient seeks medical care shortly after the onset of symptoms. The early lesion may cause little or no obstruction, and diagnostic roentgenologic studies may show few or no apparent abnormalities. Even esophagoscopic study, at this stage, may fail to reveal gross evidence of tumor. The radioactive phosphorus (^{32}P) test with a Geiger counter should be employed in such cases, and may result in a positive diagnosis in time for curative resection.[6]

Although unfortunately few early cases are available for study, it appears obvious that a defeatist attitude is not justified in cancer of the esophagus. Adequate education of the public as to the extreme importance of any symptoms of dysphagia, and awareness of the physician that often only a complete study will reveal the resectable le-

sion, should result in at least slightly better prognosis. It is particularly important that the physician not rely solely on negative results of roentgenologic studies. If facilities for esophagoscopy, esophageal washings for Papanicolaou smears, and ^{32}P studies are not available in the locality, and if the patient continues to have dysphagia or pain on swallowing and a nondiagnostic esophagram, there is a clear indication to send the patient to a facility where these further studies can be carried out. The apparent reluctance of some physicians to subject their patients to endoscopy under these circumstances is unjustified, considering the relative ease with which it can be performed and the extremely poor prognosis associated with advanced cancer of the esophagus. Neglect of all possible diagnostic aids early in the course of symptoms may cost the patient his life.

Case Reports

Case 1.—A 54-year-old white man was seen first because of substernal discomfort of two months' duration. Swallowing gave the patient the impression of a disagreeable sensation in the midsternal region, which was accentuated by swallowing hot or cold foods. Aspirin relieved the discomfort. Before referral, his physician had obtained a normal roentgenogram of the esophagus, and esophagoscopy had produced completely negative results. However, an ulcerative lesion of the soft palate was found. A biopsy specimen showed squamous cell carcinoma, grade 2. The patient was referred for treatment of the lesion of the palate. Before radiotherapy for this lesion was started, he still complained of substernal burning, and an esophagram failed to show any detectable lesion of the esophagus. An esophagoscope was passed with ease to a depth of 42 cm. and no gross lesions were seen on the initial passage. Phosphorus 32 readings were taken from 42 cm. up to 20 cm., and at the 32 to 25 cm. level there was a definite diagnostic differential. The counter was removed, and the esophagoscope was passed again to 30 cm., where a small exophytic tumor of the esophagus was visualized. A biopsy specimen was taken and read as squamous cell carcinoma. At this point, irradiation to the palatal lesion, which had already been started, was withheld and the esophageal lesion was resected by esophagogastrostomy and pyloromyotomy. The resected specimen showed grade 3 squamous cell carcinoma infiltrating the superficial area of the muscular coat, but not beyond this level; all nodes were negative for tumor. The patient improved, and his palatal lesion responded rapidly to continued radiotherapy. One month after surgery he was relatively asymptomatic, was eating well, and was able to swallow normally.

Case 2.—A 51-year-old white man had dysphagia for nine months before he was first evaluated in the hospital. Six months previously, esophagoscopy and roentgenologic studies had revealed no lesion of the esophagus. The dysphagia eventually became worse. He was unable to swallow anything but liquids, and a repeat esophagoscopy and esophagram were carried out prior to referral. This time, both procedures showed carcinoma, and a biopsy specimen revealed squamous cell carcinoma. At esophagoscopy, large exophytic masses of tumor could be seen at 28 cm. extending longitudinally down the esophagus. The instrument could be passed beyond the masses into the fundus of the stomach. Phosphorus 32 readings were definitely positive over an area of 34 to 28 cm. from the incisor teeth. There was no evidence of distal metastases. The patient was given preoperative irradiation with cobalt 60, with a total tumor dose of 2,000 rads on three consecutive treatment days. One week later he underwent esophagectomy, esophagogastrostomy, and pyloromyotomy. He seemed to do well initially, and then jaundice, apparently obstructive, developed. An exploratory laparotomy was carried out, but no organic obstruction was found. A liver biopsy examination showed moderate retention of bile in the small canaliculi only. Following surgery, the jaundice continued; the patient's condition deteriorated, and he died in a state of pulmonary insufficiency. There was no postmortem examination.

Stomach.—The symptoms associated with most cancers of the stomach are even less likely to produce prompt visits to the physician than those of cancer of the esophagus. Patients with hemorrhage or symptoms of obstruction early in the course of their disease are most fortunate. Any patient past the age of 50 who has symptoms involving the upper gastrointestinal tract must be considered a candidate for gastric cancer. If there is no free acid in the stomach, and an intrinsic factor defect as well,[7] with or without overt pernicious anemia, the chances that gastric neoplasia is present are considerably increased. Since there is no key symptomatology, most reliance must be placed on objective examinations to establish or rule out the diagnosis of gastric cancer.

Roentgenoscopy constitutes the first line of diagnosis, but is by no means infallible. The age group yielding the highest incidence of gastric carcinoma also produces the greatest incidence of cardiac, pulmonary, and arterial disease, so that a careful evaluation by all possible means is necessary to determine the advisability of exploratory surgery. When the roentgenological evidence is less than conclusive, search for other disease capable of causing the same symptoms, gastric washing for Papanicolaou smears, and endoscopy are indicated. In some institutions, as at the M. D. Anderson Hospital, endoscopy is routinely used with roentgenographic examination, no matter what the findings, since the two ideally complement each other.

A recent informal check of 97 consecutive cases of gastric cancer showed that 96 were diagnosed unequivocally by one or both methods. In the case with equivocal findings, suspicion was strong enough to prompt exploratory surgery. Similar results were obtained previously in 100 consecutive cases of gastric ulcer.[2] With the improved endoscopic instruments, essentially all patients may undergo gastroscopy. Age or general debility are not contraindications. A gastric washing which shows positive malignant cells is also definite evidence, but this test has not been as reliable an aid in our hands as endoscopy and roentgenoscopy; it is difficult to train and retain adequate technicians. The ^{32}P test is, so far, unreliable in the stomach. Use of the gastrocamera for recording gross appearance of gastric carcinoma gives excellent results, but the employment of this instrument as a sole diagnostic aid cannot be recommended at this time.[3]

The fallibility of roentgenoscopy in the diagnosis of gastric lesions must be recognized. Unfortunately, nondiagnostic roentgenoscopy findings are often a signal to dismiss the possibility of carcinoma, despite the persistence of symptoms. Conversely, evidence of tumor, however questionable, on x-ray films has been used as the sole basis for exploratory surgery in certain patients who were poor risks on general medical grounds. Careful use of all methods of diagnosis should eliminate both of these undesirable situations.

Case Report

Case 3.—A 59-year-old white man was first seen in 1959; he had epigastric pain for which no cause had been found. Three roentgenograms of the upper gastrointestinal tract had been reported to be within normal limits, although no free acid was found by gastric analysis. Gastroscopy revealed a large, ulcerating carcinoma of the greater curvature of the stomach. A repeat roentgenographic examination prior to surgery failed to show the lesion; despite this, a large carcinoma of the stomach was found at laparotomy, and total gastrectomy left only a small cuff of stomach attached to the esophagus. Following this, the patient did well for approximately 5½ years, during which, with frequent small feedings, he was able to carry out his normal work and lead a fairly normal life. During the last six months of this period, he noted increasing difficulty in swallowing, with some sticking of food in the lower esophagus. A roentgenogram showed no pathologic changes. Esophagoscopic examination showed a roughened area at a depth of 34 cm. on the right lateral wall of the esophagus; a tumor was suspected. The esophagoscope could be passed well beyond this area. Phosphorus 32 readings taken from 42 to 34 cm. were definitely positive when compared with background readings above this level. The diagnosis was probable recurrent carcinoma involving the lower esophagus, and biopsy specimens showed mucin-producing adenocarcinoma. At surgery, a large mass of tumor was found involving the whole diaphragm as well as the lower esophagus. Resection was not possible. A plastic tube prosthesis was placed in the esophagus, and the patient appeared to do fairly well for several months.

Pancreas.—There is no characteristic clinical picture of pancreatic cancer. In the nonjaundiced patient, abdominal pain without diagnosable source, occurring in a person who has recently developed diabetes, may present a problem which can be solved only by exploratory surgery. Unfortunately, when cancer of the pancreas is found in such a patient, it is invariably too disseminated for surgical resection. When obstructive jaundice secondary to cancer of the head of the pancreas calls attention to the diagnosis, resection is seldom possible for the same reason. The sole exception is the ampullary carcinoma, a relatively rare lesion.

Secondary aids, such as the secretin test, Papanicolaou smear tests of duodenal aspirates, with or without tetracycline fluorescence, or scintiscans employing selenomethionine Se 75,[1] have not solved the problem of earlier diagnosis. The presence of pancreatic carcinoma may be confirmed by any or all of these means, but by the time they are employed, the disease is too advanced for definitive operative intervention. At present, there is no reliable means of early diagnosis of this tumor.

Liver.—Primary cancer of the liver, unless discovered accidentally at laparotomy undertaken for some other reason, presents itself in most cases as an upper abdominal mass, often tender. Despite present capabilities of presurgical diagnosis, the prognosis remains extremely poor. The use of liver scans with radioactive rose bengal or similar isotopes, liver biopsy, or arteriograms may serve to delineate the tumor clearly, but the common experience at laparotomy is to find that the lesion is nonresectable. So far, successful attempts at earlier diagnosis have not improved the mortality rate from this disease.[8]

Rectum and Large Bowel.—Melena, bleeding, or pain in the rectum are still the main symptoms to be investigated in rectal or colonic cancer. "Ribbon" stools are not characteristic, although permanent change in bowel habit may be significant. The barium enema examination of the large bowel and

sigmoidoscopy will reveal most of these tumors. The two procedures always should be carried out together, and are the best accepted and most fruitful association of endoscopy and roentgenologic examination in the gastrointestinal tract. Failure to perform one or the other of these procedures accounts for most of the missed diagnoses in such tumors, while failure to analyze the stool for ova and parasites is the most common error in incorrectly diagnosing inflammatory disease as cancer. At least 80 percent of rectal cancers can be felt with the examining finger. Colonic washings for Papanicolaou smears have proved inaccurate, but in questionable cases the ^{32}P test may have a limited value in the differentiation of rectal lesions.[9]

Treatment in Confirmed Neoplasia

When the diagnosis can be confirmed or is strongly suspected prior to surgery, the patient must be thoroughly examined for evidence of metastatic disease. This is most frequently found in distal lymph nodes and liver, bone, or lung metastases; the usual sites at the time of initial surgery are nodes or liver. Lymphadenopathy or pelvic metastases (Blumer's shelf) may be readily detected on digital examination. If the liver is enlarged, or bromsulphalein retention or alkaline phosphatase level are elevated, a liver scan using radioactive rose bengal or gold Au 198 is performed. Where liver metastases are indicated by some or all of these studies, percutaneous liver biopsy will give histological confirmation of metastasis in 75 percent of patients.[10] A chest roentgenogram is, as a rule, accurate in detecting metastases at this stage; a bone survey may be done if indicated, although bone involvement is rare in gastrointestinal cancer.

With both primary and metastatic disease confirmed prior to surgery, the decision to resect the primary lesion must depend on the general estimate of length of survival time, as well as possibilities of complications from the primary lesion during this period. As a rule, an attempt to improve the patient's general condition by resecting as much of the primary tumor as possible should be made when there is obstruction or hemorrhage. Many colonic cancers grow slowly, and with the primary lesion resected the patient may survive for several years even when there are liver metastases. With this type of tumor, "second look" procedures are justified under certain circumstances because of the frequently slow rate of growth.

The patient with nonresectable tumor at the time of exploratory surgery is frequently referred in haste, within a matter of days after his operation, with the idea of starting chemotherapy as soon as possible. The most effective chemical agents for the treatment of gastrointestinal malignancy are all toxic, and should not be begun immediately after surgery in the usual debilitated patient. As a rule, several weeks should be allowed for wound healing and improvement in general health and nutrition. Even then, since any remission is likely to be short-lived, institution of chemotherapy is best delayed until there is definite evidence of: (1) tumor progression, (2) severe pain problem, or (3) impending obstruction. The advantages must then be weighed against the possibility of hemorrhage following tumor necrosis, or deterioration from the general toxicity of the drug. True remissions with chemotherapy have been rare in our experience, and have occurred in adenocarcinoma of stomach, colon, and rectum. They have not been seen in squamous carcinoma of the esophagus, adenocarcinoma of the pancreas, or primary carcinoma of the liver. The drug of choice at present is 5-fluorouracil, which is given in an initial loading course to the point of toxicity, then continuously on a once-weekly basis. The initial course requires hospitalization, but later weekly injections are given to outpatients. This method appears to be as effective and is far less strain than intermittent courses taken to full toxicity at four- to six-week intervals. Chemotherapy by infusion for primary or metastatic disease of the liver, despite occasional brilliant results, presents many technical difficulties which limit the usefulness of this type of administration.

Radiotherapy is seldom employed in treatment of primary gastrointestinal cancer except as palliation in esophageal carcinoma. In selected cases, it is useful in controlling the pain and discomfort of bone metastases and limiting growth of nodal spread. Radioactive gold grain implants are presently being tried as palliation in suitable patients with carcinoma of the pancreas, but this method is still in the experimental stage.

Summary

The diagnosis and treatment of the most common malignant tumors of the gastroin-

testinal tract is briefly reviewed in the light of recently introduced methods. Wider use of all diagnostic means now available would result in more accurate and earlier diagnosis in cancer of the esophagus and stomach. Primary cancer of the liver and cancer of the pancreas still defy early preoperative diagnosis, despite a number of newly devised procedures for this purpose. Colon and rectal cancers may be diagnosed with great accuracy by means now available, and while improvement is clearly possible, greater accuracy is attained in this area than in any other. When curative resection is not possible, palliation must be obtained by judicious surgery and properly timed chemotherapy.

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Tubs for New Mothers

"The anatomy of the female is such that the genitalia cannot be adequately cleansed in the shower," say two obstetricians who recommend tub baths as part of standard care for postpartum patients. Drs. R. A. Smith and J. E. Hodgson, Minnesota, found that in 12,150 such patients who bathed in tubs instead of showers, physical comfort was improved; nursing time was reduced; bladder function was improved; and morbidity was exceedingly low. Hospital obstetrical units should be equipped with about one tub per six patients, said the doctors, who reported their findings during a district conference of the American College of Obstetricians and Gynecologists.—*Medical World News, Nov. 4, 1966.*

Some Newer Aspects Of the Carcinoid Syndrome

John M. Eiband, M.D.; Harold Brown, M.D.; Charles H. Gregory, M.D.; and Michael E. Kahil, M.D.

Carcinoid disease is a spectrum of protean clinical manifestations and biochemical abnormalities consisting not only of the classical disorder but variant syndromes as well. Because of the favorable outlook the diagnosis should be established by morphological and biochemical means.

REVIEW ARTICLES

IN THE 1950's, the now familiar classical or florid carcinoid syndrome was defined.[1] It is characterized by episodes of flushing, diarrhea, and asthma, and as a late accompaniment, valvular lesions of the right side of the heart. The most common source of the primary tumor is the small bowel from which there are local invasion and extensive metastases to regional lymph nodes and to the liver. Associated biochemical abnormalities are elevated levels of serotonin in the blood and an increased excretion of 5-hydroxyindoleacetic acid in the urine.

In the past few years, however, substantial deviations from this classical syndrome have been noted. Atypical signs and symptoms may occur. The tumor may present in an unusual location or display unusual morphologic characteristics. Finally, a variety of humoral substances may be produced. Thus, a more appropriate name for the whole group of disorders may be "carcinoid disease" or, as suggested by Sjoerdsma, "carcinoid spectrum."[2]

The purpose of this presentation is to review some of the more recent concepts regarding the diagnosis, biochemistry and therapy of carcinoid disease and to describe certain clinical features observed in 16 patients.

Location of Tumor

Most carcinoid tumors are of gastrointestinal tract origin and can arise anywhere in the gut distal to the esophagus.[3] They may also begin in bile ducts, pancreas, bronchus, ovary, or testis.[3,4] The syndrome is usually not associated with tumors of the appendix, rectum, or duodenum, however, either because they elaborate no humoral substance or because they do not metastasize extensively to the liver.[3] It should be emphasized that the functioning carcinoid syndrome does not ordinarily appear until extensive metastases are present, usually in the liver. Exceptions to this generalization occur when the primary tumor drains directly into the systemic veins as happens with carcinoids of the ovary, testis, and lung.[2] In these instances a much smaller lesion, or one secreting less of the active substance, may produce the syndrome (Table 1).

Biochemistry

Serotonin, which is active in stimulating the contraction of smooth muscle, originates from dietary tryptophan via the precursor 5-hydroxytryptophan. The serotonin is then converted by monoamine-oxidase to the inactive metabolite 5-hydroxyindoleacetic acid (5-HIAA) which is recovered in the urine (Fig. 1).

Polyendocrine Aspects

Instead of secreting serotonin alone, some carcinoid tissue, particularly that from the stomach or bronchus, also may be associated with the increased production of the serotonin precursor 5-hydroxytryptophan.[2,5,6] In these cases, the flush and diarrhea may occur with relatively normal levels of serotonin in the blood and only moderately increased amounts of 5-HIAA in the urine. Increased concentrations of urinary serotonin and 5-hydroxytryptophan are found, however. Oth-

er tumors from both intestinal and extra-intestinal primary sites have been associated with the increased production of bradykinin, histamine, catecholamines, an insulin-like substance, melanin-stimulating hormone, and ACTH.[7] One patient in this group had an ovarian carcinoid associated with raised urinary 5-HIAA levels and Cushing's syndrome. Indirect evidence suggested an ACTH-like activity of the tumor.

Signs and Symptoms

Most patients with carcinoid tumors are asymptomatic and the lesion is found unexpectedly at the time of operation or incidentally at necropsy. Five of our patients had carcinoids discovered more or less unexpectedly at the time of operation for some other condition.

The Flush

Twelve of our 16 patients had flushing at the time they presented. Generally, the flush appears over the face and neck and persists usually for minutes but may last for days. It may be induced by a variety of stimuli including emotion, physical exertion, eating, defecation, ingestion of alcohol, compression of the tumor, and injections of epinephrine or norepinephrine.[8] Permanent cutaneous phenomena may be seen after varying periods of time. These include telangiectasia, hyperpigmentation, pellagrous changes, and patchy cyanosis even without heart disease.

Gastrointestinal Symptoms

Intermittent diarrhea and borborygmi, tending to occur at the time of the flush, are the most characteristic gastrointestinal manifestations of this disease. These symptoms were present in 13 of the 16 patients. In 3 they were the only symptoms. The stools tend to be watery, of unremarkable color, relatively inoffensive to the smell, and lacking in pus or blood. Abdominal pain, occasionally severe enough to mimic acute intestinal obstruction, occurred in 3 of the patients and subsided with conservative management. Rarely, a malabsorption syndrome occurs.[9] One of these patients may have had such an abnormality.

Because of the submucosal location of carcinoid tumors and their rare tendency to ulcerate, these lesions infrequently cause gastrointestinal bleeding.[3] Two of the 16 patients exhibited gastrointestinal blood loss possibly resulting from a carcinoid tumor. One presented with occult bleeding and the other had gross, presumably lower gastrointestinal, hemorrhage.

Table 1.—*Variant Syndromes.*

Signs and Symptoms	Tumor
Asymptomatic	Primary in stomach, lung,
Gastrointestinal bleeding	ovary, testis
Intestinal obstruction	Noncarcinoid tumors in
Malabsorption syndrome	pancreas, lung, thyroid
Cushing's syndrome	± Extensive metastases
Acromegaly	
Hypoglycemia	

Biochemical Abnormalities

Minimal Increase:
- Serotonin in blood
- 5-HIAA in urine

Polyendocrine Potential:
- Serotonin precursor (5-hydroxytryptophan)
- Histamine
- Bradykinin
- Insulin-like substance
- ACTH activity
- Melanin-stimulating hormone
- Catecholamines

Since extensive hepatic metastases are the rule in the carcinoid syndrome, prominent hepatomegaly is a frequent accompaniment and may be helpful in diagnosis. Of the patients in this group, 12 had hepatomegaly when first seen. One patient had an enlarged, tender liver with a hepatic friction rub and fever as part of her illness. Another patient had mild icterus associated with hepatic metastases.

Peptic ulcer has been reported in up to 20 percent or more of cases,[10,11] No such incidence was apparent in this group. In one of our patients, a carcinoid was discovered at the time of emergency celiotomy for perforated duodenal ulcer. The neoplasm was not functioning at the time so it is unlikely that the two were related.

Cardiovascular Disorders

About 50 percent of patients with the florid syndrome will develop endocardial lesions of the right side of the heart involving primarily the tricuspid and pulmonic valves.[12,13] Four of the patients reported

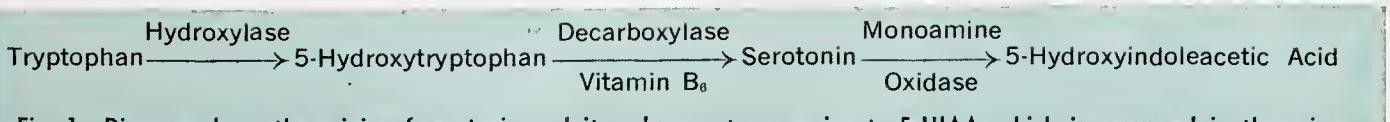

Fig. 1. Diagram shows the origin of serotonin and its subsequent conversion to 5-HIAA, which is recovered in the urine.

upon here had this type of cardiac disorder. The left side of the heart may be affected in patients with an atrial septal defect or in those with a bronchial carcinoid.

Profound arterial hypotension may be associated with spontaneous flushes, during the induction of anesthesia, or during palpation of the tumor at the time of operation. Epinephrine or norepinephrine may produce hypotension so that these agents should be avoided in the patient with the carcinoid syndrome who develops shock.[2] In these cases methoxamine, and perhaps angiotensin, are the vasopressors of choice.[2]

Asthma

Although asthma is listed as part of the syndrome, it is the least common of the major manifestations. Only one patient in this group had asthma and another had periodic dyspnea without wheezing.

Diagnosis

The diagnosis of carcinoid disease rests upon finding the typical morphological or biochemical abnormality. At times the microscopical features of carcinoid may be mistaken easily for those of adenocarcinoma of more malignant potential. Three patients in this group were initially thought to have metastatic adenocarcinoma of the liver. Subsequent events proved the diagnosis of carcinoid in all three instances. Because of the much more favorable prognosis it offers, it is important to consider carcinoid as a possibility in any case of well differentiated adenocarcinoma metastatic to the liver.

At other times noncarcinoid tumors may result in the functioning syndrome. Such examples are adenocarcinoma of the pancreas, oat cell carcinoma of the lung, and carcinoma of the thyroid.[13]

The most practical biochemical test available for detecting functioning carcinoid tumors is the determination of the amount of 5-HIAA secreted in the urine during a 24-hour period. Normally, this value is 2 to 8 mg. Patients with the functioning syndrome usually have well over 50 mg. per 24 hours. A normal or minimally increased value for urinary 5-HIAA strongly mitigates against the diagnosis of functioning carcinoid syndrome. Rarely, however, patients with the well developed disorder may have only minimal increases in urinary 5-HIAA excretion. When this is the case, increased amounts of 5-hydroxytryptophan, serotonin, or histamine are present in the urine and blood. One of our patients, a man with a bronchial carcinoid, had episodes of severe flushing and diarrhea lasting several days at a time. Only 54 mg. of 5-HIAA was detected in a 24-hour urine sample, but the histamine content of blood and urine was greatly elevated.

By the method usually employed in the determination, abnormalities in urinary 5-HIAA may occur in the absence of the carcinoid syndrome.[14] Moderately increased urinary excretion levels may occur in sprue, Whipple's disease, and diarrhea of other causes, or may follow the ingestion of large quantities of bananas, tomatoes, pineapple juice, and walnuts. Falsely high urinary levels may result from the ingestion of phenacetin, para-aminophenol, mephenesin, methocarbamol, and glyceryl guaiacolate. Falsely low levels resulting from a chemical interference with the test may be associated with the administration of phenothiazines. In the 1966 issue of the "Physicians' Desk Reference," more than 140 various therapeutic agents are listed which contain one or more of the foregoing ingredients (Table 2). The importance of such a large number of therapeutic agents causing interference with this test is self-evident. In case of doubt, a fluorometric determination is available which will detect true 5-HIAA concentration regardless of the presence of metabolites which might interfere with the standard test.

It is obviously important that the laboratory be well versed in the methodology of the procedure for detecting 5-HIAA and have critical quality control in addition. One patient has been seen who had intermittent flushing and elevated amounts of 5-HIAA in two 24-hour urine samples. He underwent exploratory celiotomy but no carcinoid was found. The elevation in 5-HIAA level was later found to be a laboratory error.

TABLE 2.—*Interference with Urinary 5-HIAA.*

Reaction	No. Compounds in PDR
False Increase	
Phenacetin	40
Para-aminophenol	46
Mephenesin	8
Methocarbamol	1
Glyceryl guaiacolate	28
False Decrease	
Phenothiazines	18
Total	141

The induction of a typical flush by the intravenous administration of epinephrine or norepinephrine is not known to occur in any other disease except the carcinoid syndrome.[8] Advantage may be taken of this fact to provide a simple bedside diagnostic test for the patient who presents with cutaneous flushing of indeterminate cause.[15] This test seems to be reliable in about one-half to two-thirds of the cases. Since severe hypotension may be associated with the administration of epinephrine in some patients, this risk should be carefully weighed before the injection is given. The epinephrine flush test has been tried in two patients of this group who had diffuse disease but were otherwise asymptomatic or had minimal symptoms. In both instances it was negative.

Therapy

The therapy of carcinoid disease leaves much to be desired. Except for the small primary or metastatic lesion that can be completely excised, treatment is rarely curative since most of the patients have widespread disease by the time it produces symptoms. Symptomatic improvement may be achieved, however, by the use of various types of therapeutic approaches.[16]

Removal of an unexpectedly discovered primary tumor without matastases, or those neoplasms which produce symptoms early because they drain into systemic veins, may give an excellent prognosis. Complete or partial resection of metastases or other tumor masses in patients with extensive disease may improve symptoms for long periods of time. Intestinal bypass operations are rational only in the exceptional case. One of our patients had the intestinal stream diverted around an obstructing tumor and the patient's flushes disappeared.

The most widely used serotonin antagonists are cyproheptadine (Periactin) and methysergide maleate (Sansert). Cyproheptadine is the most effective agent for the relief of flushes while methysergide seems more efficacious in controlling the diarrhea. Recent reports of retroperitoneal fibrosis occurring in chronic users of methysergide may temper its future use. Tolerance to both drugs develops eventually in many instances so that intermittent therapy is often of value.

Numerous other agents have been used with varying results. In the patient with a bronchial carcinoid, adrenal corticosteroids may be of benefit.

Antineoplastic drugs occasionally help some patients but roentgen therapy is usually disappointing.

Neither a low tryptophan diet nor the administration of radioactive tryptophan have been especially rewarding. Because of its theoretical value, niacin supplementation may be helpful and probably does not harm.

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Surgery in the Management Of Carcinoma of the Cervix

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The authors describe radiation as the major procedure in the treatment of carcinoma of the cervix because of lower morbidity and mortality rates than those in radical surgical treatment. They point out, however, that surgery is important in the overall management of cervical carcinoma.

REVIEW ARTICLES

THE TREATMENT of carcinoma of the cervix by radical surgery was begun in the 1880's, and in the ensuing decades the operative procedure was developed to a high level of effectiveness and safety by Clark, Wertheim, Bonney, and Shaw. By 1920, the operative mortality had been reduced to about 15 percent, and for patients suitable for the surgical procedure, about 40 percent were alive and well five years after surgery.[1]

The discovery of x-rays by Roentgen in 1895, and isolation of radium by the Curies in 1898, led to the exploration of possibilities of therapeutic radiation for many types of cancer. Radium was first applied to cancer of the cervix in 1903. In the early years of radium therapy, crude equipment, ignorance about the destructive effects of radiation, and the lack of any system of dosimetry meant that the risk to the patient was greater than the likelihood of eradication of the disease. There was no appreciable impact on the treatment of carcinoma of the cervix until the 1920's when radiotherapy became a challenge to surgery as the treatment of choice. There was firm resistance to the new modality and Peterson stated, "He who decides that radium therapy and not surgical treatment shall be instituted in operable cases has taken upon himself a heavy responsibility if permanent cure is not obtained. It must be considered indefensible to abandon surgery in operable cases of cervical cancer."[2] In the next decade, however, the impressive evidence amassed from increasing application of radiotherapy led Shaw to state, in 1937, that "After careful observation of these cases over seven years, we have come to the definite conclusion that radium offers the best chance of cure as well as the more humane method of treatment. Therefore, strong advocates though we were of the operation (Wertheim hysterectomy) we have abandoned it."[3] Radiotherapy had thus become the treatment of choice for patients with cervical cancer until 1939, when Meigs began to use a surgical approach that was to revive an interest in radical hysterectomy a decade later.[4]

Meigs' initial series (1939-1953) of 294 carefully selected patients with primary squamous cell carcinoma of the cervix consisted of 193 stage 1 cases, 99 stage 2 cases, and 2 stage 3 cases.[5] Study of the surgical specimens showed that there were lymph node metastases in 18 percent of stage 1 cases and 40 percent in stage 2 cases. There was a five-year survival rate of 75 percent in the stage 1 group, and 54 percent in the stage 2 group. The decision to return to the surgical method depended upon such crucial problems as establishing the incidence of lymph node metastases and whether radiotherapy could be relied upon to eradicate such spread beyond the primary site. The control of the primary lesion by intracavitary radium might be determined by clinical examination, but the incidence of lymph node metastases and the response to para-

metrial irradiation could not be determined without resorting to surgical exploration.

The search for solutions to these problems led to clinical investigations in which radiotherapy was followed by surgical lymphadenectomy to determine what the incidence of residual cancer in the pelvic lymph nodes might be. Wall presented preliminary observations in such a study in 1953.[6] Retroperitoneal lymphadenectomy was performed on 20 patients with squamous cell carcinoma of the cervix. There were ten patients with stage 1 lesions, nine with stage 2, and one with an unclassified cancer of the cervical stump. In this group of patients, radium only was used preoperatively. Bilateral retroperitoneal iliac lymphadenectomy was performed four weeks after completion of this radium treatment. Microscopic examination of the dissected nodes did not show evidence of significant radiation changes. This was the anticipated result since cancericidal effects from radium alone are known to be limited primarily to the paracervical triangle.

In 1962, a series of 340 cases was reported by Fletcher, Rutledge, and Chau, in which primary treatment by intracavitary radium and supervoltage roentgen therapy were followed by lymphadenectomy.[7] In this series there were 41 stage 1, 129 stage 2, and 170 stage 3 cases. Metastases to pelvic lymph nodes were found in only one stage 1 case, 13 percent of stage 2, and in 19 percent of stage 3 cases. Assuming that comparable procedures were employed in staging and in examining lymph nodes in surgical specimens, this combined approach seems to demonstrate that metastatic disease in involved pelvic lymph nodes can be sterilized by radiation.

Ultraradical surgery for advanced pelvic cancer was first done by Brunschwig in 1947.[8] The exenteration operation was based on the observation that in many patients who die from cancer of the cervix, the growth had not spread beyond the pelvis. Brunschwig originally undertook the removal of these large masses of tumor with the affected viscera for palliation, and the early attempts were successful in demonstrating the feasibility of the operation for its palliative effects. Later, the potentiality of ultraradical surgery to prolong life was realized, so that today exenteration is advised only when there is a reasonable chance for cure. Brunschwig showed that all the pelvic viscera could be safely removed, and that with the exception of the external iliac arteries, all the pelvic blood vessels could be tied on one or both sides without the risk of gangrene of the lower limbs. He also showed that it was not necessary to cover the pelvic floor with peritoneum although the large raw area certainly increased the incidence of postoperative small intestinal obstruction. By the end of 1958, Brunschwig and his team had completed 592 exenteration operations with an operative mortality of 17 percent and a five-year survival rate from 388 cases of 17 percent.[9]

At Baylor University College of Medicine Affiliated Hospitals, radiation is a major weapon in the treatment of carcinoma of the cervix, but surgery has a definite and important place in the overall management. It is the purpose of this article to present our concept of this vital role.

Primary Management

Early Lesions.—In the early cases (stages 1 and 2), radiotherapy is the primary treatment of choice. It has superseded radical hysterectomy because its high salvage rate has much lower attendant morbidity and mortality rates. In the most expert hands, both methods cure approximately 75 to 80 percent of stage 1 cases, and 55 to 60 percent of stage 2 cases.[1, 3, 5, 10, 11] Two-hundred eighty-three cases were recently reported by Wall and associates.[12] In the stage 1 group there was an absolute survival of 86 percent, with 63 percent in the stage 2 group and 41 percent in the stage 3 group.

Ureteral injury is the major complication after the radical operation, even though the mortality rate of the procedure has been reduced from 20 percent to less than 1 percent. The incidence of ureteral damage remains at about 12 percent. The occurrence of ureterovaginal fistula was a complication that plagued Wertheim and continues, impartially, to plague the surgeons of today. Vesicovaginal fistulas occur in approximately 2 percent of patients, and damage to the rectum in less than 1 percent. With properly executed radiation therapy, the incidence of major complications is less than 5 percent.

In the past, various combinations of radiation and surgery have been tried in an effort to improve the salvage rate in these early lesions. Full irradiation followed by planned radical hysterectomy some 6 to 12 weeks later has been extensively evaluated by Locke, Welch, and others.[13-15] In none of these large series in which combined meth-

ods were used were treatment results proved superior to radiotherapy alone. In addition, these series have shown conclusively that full radiation therapy and major radical surgery are not combined without a significant incidence of disastrous complications. Treatment of the primary lesion by radiation followed by removal of the regional lymph nodes has also been fully evaluated. External therapy can eradicate metastatic disease in the pelvis so that node dissection is unnecessary. When node dissection is done after radiation therapy there is a significantly high incidence of lymphatic complications, particularly pelvic lymphocysts.[16] This complication develops frequently after the transabdominal approach, probably because dissection is more extensive. No absolute indications for radical surgery exist in early cases. We do, however, recognize that there may be specific reasons for choosing radical hysterectomy as primary treatment. Occasionally there are patients with cancer of the cervix who may not be suitable candidates for radiotherapy. A mentally ill patient, for example, who is too hyperactive to lie still for roentgen therapy or will not return for more than one treatment may necessitate use of either a single heavy radium application or surgery. In such instances, we would prefer operative treatment. Infrequently, a patient may refuse radiotherapy, and in such instances, if it is at all feasible, surgery is preferable.

From time to time, other indications for surgery have been suggested, some possibly authentic. Carcinoma of the cervix in a pregnant patient has been cited as a surgical problem, but the results with radiotherapy are equally as good.[17, 18] However, at present we do a cesarean section or hysterotomy, and, if the lesion is operable, an immediate radical hysterectomy. We believe this approach is simpler in this particular situation although there is no demonstrable superiority in survival rate. Large fibroids occasionally are annoying for they enlarge and distort the uterus or endocervical canal. Patients with fibroids are treated with radiotherapy. The acute degeneration in myomata reported by others has not occurred in our experience. In applying vaginal sources, an unusually small vagina may constitute a difficulty; however, with close cooperation with the radiotherapist, a treatment plan can usually be successfully individualized. Acute salpingitis should be treated conservatively with antibiotics and rest. When it has subsided, bilateral salpingo-oophorectomy with subsequent radiotherapy gives results that are at least as good as, and possibly better than, those in other irradiated patients. Preservation of ovarian function in a young woman may also be an indication for surgery, as may be demonstration of a fixed loop of small intestine in the pelvis. Irradiation for adenocarcinoma has afforded poorer results than in squamous carcinoma, according to some physicians, but has not, in our experience, been true. The discovery of an adnexal mass that may be an ovarian neoplasm is another relative indication for primary surgery.

Advanced Lesions.—It is pertinent to note that cases of advanced carcinoma of the cervix are unnecessary today. They usually represent neglect in seeking medical care, or neglect in establishing an early diagnosis. Management of these advanced lesions is difficult and in general must be individualized depending upon the clinical extent of the disease and the anatomical distortion produced. The majority of cases in stage 3 can be treated effectively with radiotherapy alone, with an anticipated salvage rate of approximately 30 percent.[12, 19-22] If operation was performed primarily, a substantial proportion would require removal of the bladder or rectum, or both. This excessive loss of function can be avoided in the irradiated patient.

In the patient with late stage 3 or stage 4 carcinoma of the cervix, a combination of therapeutic modalities may well increase the salvage rate from the otherwise low figure of less than 10 percent.[12, 19-22] If distant metastases to lymph nodes, lung, or bone are evident, it is obvious that the disease is incurable. There are, however, patients in this distressing group with advanced cancer that may be limited to the pelvis. Whether such disease is advanced beyond possible cure by radiotherapy, surgery, or a combination of these methods may not be determined until treatment has been attempted.

In these patients, therapy is planned to allow a flexible approach for maximum benefits at minimum risks of morbidity. Radiation is begun and approximately one-half tolerance dose is given to the entire pelvis. If the lesion responds dramatically, then further radiotherapy is given to the tolerance level in a highly individualized manner. Depending upon the extent of the lesion, and the organs involved, small field external

therapy, interstitial radioactive isotopes, or intracavitary radium may be used. If the lesion does not respond well, the surgical component of combined therapy is considered. Pelvic exenteration calls for careful judgment, and should be done only if there is a reasonable chance for cure. The radiation is then considered to be preoperative, and will usually facilitate the dissection.

Persistent Disease.—If carcinoma of the cervix persists or recurs after the initial attempt at therapy, there is often a second chance at cure since this lesion remains localized for relatively long periods of time. Although the term "persistence" is generally used synonymously with the term "recurrence," we believe the former to be the more accurate term. Persistent disease is defined as an instance of tumor that apparently heals under therapy and then returns after a specific period of time. At our institution, this time interval is five years.

Surgery is the treatment of choice in cases of persistent tumor after radium and x-ray therapy. This situation, to us, represents the most important indication for ultraradical pelvic surgery. Pelvic exenteration is now an accepted and proved treatment procedure and after extensive evaluation it may be offered to selected patients. Secondary irradiation or chemotherapy, either systemic or regional, has little to offer these individuals.

Exploration and evaluation of resectability are indicated if no distant metastases can be demonstrated. Exploratory laparotomy is an acceptable diagnostic procedure, and our philosophy is to be extremely liberal in bringing patients to exploration but extremely critical and conservative in proceeding with resection. Such local signs or symptoms as leg edema, back pain, or ureteral obstruction are not considered by us to be absolute indications of nonresectability. Actually, we have found fibrosis to be the offender in many of these cases. A lesion cannot be deemed inoperable unless operation is done. Our only contraindications to exploration are distant metastases and medical fragility.[23]

Proceeding with resection is a different matter. It is unusual to be able to resect a persistent lesion by radical hysterectomy alone. Consequently, in most instances, resection eventually results in an exenterative procedure. The goal of this operation is cure, not palliation. It should be done only after extensive evaluation at the operating table. Contraindications to be noted at surgery are intraperitoneal disease, involvement of paraaortic lymph nodes and local extension from the parametrial tissues into the fascia overlying the obturator internus muscle or sciatic nerve. If the lesion is resectable, the urinary diversion of choice is the ileal or sigmoid conduit.

This is an ultraradical surgical procedure and major postoperative complications are to be expected. The incidence of immediate major complications in most larger series is approximately 35 to 40 percent.[24-27] About 10 to 15 percent will require reoperation for complications that occur in the early postoperative period. The major problems are intestinal obstruction, difficulty with urinary diversion, perineal fistulas (urinary or intestinal), hemorrhage, fluid and electrolyte imbalance, and wound infection.

Once the immediate postoperative period is over, late problems can still be expected. Approximately 30 percent of patients will require surgery for later complications. These complications are related mainly to the urinary diversion, the external stomata, and the large pelvic defect which predisposes to fistulas and intestinal obstruction.

The overall results with this procedure show a five-year survival rate of approximately 25 to 30 percent. The immediate operative mortality rate is about 15 to 20 percent, and the incidence of late deaths is somewhere between 50 and 60 percent.[9, 24-27] Most of these late deaths are attributable to persistent disease and occur within the first two years after operation. With rigid criteria for resection, operative experience, and refined and sophisticated preoperative and postoperative care, an increase should be seen in the five-year survival rate with a decrease in the immediate operative mortality. This has been the trend since the procedure was first done in 1947. Brunschwig's operative mortality during the first several years was 25 percent.[8] Bricker reported 13 percent in 1956, Parsons 15 percent in 1958, and Rutledge 17 percent in 1965.[24, 27, 28]

Problems in Diagnosis.—It is well known that radiation can produce complications in the pelvis that resemble active disease.[29] The classical indications of persistent disease are leg edema, sciatica, ureteral obstruction, and fixed mass attached to one or both pelvic walls, colloquially termed the frozen pelvis. These signs and symptoms can all be produced by radiation fibrosis. Because of this, the care of patients with cancer of the cervix can become increasingly complex. Therapeu-

tic decisions in identical situations may vary greatly depending on whether the cause is radiation fibrosis or cancer. It does become mandatory to insist on histologic confirmation of disease so that a definite course may be followed. Since management is properly determined only by correct diagnosis, there is no room for assumption. To treat radiation injury as cancer will increase the injury, and to temporize with persistent cancer in the belief that it is fibrosis can result in loss of the chance for cure.

When positive biopsies can be obtained transvaginally, there is no diagnostic problem. The cervix and the upper vagina are the recipients of maximum therapy and disease areas are not as frequently the sites of persistent carcinoma as are the parametria and pelvic nodes. Exploration is often the only means available to ascertain the presence of persistent disease. There should then be no hesitation in proceeding with diagnostic laparotomy. This principle must, of course, be adapted to the circumstances, since, for example, a patient with demonstrable distant metastases or in precarious medical condition from other causes is obviously unsuitable for surgery.

Exploration must be extensive and should be done by a surgical team qualified to proceed with radical resection if operable disease is found. The para-aortic and iliac lymph nodes must be carefully evaluated and biopsied. The paravesical and pararectal spaces must be examined and biopsy specimens taken from the cardinal and uterosacral ligaments. The sciatic nerve area must also be thoroughly evaluated. Any conclusion regarding the presence or absence of tumor can be made only after extensive exploration.

Radiation Complications.—Major complications are defined as those which require corrective surgical procedures or produce permanent disability, and after properly executed radiation are unusual. In the series reported by Wall and associates, there were nine major complications among the 208 patients for a complication rate of 4.3 percent.[12] When the dosimetry is reviewed, it usually becomes apparent that in most cases there have been errors in the application of radium or in external beam therapy so that tolerance has been exceeded either in total dose or dose rate. In other instances, the therapy will be found to have been well executed but there is an altered tolerance to radiation on the part of the patient. Some of the factors that may influence tolerance are age, general health and nutrition, inflammation, and previous pelvic surgery. The majority of complications can be avoided if particular attention is paid to the radiation dosimetry during the course of therapy, as demonstrated by our own low complication rate.

When radiation complications occur, their management is often as challenging as the problems of the original disease and more difficult to solve. Surgery is the preferred method of management in these unfortunate problems.

Such rare rectal problems as severe proctitis, stricture, or fistula require proximal fecal diversion. If the procedure is considered to be temporary, a transverse colostomy is done, or, if permanent, a sigmoid colostomy. Resection of the involved segment of bowel and a pull-through procedure may be indicated at a later date. A localized injury to the sigmoid may be resected and end-to-end anastomosis performed.

Vesicovaginal fistulas should be rare if precision dosimetry has been used, but can present extremely difficult problems. Occasionally, they can be repaired locally. Bastiannse, Mandicas, and Ingelman-Sundberg deserve credit for having developed modern techniques in the repair of postradiation fistulas.[30-33] Scrupulous dissection of the fistula and surrounding tissues, and the bringing of well-nourished tissue to the fistula site are imperative. The bulbocavernosus muscle, pubococcygeus muscle, rectus, adductor longus, or the gracilis muscle may be used. In most, local repair is not successful and permanent urinary diversion must be performed. The ileal conduit is the procedure of choice.

Ureteral obstruction secondary to periureteral fibrosis is a difficult problem and treatment for it must be individualized.[29] The function is not compromised, only careful observation is indicated. With decreasing renal function, freeing of the ureter from its fibrotic bed is the procedure of choice. Occasionally, ureteroneocystotomy or a bladder flap procedure may be needed, although these reimplantation procedures can fail because of radiation changes in the bladder. In rare instances, permanent urinary diversion may be required.

Palliation.—Palliation is the application of certain therapeutic measures for the pur-

pose of improving the condition of the patient with "incurable" disease. Unfortunately, palliation has come to have the connotation of futility which inhibits the careful effort that could mean effective and useful rehabilitation for many patients in the uncured group.

Selected surgical procedures may be effective in diminishing requirements for medication and nursing care even if the course of the disease itself is not changed. Cordotomy may be indicated for sciatic pain. Colostomy will be a welcome procedure in the patient with a rectovaginal fistula or partial rectal obstruction. Bilateral hypogastric artery ligation is an effective measure for controlling hemorrhage. Urinary diversion may occasionally be indicated in the patient with a large vesicovaginal fistula, who is otherwise leading a useful life.

Summary

At the Baylor University College of Medicine Affiliated Hospitals, radiation is the major procedure in the treatment of carcinoma of the cervix. It has superseded surgery because its high salvage rate is attended by much lower morbidity and mortality.

Surgical therapy is, however, important in the overall management of cervical carcinoma. Radical hysterectomy is rarely done for early lesions, but various combinations of radiation and ultraradical surgery are used for advanced cases. Ultraradical surgery becomes the treatment of choice when there is persistent disease after adequate irradiation. Seasoned judgment is vital in resolving diagnostic problems in patients treated for carcinoma of the cervix, in those with radiation complications, and in support of the patient with incurable disease.

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Old and New Methods of Therapy For Carcinoma of the Cervix

Report of 170 Cases

Maurice C. Archer, M.D.

Newer methods of treatment have increased the survival rate of patients who have late stage cancer of the cervix, and have provided cures for an increased number of patients with earlier stages of carcinoma.

REVIEW ARTICLES

RADIUM was first applied to carcinoma of the cervix by Abbe[1] in 1903. The fundamentals of this therapy—definite principles, underlying action, and techniques of application—were developed in 1914-1915 by Bailey and associates.[1]

In 1937 Fletcher Shaw,[1] one of Britain's ablest surgeons, wrote, "After careful observation of these cases for over 7 years, Professor Dougal and I have come to the definite conclusion that radium offers the best chance of cure as well as being the humane method of treatment. Therefore, strong advocates though we were of the operation, we have now abandoned it." This established the combined administration of intracavitary radium and external roentgen rays as standard therapy for carcinoma of the cervix.

Interstitial radium therapy, suggested by Alexander Graham Bell[1] in 1903, was accomplished by Donaldson in 1915 and later was used by Pitts and Waterman in 486 cases.

Clinical Varieties

Cancers of the uterine cervix can originate either in the portio vaginalis or in the endocervix. The three main varieties are as follows:[2]

Exophytic Tumors.—There are two types: (1) Those which originate on the portio vaginalis grow outward in a cauliflower shape, and primarily involve one lip of the cervix. These are friable and bleed easily. (2) Those originating in the endocervix produce a concentric enlargement of the cervix. These may reach huge dimensions, extending from one pelvic wall to the other. They grow rapidly and have a high degree of metastatic aggressiveness. This type of tumor forms large, cylindrical masses protruding downward into the vagina. The cylindrical masses are radioresistant, and are present in 25 percent of the cases at autopsy, but in only 6 percent of the living group.

Infiltrative Tumors.—Originating most often in the endocervix, these tumors are stony hard and regress little with external irradiation. A positive diagnosis from biopsy is difficult to obtain even in an advanced case.

Ulcerative-type Lesions.—These erode the entire cervix, and replace the vault with a large crater exhibiting indurated edges. The uterine cavity may be replaced by cancerous tissue.

Clinical Biological Features And Routes of Spread

Cancers of the uterine cervix are squamous cell carcinoma, adenocarcinoma, or adenoacanthoma, the vast majority being squamous cell carcinoma. Little is known of the frequency of involvement of the lymphatic nodes of the pelvic wall in cases of adenocarcinoma and adenoacanthoma. It is assumed that their routes of spread are similar to those of squamous cell carcinoma.[1]

Squamous cell carcinomas of the cervix have three main routes of extension:[1] (1) Into the vaginal mucosa at a considerable distance beyond the palpable or visible disease. (2) Into the myometrium of the lower

uterine segment and from there to the whole corpus. This type of extension is more likely in carcinomas originating in the endocervix. (3) Into the network of lymphatics of the paracervical areas around the ureters lateral to the internal os and lower uterine segment, fanning out into the medial aspects of the broad ligaments.

Radiosensitivity

Radiosensitivity[2] depends upon the status of the tumor bed, the clinical variety of the tumor, its size, and its histologic type.

Radiosensitivity does not have direct correlation with radiocurability. The fast growing, concentric tumors of the cervix are radiosensitive, regress rapidly, and can be controlled with local treatment, but they have a high degree of metastatic aggressiveness. The survival rate of patients with this type of tumor is lower than for those whose tumors are less sensitive to radiation.

Early Treatments of Choice

Radiotherapy.—The preferred methods are (1) intracavitary radium combined with external irradiation; (2) interstitial radium combined with external irradiation; (3) transvaginal roentgen rays combined with external roentgen rays; or (4) intracavitary radium combined with transvaginal cone therapy and external irradiation.

Operative.—Procedures of choice are (1) radical hysterectomy (Wertheim) with lymphadenectomy; (2) radical vaginal hysterectomy (Schauta); or (3) simple complete hysterectomy.

Combined Therapy.—Usual procedures are (1) radical hysterectomy combined with external irradiation, or (2) intracavitary radium combined with either transperitoneal or extraperitoneal lymphadenectomy.

Contraindicated Surgical Approach

Amputation of the cervix or supravaginal hysterectomy should not be considered for treatment of invasive carcinoma of the cervix. In some cases, stage 1 invasive cancer has been mistakenly classified as preinvasive or epithelial carcinoma in situ, stage 0; surgeons performing hysterectomies in these cases have inadvertently cut through malignant tissue, leaving residual areas of carcinoma. This surgical procedure also makes treatment difficult, since it leaves no uterus in which to place radium. A number of such cases, however, have been salvaged since the development of supervoltage x-ray and cobalt 60 therapy.

Brunschwig developed the exenteration procedure which has occasionally been useful in treating patients who have highly radioresistant tumors.[1]

Transvaginal Cone Therapy

Intracavitary radium therapy only, without other irradiation, frequently fails to cure the lesion because little radiation reaches areas beyond 2 cm. from the point source of radium.

Transvaginal cone therapy came into effective use in 1920. Erskine[6] did considerable work with transvaginal cone therapy and believed his results to be spectacular. He found that this efficiently destroyed primary tumors, and he developed a four-bladed speculum to aid in treating carcinoma of the cervix.

Elkins[7] also worked with transvaginal cone therapy, and stated that lethal or near-lethal doses may be delivered to parametria without applying a destructive dose to the cervix. He found that three-year survival rates of patients with stage 4 cancer were greatly increased when this method was used.

Wasson[8] found that transvaginal cone therapy combined with external irradiation produced excellent results in stages 1 and 2, and good results often were noted in stage 3 cases. He also reported cures of residual tumors following panhysterectomies. (I treated one such patient who survived 16 years before breakdown of the parametrium.)

Bouslog[9] was not satisfied with the results he obtained with radium implants and external irradiation, and developed a multiple port method using five overlapping fields with a metal cylinder. Using transvaginal cone therapy, he increased the survival rates in stage 3 and 4 cases.

Taylor and Twombly[10] considered that they had best results using intracervical radium combined with transvaginal cone therapy and external irradiation. This mode of therapy was the treatment of choice until development of supervoltage x-ray and cobalt 60 therapy.

The early changes noted during transvaginal cone therapy are (1) clearing of infection, (2) cessation of bleeding, (3) early manifestation of healing with diminution of pain, and (4) healing of the local lesion in six to eight weeks.[3]

Technical factors used in transvaginal

cone therapy are as follows: 220 kv.; 20 ma.; 36 cm. target skin distance; 0.5 mm. copper and 1.0 mm. aluminum filter; and five ports: 4,000 roentgens in air direct to cervix, 4,000 R. in air to each lateral fornix, 2,000 R. in air to the posterior edge of cervix slightly toward the rectum, and 2,000 R. in air along the edge of the cervix directed toward the bladder. External irradiation factors are 220 kv.; 20 ma.; Thoreous filter; 50 cm. target skin distance; and two ports: posteroanterior and anteroposterior alternately, 150-200 R. in air daily, 200 R. in air per port.

Present Mode of Therapy

Our most recent method of treatment of carcinoma of the cervix is the combination of intracavitary radium immediately followed by supervoltage therapy. This report summarizes our experience in treating 34 patients with supervoltage radiation; cobalt 60 was used in some of these cases.

The Ernst applicator used for the radium implantation consists of an intracervical tandem of three segments, each containing 10 mg. of radium. The two center colpostats contain no radium. The four outer colpostats (two on each side) contain a total of 40 mg. (10 mg. each) of radium. The dosage is approximately 10,000 gamma R. to the cervical os, 7,500 gamma R. to point A, and 2,500 gamma R. to point B (Fig. 1). Radium remains for 96 hours, with a total dose of 6,720 mg.-hr.

We realize that other authors, particularly

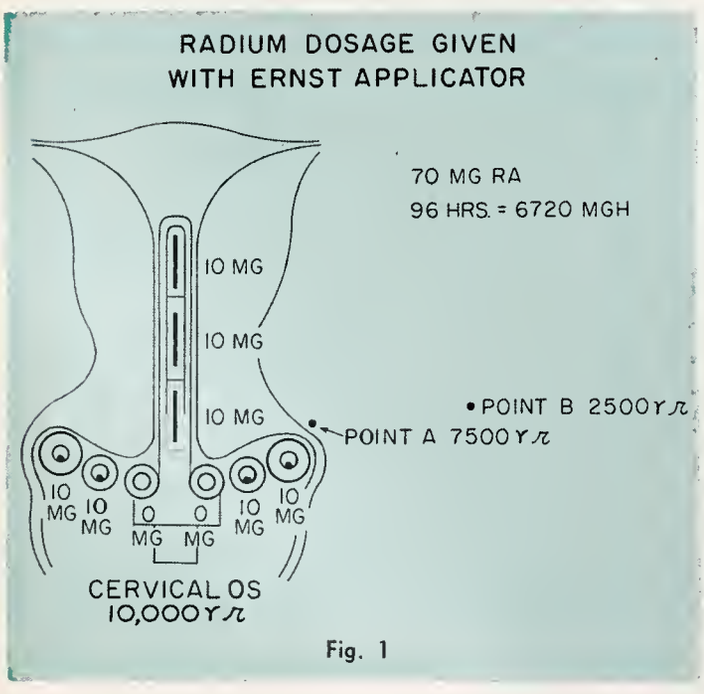


Fig. 1

in the teaching institutions where the full cost of treatment is not always borne by the patient, strongly advocate two or more separate radium applications. However, most of our patients cannot afford two or three hospitalizations, and we have found that by leaving the radium in place for 96 consecutive hours, the patient is subjected to only one hospital admission. We have encountered no undue sequelae during the many years that we have used this method.

Within 24-48 hours after radium therapy is completed, 2 m.e.v. supervoltage therapy is started, using a Van de Graaf generator. A two-port method is used, 378 sq. cm. fields, posteroanterior and anteroposterior with a two-inch lead block over the cervix and uterine canal for protection. Patients are treated daily, 200 R. tumor dose per day, five days a week, giving a total tumor dose of 4,000 R. This brings the total dose to point B and the pelvic wall to approximately 6,500 R.

Cancer of the Cervical Stump

The incidence of cancer in the cervical stump is about 2 percent, and these account for 6 to 8 percent of all cancers of the cervix.

Methods of treatment are (1) intracavitary radium, if applicable, with only one capsule placed in the cervix, and (2) transvaginal cone therapy to the cervix, followed by supervoltage external irradiation. The latter procedure is, in many cases, superior to intracavitary radium therapy, and is especially useful when intracavitary radium cannot be used.

Results of Treatment of 170 Cases

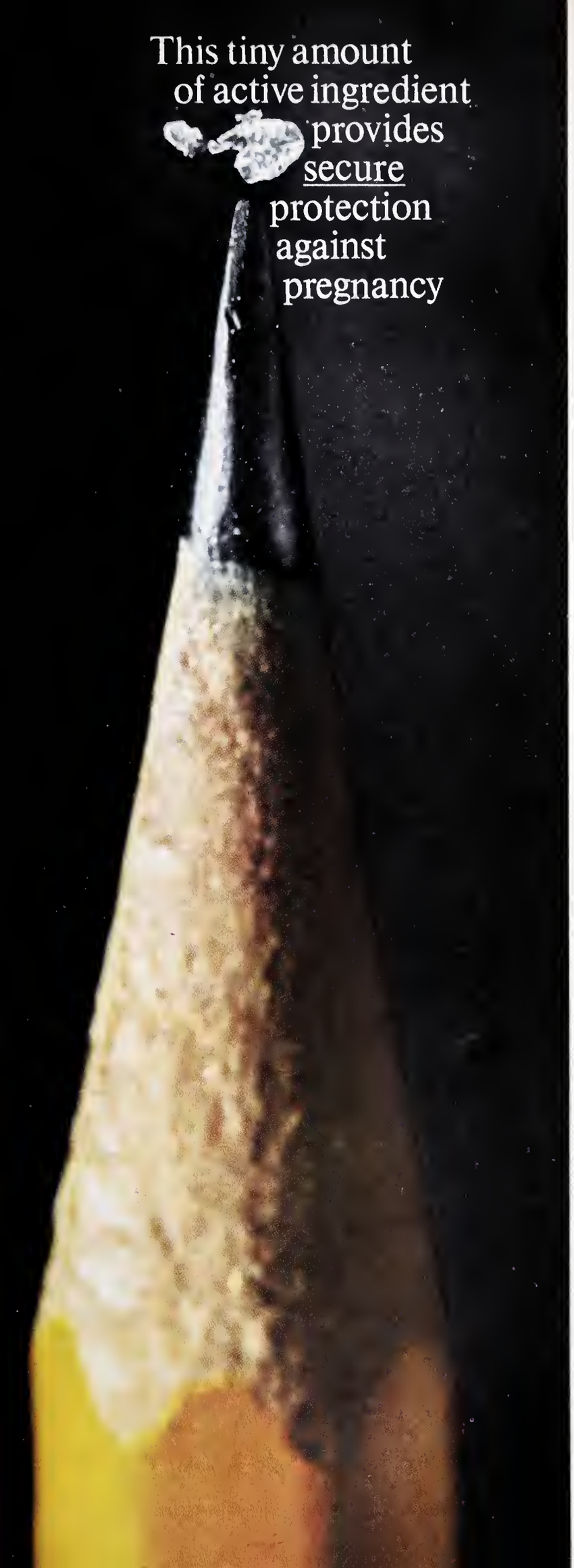
Tables 1-4 show the increase in survival rates, especially in patients with late stages of cancer, as newer and more efficient equipment and methods of therapy have been devised. Patients in this series were treated over a 15-year period.

Tables 3 and 4 demonstrate that since development of supervoltage equipment, the survival rate of patients with advanced stages of carcinoma of the cervix has been increased.

Conclusions

Advantages of using supervoltage therapy are:

1. Total treatment time is about one month (200 R., five days a week, plus radium) whereas orthovoltage, radium, and



Massive, worldwide clinical experience

Weight gain as infrequent as weight loss

New freedom from undesirable effects

New low cost

Contraindications—Mammary or genital carcinoma should be ruled out prior to administering Ovulen. Undiagnosed vaginal bleeding is also a contraindication to Ovulen use. A history of thrombophlebitis or pulmonary embolism, or both, is a contraindication to the use of Ovulen, unless its use is judged by the physician to be necessary despite the possible risk. Ovulen should not be used in women with suspected or overt liver dysfunction or disease. Ovulen is contraindicated in pregnant and nursing women and in patients with a history of cerebrovascular accident.

Warnings—Medication should be discontinued pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. Medication should be withdrawn if examination reveals papilledema or retinovascular lesions. Since the safety of Ovulen therapy in pregnancy has not yet been established, it is recommended that, in a patient who has missed two consecutive menstrual periods, pregnancy be ruled out before oral contraceptive therapy is continued.

Precautions—Because Ovulen may aggravate a tendency toward fluid retention in some patients, it should be administered cautiously to patients with a history of renal or cardiovascular disease (including hypertension), asthma, epilepsy or migraine. Any possible influence of long-term Ovulen therapy on pituitary, adrenal cortical, ovarian, hepatic or uterine functions requires further study. Oral contraceptives also should be administered cautiously to diabetic patients since a decrease in glucose tolerance has been observed in some patients taking these drugs. Patients on Ovulen may occasionally show abnormal glucose tolerance tests, but this does not necessarily indicate the presence of diabetes. Significant increases in platelet count, prothrombin and proconvertin tests, plasma thrombotic activity and plasma proteolytic activity have been reported.

Since estrogens may affect results of serum protein bound iodine and other thyroid function tests, these tests should not be considered definitive until Ovulen therapy has been discontinued for at least sixty days. Adrenal steroid serum levels and excretion may be affected by estrogens; the Metopirone® (SU-4885) test of pituitary-adrenal function may also be depressed. Abnormalities in hepatic function tests have also been reported, including some interference with dye excretion by the liver. This interference may give rise to Bromsulphalein® retention and jaundice in susceptible individuals. Serious liver dysfunction should be ruled out before continuing Ovulen administration when abnormalities in liver function tests occur.

Patients with a history of psychic depression should be observed carefully during treatment with oral contraceptives, and such treatment should be discontinued if depression recurs to a serious degree. Pre-existing fibroids may increase in size during Ovulen therapy. Such fibroids may regress to pretreatment size after Ovulen is stopped. In the event of breakthrough bleeding the possibility of nonfunctional causes should be borne in mind. Additional means of contraception should be used during the first seven days of Ovulen administration in the first treated cycle, because early ovulation may possibly occur.

Side Actions—The following adverse reactions have been reported with Ovulen; however, a causal relationship to Ovulen administration has not been established in all of the listed complaints: headache, dizziness, depression, breast complaints, amenorrhea, chloasma, vomiting, allergy, edema, migraine, pulmonary embolism, thrombophlebitis, visual difficulties, nervousness, rash, itching, decrease in libido, tiredness, malaise, hair loss and hair growth. A small incidence of nausea, spotting and breakthrough bleeding has been reported; these complaints tend to diminish markedly or disappear after the first cycle of treatment. Some of these side actions have required discontinuance of the drug.

Dosage—One tablet daily for 20 consecutive days beginning 5 days after the onset of menstruation.

Before prescribing see Detailed Product Information, Ovulen.

An extensive list of references on Ovulen is included in the literature mailed to physicians.

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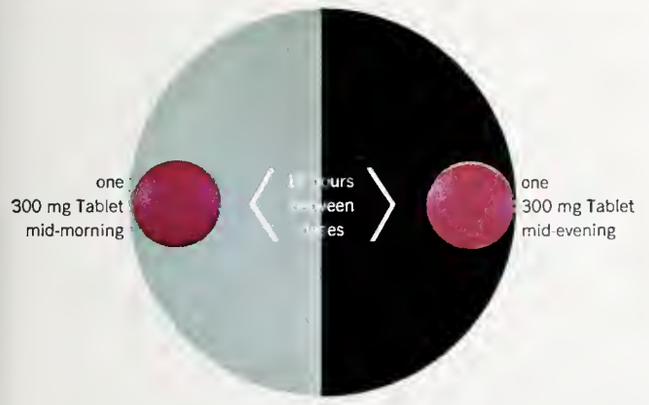

It's made for b.i.d.

Effective in a wide range of everyday infections — respiratory, urinary tract and others — in the young and aged — the acutely or chronically ill — when the offending organisms are tetracycline-sensitive.

Contraindication — History of hypersensitivity to demethylchlortetracycline.

Warning — In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort.

Precautions and Side Effects — Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

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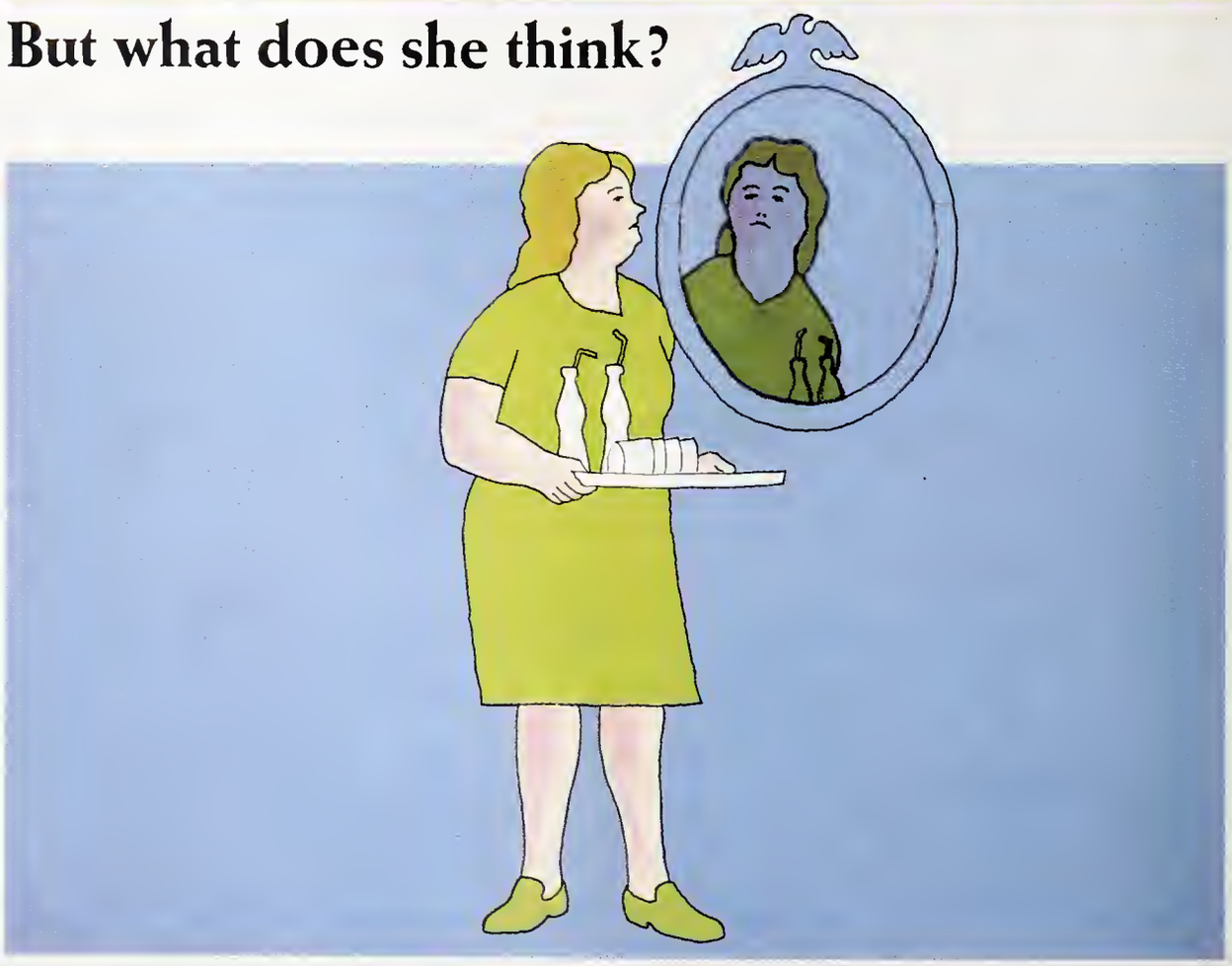

Many overweight patients can benefit from the appetite control provided by the sustained anorexigenic-tranquilizing action of BAMADEX SEQUELS: anorexigenic action of amphetamine; tranquilizing action of meprobamate; prolonged action through sustained release of active ingredients.

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DEXTRO-AMPHETAMINE SULFATE (15 mg.) WITH MEPROBAMATE (300 mg.) SUSTAINED RELEASE CAPSULES

to help establish a new dietary pattern

Contraindications: Dextro-omphetamine sulfate: in hyperexcitability and in agitated prepsychotic states. Previous allergic or idiosyncratic reactions to meprobamate.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching ond, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring olertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriote caution is recommended with patients prone to excessive drinking. In patients prone to both petit ond grand mal epilepsy meprabamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulatian af the central nervous system, jitteriness and insamnia or drawsiness.

Dextro-amphetamine sulfate: Insamnia, excitability, and increased motor activity are camman and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libida, and hallucinations have olsa been observed, especially in mentally ill patients. Rebaund fatigue and depressian may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may accur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic ar idiosyncratic reactions: maculopapular rash, acute nonthrambacytapenic purpura with petechiae, ecchymases, peripheral edema and fever, transient leukopenia. A case of fatol bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agronulacytasis and thrombocytopenic purpura, and a fatal instance af aplastic anemia, but only when other drugs known ta elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visuol accommodatian. Massive overdasage may produce drowsiness lethargy, stupor, ataxia, coma, shack, vosomotor ond respiratory callapse.

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TABLE 1.—*External and Transvaginal Cone Therapy Only—Orthovoltage.*

Group	No. Patients	Died Before 5 Years	Died After 5 Years	Living Patients	5-Year Survival Rate (%)
Stage 1	11	0	0	11	100
Stage 2	28	2	0	26	93
Stage 3	19	14	2*	3	26
Stage 4	24	22	1 †	1 ‡	8
Totals	82	38	3	41	
Overall Five-Year Survival Rate					54

Twenty of these patients were given radium therapy earlier (one month to two years) by another physician.

*Both died after ten years, one of recurrent and one of a new primary cancer.

†Seven years.

‡Recurrence at two years successfully treated with supervoltage (2 m.e.v. unit).

TABLE 2.—*Radium Therapy Using Clark Applicator Combined with External and Transvaginal Cone Therapy.*

Group	No. Patients	Died Before 5 Years	Died After 5 Years	Living Patients	5-Year Survival Rate (%)
Stage 1	2	0	0	2	100
Stage 2	30	3*	2†	25	97
Stage 3	17	12	0	5	29
Stage 4	5	5	0	0	0
Totals	54	20	2	32	
Overall Five-Year Survival Rate					67

All patients were given radium therapy for a total of 3,600 mg.-hr. followed immediately by x-ray therapy.

*One death, after three years, was from a cardiovascular accident; one at four years was from a new primary. Since autopsy showed no malignancy involving the cervix, these two cases were considered five-year survivals.

†One death at 10 years was from recurrent disease, and one at seven years was from a new primary.

TABLE 3.—*Supervoltage Therapy.*

Treatment	No. Cases	Deaths		Survivors	
		No.	Stage CA	No.	Stage CA
Rotational Cobalt 60 2 m.e.v. Combined with Transvaginal Cone Therapy	4	2	4	2*	4
	2	1	4	1†	3
Radium Therapy with Ernst Applicator Combined with 2 m.e.v. Supervoltage	28	5	(4) 4 (1) 3	23‡	(1) 4 (5) 3 (17) 2

*Patients living at two and six years.

†Patient living at three years.

‡Three patients living at five years; seven at three years; six at two years; four at one year; three at less than one year.

transvaginal cone therapy take about three months.

2. There is less skin irradiation reaction. A total of 170 patients with carcinoma of the cervix are reported. Results of treatment with orthovoltage external and transvaginal cone therapy, radium therapy combined with orthovoltage external and transvaginal cone therapy, and radium therapy combined with supervoltage external irradiation were compared. There was a remarkable improvement in survival rate in patients with stage 3 and 4 carcinomas treated by radium therapy using the Ernst applicator technique combined with supervoltage external irradiation (47 percent) compared with survival rate (0-29 percent) in the groups treated with orthovoltage transvaginal cone therapy and external irradiation, with or without radium therapy.

With the combination of radium therapy using the Ernst applicator and supervoltage

TABLE 4.—*Comparative Survival Rates with Different Treatment.*

Group	Total	Survivals	Deaths	Survival Rate %
External and Transvaginal Cone Therapy	82	44*	38	54
Radium Therapy Using Clark Applicator Combined with External and Transvaginal Cone Therapy	54	36*	18	67
Supervoltage Therapy†	34	26	8	77

*Five years or more.

†In most cases, the method of choice is radium therapy using the Ernst applicator and supervoltage. In some cases, supervoltage and transvaginal cone therapy were combined, and rotational cobalt 60 therapy was given in selected cases.

external irradiation, we are now able to get sufficient dosage in the parametria (point B) and pelvic wall. The total dose in most cases is 5,000-6,500 R.

Radiation Center of Fort Worth

The Radiation Center of Fort Worth is a community facility established and operated by a nonprofit organization, the Radiation and Medical Research Foundation of the Southwest. The Center is unique in Fort Worth and West Texas. It does not duplicate the services of, or compete with, any other institution in the area. A community facility offers the most economical means of providing costly radiation equipment and specialized personnel. All qualified radiologists in the area may use its facilities in treating their patients. No patient is turned away for lack of funds. The Center is supported by financially able patients who pay for their treatments, and by continuing contributions for improvements and for free treatment of patients who cannot pay, which is one patient of every four. The Center houses a 2 m.e.v. Van de Graaf unit, a cobalt 60 unit, and all facilities for radioactive isotope work. The radiation Center was opened and its facilities were made available to radiologists of the area in 1958.

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Dr. Archer, 800 Fifth Avenue, Fort Worth 76104.

About Toilet Training . . .

"Rare indeed, in all of nature, is the healthy animal that continues to soil himself or his nest. I believe a little faith would show that Homo sapiens, without special training, is equally capable of such a performance," wrote Dr. S. J. Arnold, Morristown, N.J., to the editor of *Medical Tribune*. In the main, however, his letter was a plea for extensive and thorough examination of patients who have enuresis, since "genitourinary disease may be extremely subtle, extremely difficult to detect, yet extremely pertinent."

Clinicopathologic Conference

James Neil Gilliam, M.D.; John T. Davis, M.D.; Jean D. Wilson, M.D.; Fouad A. Bashour, M.D.; J. R. Crout, M.D.; William Shapiro, M.D.; W. F. Miller, M.D.; Burton Combes, M.D.; and Bruce D. Fallis, M.D.

The patient's previous diagnoses included myotonia dystrophia, chronic atrial flutter, bronchopulmonary disease, and diabetes mellitus. He had pains in his neck and chest and came to the emergency room. After preliminary examination he collapsed and died 24 hours later.

DR. GILLIAM: This 57-year-old white man had past diagnoses of myotonia dystrophia (August, 1963), chronic atrial flutter (April, 1962, Fig. 1), adult-onset diabetes mellitus (September, 1962), and chronic bronchopulmonary disease secondary to his muscle disease. His third and final admission to Parkland Memorial Hospital was on Dec. 6, 1965. He came to the emergency room because for four days he had felt a vague pain in his neck and the right side of his chest. The pain was described as "knife-like" without radiation or pleuritic components. In addition, the patient said he had had a "cold" for the past three or four weeks, that his chronic cough had become more severe, and that his throat became sore one week prior to admission.

A brief physical examination revealed normal temperature and blood pressure of 126/90 mm. of mercury. Bilateral rhonchi with scattered wheezes at the right lung base were heard. He was sent to the Radiology Department for a chest film. When he returned, he suddenly became weak, and his blood pressure fell below a measurable level. An electrocardiogram showed atrial flutter with variable atrioventricular conduction and a ventricular rate of approximately 150 per minute. The ST segments in V_3 through V_6 were elevated (Fig. 2). Aramine therapy was started immediately; the patient was given 100 mg. of Dilantin intravenously and was admitted to the ward.

On admission to the ward the patient was responsive but somnolent. He complained, in garbled speech, of being hungry and of having pain in his neck and throat. Additional history was unobtainable.

His past history, obtained from his old chart, was as follows:

First Admission: The patient entered the Surgery Service in September, 1950, with complaints of mid-epigastric pain and weight loss. An upper gastrointestinal series showed a scarred duodenal bulb without evidence of active disease.

Second Admission: In September, 1962, he was seen in the Medical Service because of weakness, weight loss, pain in lumbar area and lower abdomen, and severe nocturnal muscle cramps. He had also noticed progressive dyspnea on exertion. An ECG revealed atrial flutter. Other studies showed a functioning thyroid nodule on the left lower pole and a diabetic glucose tolerance curve; barium enema and upper gastrointestinal series revealed no abnormalities. Diagnoses were diabetes mellitus, diabetic neuropathy, chronic atrial fibrillation, and functioning thyroid nodule in left lower pole, euthyroid. Orinase and digitoxin were prescribed, and the patient was discharged.

Outpatient Course: He continued to re-

This is the seventh in a series of clinicopathologic conferences from The University of Texas Southwestern Medical School, and Parkland Memorial Hospital, Dallas. Dr. Bruce D. Fallis, professor of pathology, is guest editor for the series. Other participants this month are Drs. James Neil Gilliam, former rotating intern and resident in internal medicine at Parkland Memorial Hospital; John T. Davis, assistant professor of radiology; Jean D. Wilson and William Shapiro, assistant professors of internal medicine; and Fouad A. Bashour, J. R. Crout, W. F. Miller, and Burton Combes, associate professors of internal medicine.

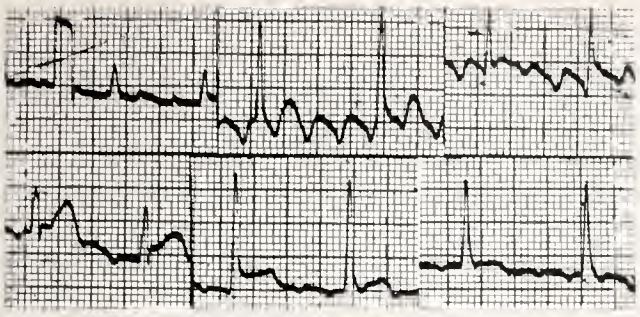

Fig. 1. Electrocardiogram taken April 16, 1962. Top row, left to right, leads I, II, and III. Bottom row, left to right, leads V_4, V_5, and V_6. Atrial flutter with varying atrioventricular conduction and moderate ST elevation in the precordial leads is seen.

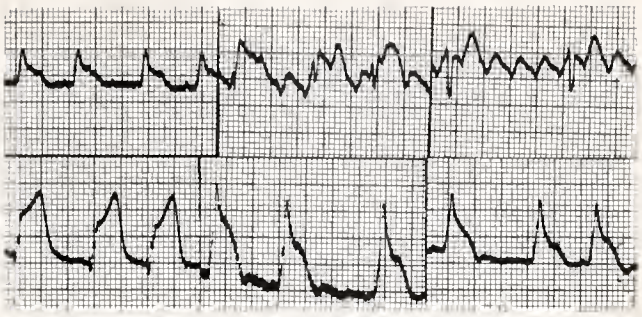

Fig. 2. Electrocardiogram taken Dec. 6, 1965. Top row, left to right, leads I, II, and III. Bottom row, left to right, leads V_4, V_5, and V_6. Note the ST elevation seen in lead I and the precordial leads (contrast with Fig. 1). Atrial flutter with varying atrioventricular conduction remains.

ceive treatment as an outpatient, and his diabetes was controlled with Orinase, 3 gm. per day. He had recurrent episodes of acute bronchitis, for which intermittent courses of antibiotic therapy were given.

In August, 1963, he was seen in the Neurology Clinic. A diagnosis of myotonia dystrophia was made on the basis of bilateral cataracts, frontal balding, wasting of temporalis and sternocleidomastoid muscles, and testicular atrophy. An electromyogram confirmed the clinical impression.

Lung function studies, done in the clinic in September, 1965, revealed a total vital capacity of 2,500 cc. (61 percent of predicted) with a 0.5 second forced expiratory volume of 1,300 cc. (52 percent of predicted).

The patient was last seen in the clinic in October, 1965. He had no specific complaints and was told to continue taking Orinase. Digitoxin had been discontinued several months earlier.

Physical Examination: At the time of his third admission, the patient's pulse was 140 and irregular; blood pressure palpable at 90 mm. of mercury systolic (on Aramine); respirations 16-18 per minute and shallow; and temperature was 98 F. He was thin and appeared to be chronically ill, and his skin was ashen. He had generalized muscular atrophy with pronounced wasting of the neck and temporalis muscles with temporal hollowing and frontal baldness. His pupils were equal and reacted sluggishly to light; bilateral cataracts obscured his fundi. His neck was supple, and a 4 cm. nodule was present in the left lower pole of the thyroid. His neck veins revealed flutter waves without evidence of increased venous pressure. His chest was thin; expansion was symmetrical but of small magnitude, and coarse rales and rhonchi were heard bilaterally both on inspiration and expiration. No wheezes, fine rales, or evidence of consolidation was detected. His heart sounds were muffled. There was no gallop, rub, or murmur, and there was no evidence of cardiomegaly. Abdominal examination was unremarkable. He had bilateral testicular atrophy. He had no clubbing or peripheral edema. Neurological examination revealed uniformly depressed to absent deep tendon reflexes without pathological reflexes, spontaneous movement of all extremities, and no evidence of a focal motor or sensory lesion. Percussion of the thenar muscles produced contraction and adduction of the thumbs with slow relaxation.

Initial laboratory data included hemoglobin, 15.9 gm. per 100 ml.; white blood cells, 12,700 per cu. mm., serum glutamic oxaloacetic transaminase, 682 units; and normal values for serum electrolytes. The sputum contained many neutrophils and a mixture of gram-positive and gram-negative bacteria.

Ward Impression: (1) Acute anterior myocardial infarction; (2) acute and chronic bronchitis; (3) myotonia dystrophia with generalized muscular atrophy, restrictive ventilatory defect, and questionable myotonia heart disease; (4) adult-onset diabetes mellitus.

Hospital Course: (Admitted at 5 p.m., Dec. 6, 1965.) The patient's blood pressure was maintained at 90 mm. of mercury, systolic, with intravenous Aramine therapy. Other treatment included inhalation of oxygen with warm mist, intermittent positive pressure breathing every two hours, and penicillin, 600,000 units intramuscularly, and kanamycin, 500 mg. every 12 hours. His arrhythmia was treated with intravenous Lanoxin. Studies of blood drawn shortly

after the patient had received oxygen revealed values as follows: serum pH, 7.26; carbon dioxide pressure, 128; oxygen saturation, 98.5 volumes percent; carbon dioxide combining power, 24 mEq. per liter. Sodium bicarbonate, 44 mEq. per liter, was added to his intravenous fluids. Digitalis reduced his ventricular rate from 140 on admission to 90-100 by the following morning. It became increasingly difficult to maintain his systolic pressure above 80 mm. of mercury, and the patient became progressively more obtunded and had increasing difficulty handling secretions. A repeat ECG revealed complete left bundle branch block. Because of his rapidly deteriorating clinical condition, marked primarily by ineffective clearing of bronchial secretions due to weak cough and inability to use ventilatory assistance, a tracheostomy was performed, and subsequently constant intermittent positive pressure breathing and intermittent tracheal suction were used. The patient died about five hours later, approximately 24 hours after admission.

QUESTION: Was there a history of muscle disease in the patient's family?

DR. GILLIAM: Members of his family denied any muscle disease. However, at least two of his brothers had the characteristic facial features of myotonic muscular dystrophy, and they had definite myotonia as shown by their difficulty in relaxation when we shook hands with them.

Radiological Findings

DR. DAVIS: A review of the gastrointestinal films taken in 1962 and 1963 shows nothing abnormal.

Chest films made in the emergency room just prior to the patient's admission do not show any infiltrates; the heart is not enlarged. There is hypovascularity of, and some bullous lesions in, both apices. The lung fields are otherwise clear. The bony structures are normal. Little muscle tissue is apparent, which is compatible with what has been described clinically.

DR. BASHOUR: Was there any evidence of left ventricular failure?

DR. DAVIS: I don't think so. The pulmonary vasculature is not prominent.

Clinical Discussion

DR. WILSON: Myotonia dystrophia, or Steinert's disease, is inherited as an autosomal dominant defect and is characterized clinically by muscular weakness and wasting, myotonia, cataracts, frontal baldness, and testicular atrophy. In most cases the disease is mild, and most patients live a normal life span. Only in occasional patients, such as this one, who have unusually severe manifestations of the disease, is there extensive enough involvement of either the respiratory or other muscles that the disease proves fatal. When extensive involvement of skeletal muscles is present, there is often involvement of the heart, just as occurs in progressive muscular dystrophy. In a brief survey of the literature I could not find documentation of an increased incidence of diabetes mellitus in this disease, and apparently this patient has two hereditary diseases, diabetes mellitus, inherited as a recessive trait, and myotonia dystrophia, inherited as a dominant trait.

DR. BASHOUR: Involvement of the heart is frequent in patients with myotonia dystrophia, and the electrocardiograms obtained on this patient are interesting. In April, 1962, the patient had atrial flutter with a ventricular response of 75 per minute. At that time, he had an ST elevation of about 3 mm. in V_4 with T wave inversion in V_6 (Fig. 1). In subsequent electrocardiograms, the ventricular response rate varied between 50 and 60 per minute. On Dec. 7, 1965, the electrocardiogram revealed elevation of the ST segment in V_3 through V_6. The ST elevation was 16 mm. in V_4 and 8 mm. in V_3. Significant ST elevation was also seen in V_1 (Fig. 2). These electrocardiographic changes were not present in April, 1962.

In cases of myotonia dystrophia, diffuse interstitial fibrosis of the myocardium may occur, and many such cases will have ECG abnormalities, sometimes even before involvement of the skeletal muscles becomes clinically manifest.

The most commonly observed electrocardiographic abnormalities include prolongation of the PR interval and of the QRS duration.[1] A few instances of Steinert's disease have been complicated by recurrent episodes of syncope of the Adams-Stokes types, sometimes terminating in sudden death. Atrial flutter was not infrequently seen in patients with myotonia dystrophia.[2]

In some cases of myotonia dystrophia, ST elevations in the precordial leads suggestive of an acute myocardial infarction have been observed in the absence of clinical or labo-

ratory evidence of acute myocardial necrosis. I would assume that the ST elevation noted in his first electrocardiogram of 1962 was the result of myocardial involvement by the dystrophic process and was not the result of myocardial infarction. However, during his last admission, the ST elevation observed in V_1 through V_6 and the disappearance of the R waves (two hours after admission) would definitely suggest an acute myocardial infarction. To my knowledge, this would be the first described case of a patient with myotonia dystrophia with an acute myocardial infarction.

It is important to stress that in this disease the myocardium is frequently involved by the dystrophy, and the addition of myocardial infarction would most likely result in acute myocardial failure. I would agree that the patient had myotonia dystrophia with cardiac involvement, and he died with an acute myocardial infarction.

DR. CROUT: My understanding of your data is that Dilantin is useful as a prophylactic measure for the prevention of ventricular arrhythmias but is perhaps not therapeutic for atrial flutter. If this is so, why was Dilantin given to this patient?

DR. BASHOUR: This is correct. I did not see this patient, but in the protocol it was stated that the ventricular response was 150 per minute. If this patient had ventricular tachycardia at that time, I would have used Dilantin; if ventricular tachycardia was not present, Dilantin was not indicated.

DR. SHAPIRO: The matter of myocardial involvement in myotonia dystrophia has been covered. Although few if any cases of myocardial infarction in myotonia dystrophia have been described, certainly it is a common occurrence in diabetes mellitus, which the patient also had, and the severity of the carbohydrate defect may have no particular relationship to the severity of the complicating atherosclerosis.

I would like to discuss briefly the use of digitalis in an acute myocardial infarction with shock, because there are some interesting data which have become available recently. The use of digitalis in acute myocardial infarction, as was used here, has been under discussion for a long time. For years it was considered dangerous, but numerous clinical studies have shown that when indications are proper, there is no danger in using digitalis in acute myocardial infarction without significant heart block. With the recent demonstration of the positive inotropic effect of digitalis in the absence of heart failure,[3] one might wonder whether there would be any benefits in improving myocardial function in the presence of myocardial infarction with shock, because a number of these patients really have cardiogenic shock with grossly deficient cardiac outputs and tight peripheral vasoconstriction. If the cardiac output is low enough, blood pressure cannot be maintained by peripheral vasoconstriction.[1] In a recent, well controlled study of myocardial infarction due to coronary embolization,[4] the central aortic pressure, the cardiac output, and the rate of rise of ventricular pressure dropped precipitously. The rapid administration of digitalis to this experimental preparation dramatically increased all three functions. Although these parameters were not returned to normal, they were grossly improved over those in similarly prepared animals that did not receive digitalis. Therefore, the administration of digitalis may give considerable benefit to the patient with acute myocardial infarction and shock, even in the absence of arrhythmias or overt congestive failure.

DR. MILLER: Involvement of the respiratory system in muscular dystrophies is largely a consequence of impaired ventilatory function with secondary impairment of bronchial clearing mechanisms, resulting in chronic bronchitis and, eventually, localized or generalized hypoventilation. Localized hypoventilation leads to the development of atelectasis.

Another manifestation seen not infrequently in patients with muscular disorders is pulmonary embolization. This is a consequence of the patient's immobile state and his general physical disability with resultant peripheral venous thrombosis. Furthermore, with impaired ventilation the susceptibility of lungs to damage as a result of emboli is considerably increased.

I doubt that anything could have been done for this patient to alter the outcome. The case has certain interesting features which allow us to discuss problems of therapy relative to the respiratory system in patients with muscular weakness. This man was suffering from cardiorespiratory insufficiency. The studies that are reported in the protocol indicate that he had rather severe discrepancies in the distribution of ventilation with respect to pulmonary blood flow that would have caused severe hypoxia.

Hypoxia was not demonstrated by the blood gas studies, because they were done after the patient received added concentrations of oxygen. However, one can assess the potential magnitude of his hypoxic disturbance by examining the relationship between the alveolar oxygen tension at the level of oxygen he was breathing and his arterial oxygen tension. With warm mist oxygen, it is likely that his inspired oxygen concentration was about 35 percent oxygen. This would result in an alveolar oxygen tension of approximately 230 mm. of mercury, yet his arterial oxygen tension was only 128, a discrepancy of 102 mm. This indicates that significant areas of lung were being perfused with blood but were not being adequately ventilated. In addition, his pH was 7.26, while his pCO_2 was only 45. If we apply these data to the Siggard-Anderson nomogram, we find that he had a base deficit of 10 mEq. This, coupled with the evidence of an ion gap, would be fairly strong evidence that he not only had mild respiratory acidosis but also metabolic acidosis, probably due to lactic acidemia.

A recent myocardial infarct with shock is often considered an absolute contraindication to ventilatory assistance. However, if inadequate ventilation is an indication for ventilatory assistance, it is never contraindicated, no matter what the circumstances may be. The hemodynamic disturbances that may occur with assisted ventilation are usually a result of inappropriate application of ventilatory assistance. If ventilatory assistance is properly applied, it need not be detrimental to the patient. Increasing intrathoracic pressure with positive airway pressure may decrease venous return to the heart, unless peripheral venomotor reflexes are intact so that venous pressure rises appropriately to maintain the filling gradient. The pressures for ventilatory assistance likely will not exceed 30 cm. of water pressure. If this causes decreased venous return, decreased cardiac output, and lowered blood pressure, the deficit is corrected simply by placing the patient in a supine position, or in addition, elevating his legs, applying pressure leg wraps, or an abdominal binder. In some instances peripheral vasoconstrictor drugs have also been used as a means of supporting venous return, but this should not be the first choice, because in patients who are severely hypoxic or have pulmonary complications vasoconstrictors may cause further decrease of pulmonary blood flow. As a primary rule one should not employ more ventilating pressure than is necessary to support the ventilation without lowering the patient's blood pressure to dangerous levels, and when higher pressures are necessary the previously mentioned measures can be used to restore effective venous return.

In the long-range management of patients with muscular weakness, a good prophylactic pulmonary program is essential, because most of these patients eventually die of respiratory failure. Essentially, the program is the same as that for patients with any kind of chronic inflammatory bronchial disease with ventilatory impairment.

1. Remove all forms of bronchial irritants from the patient's environment, especially tobacco smoke.

2. Provide for intermittent use of a bronchodilator aerosol and periods of mist inhalation to help mobilize bronchial secretions or bronchial exudates that may develop during intercurrent respiratory infections or other inflammation. Aerosol therapy should be intensified at times of ordinary respiratory infection, because the patients retain more bronchial secretions at such times.

3. If the patient's ventilatory capacity is reduced to the point where he cannot cough effectively, he should have mechanical assistance. As a rough criterion, one can utilize the ventilatory function tests as an index of whether ventilatory capacity is compatible with an effective cough. Usually, if the forced expiratory volume in a half second is reduced below 600 cc., or is less than 1,000 cc. in one second, the patient cannot cough effectively. Our patient's forced expiratory volume was not reduced to that extent, so his problem was not primarily one of ineffective cough.

Another important aspect of treatment is the availability of periodic deep breathing. Our patient's vital capacity of 2,500 cc. represents probably less than half of his total lung volume, which means that there was a significant amount of his lung that he was never able to expand completely because of muscular weakness. These incompletely expanded parts of the lung are potential sites of atelectasis and infection. A normal person periodically takes very deep breaths or sighs, thus expanding all parts of his lungs. Any patient who cannot take periodic deep breaths, whether because of muscular weak-

ness, a disturbed mental state, or other cause, needs supplementary aid with some form of mechanical device to allow him, periodically, to take extra-deep breaths, like a sigh, and to open the parts of his lungs that he is not utilizing for breath-by-breath ventilation.

DR. COMBES: An interesting protein abnormality in myotonic dystrophy has been described recently.[5] Serum concentrations of immunoglobulin G (IgG) were decreased by approximately 50 percent, on the average, in 19 patients who had this disorder. Total circulating and exchangeable pools of IgG were similarly reduced. These alterations were attributed to an accelerated catabolism of IgG. The rate of IgG synthesis was normal. The metabolic alteration is apparently caused by factors in the host, rather than by intrinsic abnormalities of the IgG. Thus, IgG prepared from a patient with myotonic dystrophy is catabolized normally when administered to normal controls. By contrast, IgG from a normal donor is catabolized at an increased rate by patients with myotonic dystrophy. Serum concentration and rates of catabolism of the other immunoglobulins, IgA and IgM, and of albumin were normal. The factors responsible for the isolated hypercatabolism of IgG are as yet unknown. Since an immunoelectrophoretic pattern of our patient's serum is not available, we cannot know whether he demonstrated a decrease in serum IgG concentration. The metabolic abnormality described by Wochner and associates[5] may serve as a means for examining the genetic transmission of this disorder.

Pathological Findings

DR. FALLIS: The patient did, of course, have myotonic muscular dystrophy, or Steinert's disease. This was established by the characteristic clinical findings, and by the existence of the disease in several brothers. Furthermore, the characteristic anatomic findings were also present at necropsy.

At the time of autopsy the patient was 5 feet 6 inches tall and weighed 100 pounds, had frontoparietal baldness, and was thin and wasted. The region of the temporal muscle on each side was depressed, and both temporalis muscles were thin and parchment-like. The sternocleidomastoid muscles were not apparent on external examination. When the skin was reflected from the neck, the sternocleidomastoid and strap muscles of the neck were represented only by thin sheets of fibrous tissue which blended with the fibrous tissue of the cervical fasciae. There was general wasting of the various muscles of the body, especially of the distal muscles of the extremities. The muscles of the thighs were the best preserved.

Microscopically, the various types of muscular dystrophy are characterized by involvement of individual muscle fibers, and both large fibers and small atrophic fibers are found in the same fascicle. Myotonic muscular dystrophy is usually a relatively mild disease, and the histologic changes are slight, consisting largely of variation of size of individual muscle fibers, invagination of the sarcolemmal membrane, and appearance of characteristic central nuclear chains. In this patient, however, the muscle changes, both grossly and microscopically, were considerably worse than in the usual case of myotonic dystrophy. There was considerable variation in the diameter of the individual skeletal muscle fibers, numerous chains of centrally located nuclei were present, and

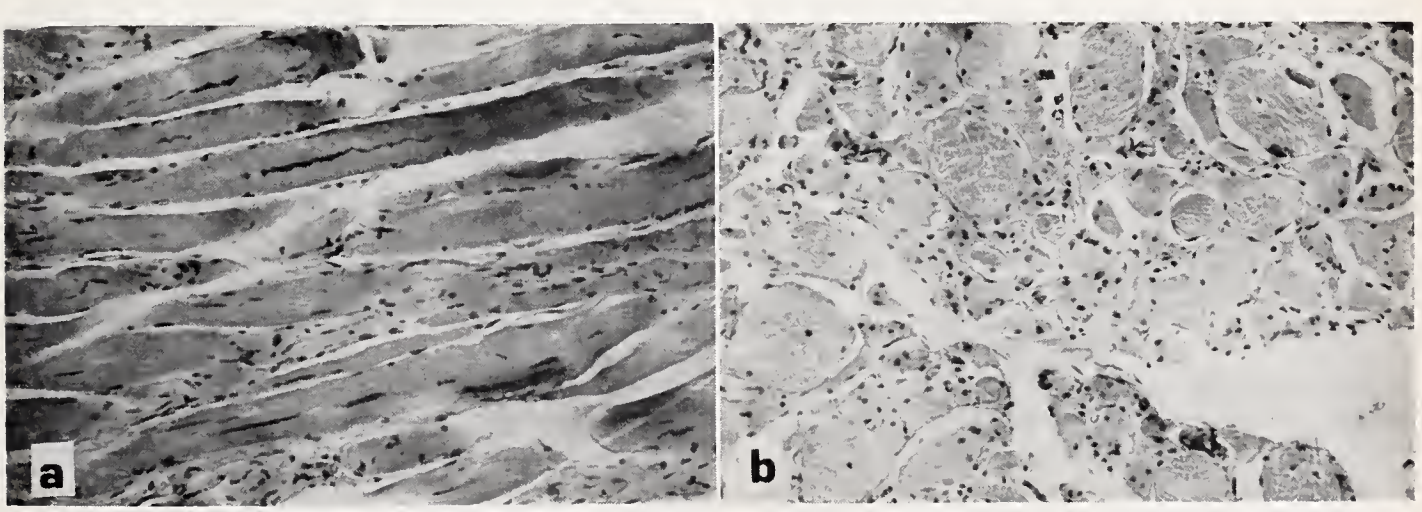

Fig. 3. Photomicrographs of skeletal muscle (low magnification) in longitudinal (a) and cross section (b). Note the central nuclear chains in the former, and the variation of fiber size in the latter.

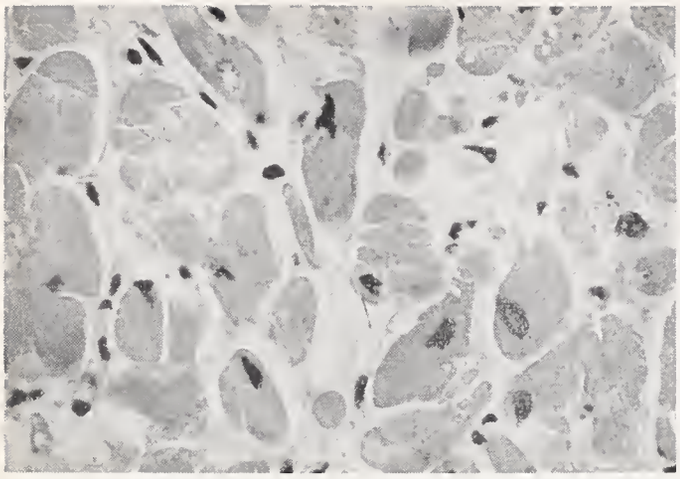

Fig. 4. Photomicrograph of myocardium (high magnification). Note the variation in fiber size. Compare with skeletal muscle seen in Fig. 3b.

Fig. 5. Photomicrograph of myocardium (low magnification). Acute myocardial infarct. The myocardial fibers are the seat of coagulation necrosis with darkly staining cytoplasm and loss of nuclei (left); the small dark spots between the necrotic muscle fibers are neutrophils. Note the viable myocardial cells just under the left ventricular endocardium (right).

some of the muscle fibers had undergone severe atrophy and consisted only of short chains of nuclei with practically no cytoplasm (Fig. 3). There was also slight interstitial fibrosis.

The heart weighed 320 gm. There was only slight coronary arteriosclerosis with slight luminal narrowing but no occlusion, either recent or old. The heart was extremely dilated, and the myocardium was thin, quite flabby, and yellowish-tan, indicating dystrophic involvement of the cardiac muscle. Microscopic study confirmed the dystrophic involvement; the major change was variation in diameter of myocardial fibers (Fig. 4). On the cut surface of the myocardium there was a large, recent transmural myocardial infarct. This infarct involved the anterior, lateral, and, to a lesser extent, the posterior wall of the left ventricle. In the infarct the myocardium was discolored, opaque, and gray-yellow. At the margins of this infarct, extending for about a centimeter into the surrounding myocardium, there was a blotchy, reddish discoloration of the myocardium, indicating a fresher zone of infarction. The main part of the infarct was about four days old, and apparently occurred at about the time he began to complain of pain in his chest and neck. The infarct had extended probably at the time of his collapse in the emergency room about 24 hours prior to his death. Microscopic study confirmed the gross changes of myocardial infarction (Figs. 5 and 6). In addition to the large myocardial infarct there was fatty degeneration of much of the remaining myocardium, indicating anoxic damage to the myocardium in regions outside the frank infarct.

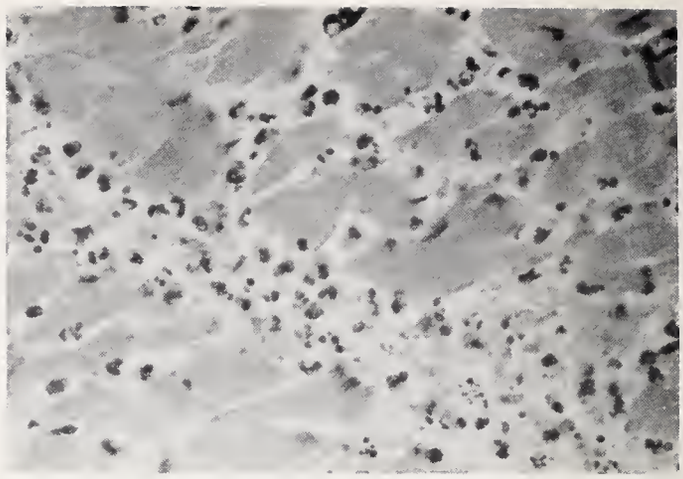

Fig. 6. Photomicrograph of myocardium (high magnification). Acute myocardial infarct. Coagulation necrosis of myocardium with neutrophilic infiltrate.

With regard to the cause of the myocardial infarct, it is apparent that it was not due to coronary arteriosclerosis per se, because the degree of coronary arteriosclerosis was slight, without significant luminal narrowing, even though the patient was diabetic. Presumably the patient suffered an arrhythmia which temporarily decreased cardiac output and coronary blood flow, and this precipitated the infarct. The subsequent extension was probably caused by a recurrence of the arrhythmia.

In addition to involvement of skeletal and cardiac muscle, myotonic dystrophy also causes changes in smooth muscles. The histologic changes in the smooth muscles are not severe, but functional abnormalities have been described in the hypopharynx, esophagus, stomach, intestines, gallbladder, urinary bladder, anal sphincter, and uterus. The hypopharynx and the upper part of the

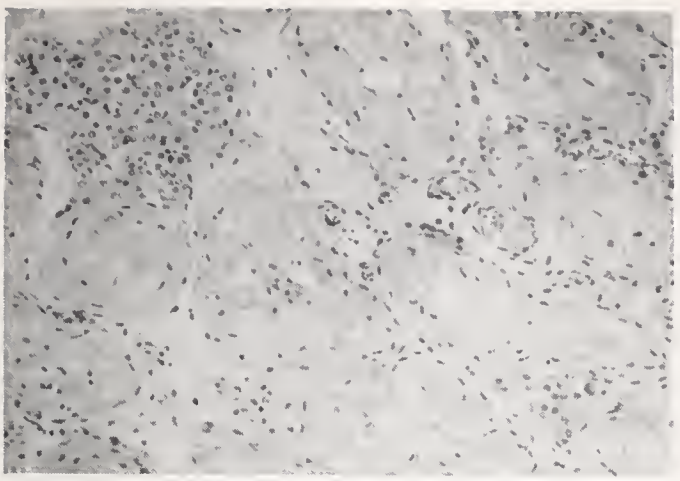

Fig. 7. Photomicrograph of testis (low magnification). The tubules are atrophic and consist largely of thickened basement membranes. A cluster of interstitial cells is present in this section (upper left).

esophagus of our patient were dilated with thin, flabby walls. Microscopic study confirmed the presence of dystrophic changes in the muscle coats. Dystrophic change in the muscles of the hypopharynx is common in persons with Steinert's disease and often causes dysphagia. The onset of dysphagia is usually gradual and may not be apparent to the patient, but it can be detected by careful clinical study. This dysphagia, in association with a poor gag and cough reflex, may cause recurrent aspiration and, eventually, pulmonary fibrosis.

The lungs weighed 1,300 gm. There was slight chronic bronchitis and obstructive emphysema. This could be the result of recurrent aspiration in association with his Steinert's disease, but the same changes could be produced by smoking or other chronic bronchial irritants. Acute pulmonary congestion and edema were present, obviously the result of the acute myocardial infarct. A tracheostomy was present, and the smaller bronchi contained abundant mucopurulent exudate. Associated with this mucopurulent bronchitis were foci of atelectasis, especially in the right lower lobe.

The liver weighed 1,160 gm. and was acutely congested with hydropic degeneration. There was a single pure cholesterol gallstone in the gallbladder, but no cholecystitis. The remainder of the extrahepatic biliary tree was normal. The pancreas, including the islands of Langerhans, was normal on gross and microscopic study. Diabetes mellitus has been described in patients with myotonic dystrophy, but whether the incidence of diabetes is higher than in the general populace is uncertain. The stomach was dilated; whether this was a reflection of smooth muscle involvement by his dystrophy or simply reflects an agonal event is uncertain.

The right kidney showed slight hydronephrosis, and there was dilatation of the right ureter. At the point where the right ureter entered the urinary bladder, it had lost its usual oblique course, which constitutes the ureterovesical valve. Instead, the ureter entered the bladder at a right angle to its wall. The urinary bladder was distended. Acute distention of the urinary bladder is a common agonal event, and it is difficult to ascribe the dilated bladder to myotonic dystrophy. However, the presence of hydroureter and hydronephrosis on the right in the absence of a mechanical obstruction is good evidence that the patient did in fact have smooth muscle dysfunction of the urinary tract. Chronic urethritis was present, but there was no cystitis or pyelonephritis.

The brain and spinal cord were entirely normal, and there were no abnormalities of the peripheral nerves.

The testes were small and brown, and microscopic examination revealed severe atrophy. Most of the tubules were reduced to hyaline cuffs, representing the thickened basement membranes, with no epithelium remaining (Fig. 7). The interstitial cells were normal.

The left adrenal gland was normal, but the right gland consisted of a small amount of gray tissue, representing the medulla, and a few minute nodules of yellow cortical tissue embedded in dense fibrous tissue. This postnecrotic scarring, or as it is sometimes inappropriately called, primary cortical atrophy, is one of the lesions that causes Addison's disease when it is bilateral. In our case, however, it was unilateral. The cause of this lesion is not certain, but is probably a temporary reduction of blood flow through the long sinusoids of the adrenal cortex. It is probable that this resulted from periods of cardiac arrhythmia in our patient. Why the lesion should be unilateral I cannot say. The thyroid was normal in size and overall shape. On cut section there were several colloid nodules; they were, however, not as large as they were described clinically. The one in the left lower pole measured only 12 mm. in diameter. The pituitary gland was normal.

In summary, the patient had myotonic muscular dystrophy with involvement of skeletal muscle, myocardium, and several viscera, notably the hypopharynx, esopha-

gus, and urinary tract. The immediate cause of death was an acute, transmural, anteroapical and lateral myocardial infarct, precipitated presumably by the cardiac arrhythmia from which the patient suffered. The myocardial infarct occurred four days prior to death and extended about a day prior to death.

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Dr. Gilliam, Goldthwaite, Texas.

Drs. Davis, Wilson, Bashour, Crout, Shapiro, Miller, Combes, and Fallis, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas 75235.

Policyholders Receive Sixth Dividend

Dr. Laurance N. Nickey, Chairman, Committee on Association Insurance Programs, announced that the sixth consecutive cash dividend has been paid under the Texas Medical Association Group Disability Insurance Program. The dividend of $133,000 was distributed pro rata to members insured during the policy year ending Oct. 31, 1966.

Dr. Nickey pointed out that this program continues to be the leader among plans of this type. In addition to the cash dividends to all policyholders, the original plan, which provided for benefits for three years, has been improved four times at no increase in premium, and it now includes long-term sickness benefits, payable to age 65 for all disabilities which occur prior to age 60. For all disabilities which occur between age 60 and 70, benefits are payable for a maximum of five years, but not beyond age 72.

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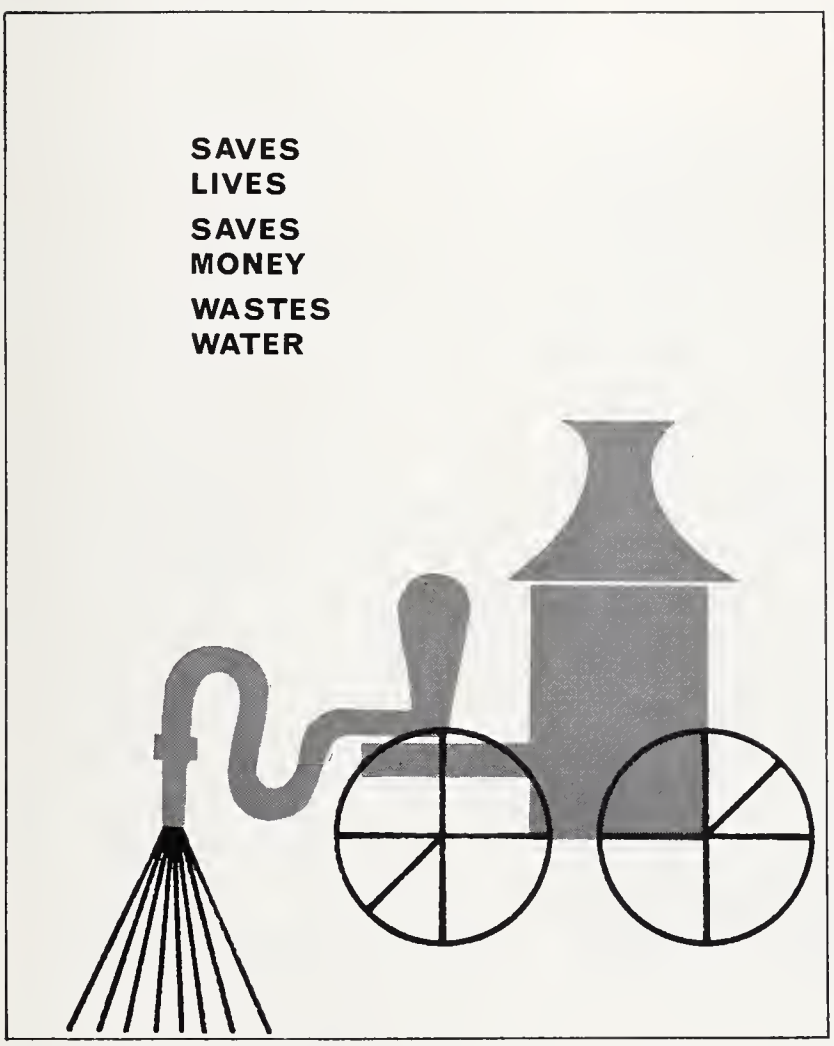

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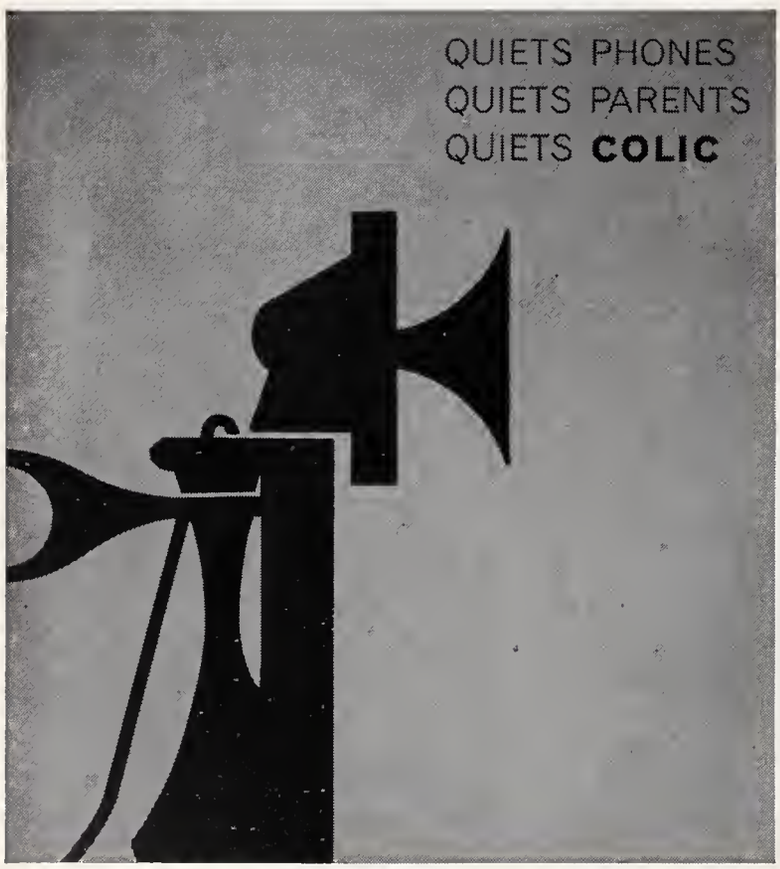

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Texas Medical Association

SYMPOSIUM FOR COUNTY MEDICAL SOCIETY OFFICERS

Saturday, January 21, 1967

Headquarters Building

TEXAS MEDICAL ASSOCIATION

Austin, Texas

FIRST MORNING SESSION
LIBRARY STACK AREA

9 a.m. ***PANEL SESSION: STIMULATING AND EVALUATING COUNTY MEDICAL SOCIETY ACTIVITIES***

Presiding:
WALTER WALTHALL, M.D., San Antonio
Chairman
Board of Councilors

1. ***STIMULATING GREATER PARTICIPATION IN COUNTY MEDICAL SOCIETIES***
C. LINCOLN WILLISTON, Austin
Executive Secretary
Texas Medical Association

2. ***A PRACTICAL APPROACH TO IMPROVING COUNTY MEDICAL SOCIETY PARTICIPATION AND OPERATION—USE OF THE COUNTY MEDICAL SOCIETY MANAGEMENT SURVEY***
JACK M. PARTAIN, M.D., San Antonio
Past President
Bexar County Medical Society

3. ***IMPLEMENTING THE SURVEY RECOMMENDATIONS***
JOHN C. MEADOWS, M.D., San Antonio
Chairman, Ad Hoc Committee to Review the Survey Report and To Make Recommendations
Bexar County Medical Society

4. ***THE IMPACT OF THE MANAGEMENT SURVEY ON THE COUNTY MEDICAL SOCIETY***
SID E. COCKRELL, JR., San Antonio
Executive Director
Bexar County Medical Society

QUESTIONS AND DISCUSSION

Members of Texas Medical Association, new and transfer members attending the Orientation Program, county medical society officers, and members of the Woman's Auxiliary to Texas Medical Association will meet jointly for the Conference on Legislation beginning at 11 a.m.

ORIENTATION PROGRAM

Saturday, January 21, 1967

Headquarters Building

TEXAS MEDICAL ASSOCIATION

Austin, Texas

FIRST MORNING SESSION
SAM THOMPSON ROOM

9 a.m. Presiding:
WENDELL H. HAMRICK, M.D., Houston
Vice-Speaker, House of Delegates
Texas Medical Association

9:10 a.m. ***AN ACCOUNTING OF STEWARDSHIP; WHAT HAPPENS TO YOUR DUES***
ELLIOTT MENDENHALL, M.D., Dallas
Vice-Chairman, Board of Trustees
Texas Medical Association

9:30 a.m. ***MEDICAL ETHICS—OBLIGATIONS OF THE PHYSICIAN TO HIS COLLEAGUES AND TO THE PROFESSION***
JAMES H. SAMMONS, M.D., Highlands
Vice-Chairman, Board of Councilors
Texas Medical Association

9:50 a.m. ***DOCTOR-PATIENT RELATIONS***
WALTER WALTHALL, M.D., San Antonio
Chairman, Board of Councilors
Texas Medical Association

10:10 a.m. ***LEGAL ASPECTS OF MEDICINE; MALPRACTICE—HOW TO AVOID IT***
SAM V. STONE, JR., LL.B., Austin
Office of the General Counsel
Texas Medical Association

10:45 a.m. ***COFFEE BREAK***

Texas Medical Association

CONFERENCE ON LEGISLATION

Saturday, January 21, 1967

Headquarters Building

TEXAS MEDICAL ASSOCIATION

Austin, Texas

Members of Texas Medical Association, new and transfer members attending the Orientation Program, county medical society officers, and members of the Woman's Auxiliary to Texas Medical Association will meet jointly for the Conference on Legislation beginning at 11 a.m.

FIRST GENERAL SESSION
SAM THOMPSON ROOM

Presiding:
JAMES D. MURPHY, M.D., Fort Worth
President
Texas Medical Association

11 a.m. ***THE PRICE OF LEADERSHIP***
HON. PRESTON SMITH, Lubbock
Lieutenant Governor
State of Texas

11:30 a.m. ***SOCIO-ECONOMIC TRENDS AND THEIR EFFECTS UPON THE PRACTICE OF MEDICINE***
ERNEST B. HOWARD, M.D., Chicago
Assistant Executive Vice-President
American Medical Association

12 noon ***DRUGS, GENERIC NAMES, AND THE 90TH CONGRESS***
C. JOSEPH STETLER, LL.B., Washington, D.C.
President
Pharmaceutical Manufacturers Association

12:30 p.m. ***LUNCHEON—BASEMENT***
Catering by Walter Jetton, Fort Worth
Compliments of the Texas Medical Association

SECOND SESSION
SAM THOMPSON ROOM

1:30 p.m. ***PANEL SESSION: MEDICAL AND HEALTH ISSUES BEFORE THE 1967 STATE LEGISLATURE***

Presiding:
HOWARD R. DUDGEON, JR., M.D., Waco
President-Elect
Texas Medical Association

IMPLEMENTATION OF TITLE XIX IN TEXAS
CHARLES MAX COLE, M.D., Dallas
Chairman, TMA Special Committee to Study Title XIX
Texas Medical Association

THE NEED TO EDUCATE MORE PHYSICIANS IN TEXAS; LEGISLATION RELATED TO MEDICAL EDUCATION
CHARLES A. LEMAISTRE, M.D., Austin
Vice Chancellor for Health Affairs
The University of Texas

THE NEED FOR MORE NURSES; PROPOSED AMENDMENTS TO THE NURSE PRACTICE ACT
ROBERT W. GRACE, M.D., Dallas
Committee on Nursing
Texas Medical Association

MENTAL HEALTH AND MENTAL RETARDATION
P. C. PALASOTA, M.D., Abilene
Chairman, Committee on Mental Health
Texas Medical Association

LABORATORY LICENSING BILL; LICENSING OF PARAMEDICAL GROUPS; OTHER ISSUES OF MEDICAL AND HEALTH INTEREST
PHILIP R. OVERTON, LL.B., Austin
General Counsel
Texas Medical Association

QUESTIONS AND DISCUSSION

CLOSING SESSION
SAM THOMPSON ROOM

Presiding:
JOHN K. GLEN, M.D., Houston
Vice-President
Texas Medical Association

3:45 p.m. ***ISSUES FACING THE 90TH CONGRESS***
HON. OMAR BURLESON, M.D., Anson
Representative, United States Congress
17th District of Texas

4:15 p.m. ***ISSUES FACING THE 60TH STATE LEGISLATURE***
REP. BEN BARNES, DeLeon
Speaker, House of Representatives
State of Texas

4:45 p.m. ***ADJOURNMENT***

6:30 p.m. ***HOSPITALITY HOUR***
Texas Employers' Insurance Association, Host
Colonnade Room, Commodore Perry Hotel

NEWS

Medical Legislation, Leadership Needs To Be Viewed at Conference, Symposium

Texas medical leaders will convene in Austin on Saturday, Jan. 22, at the Texas Medical Association's 1967 Conference on Legislation and Symposium for County Medical Society Officers. The Conference, at which physicians will study current legislative issues concerning medical personnel, will take place at the TMA Headquarters Building, 1801 N. Lamar Blvd.

LT. GOV. SMITH

Five main speakers will enlighten physicians more fully on issues before the United States Congress and the Texas Legislature, on physician leadership responsibilities, and on socioeconomic trends as they affect medicine.

An orientation program and a Symposium for County Medical Society Officers will be held separately at 9 a.m. Both of these groups, along with members of the Woman's Auxiliary to the Texas Medical Association, TMA committee and council officers and members, and other interested persons will then meet together for the Conference on Legislation, beginning at 11 a.m.

The first Legislative Conference speaker will be the Hon. Preston Smith of Lubbock, lieutenant governor of Texas. He will speak on "The Price of Leadership."

Lieutenant Governor Smith began his Texas political career in 1944 when he was elected to the state House of Representatives. He served in the House for six years. In 1956 he was returned to the legislature as state senator and served six years in the Senate. He was elected lieutenant governor in 1962 and has held that post until the present time. He has been a supporter of fiscal responsibility in government.

Dr. Ernest B. Howard of Chicago, assistant executive vice-president of the American Medical Association, will speak to the Conference on "Socioeconomic Trends and Their Effects Upon the Practice of Medicine."

DR. HOWARD

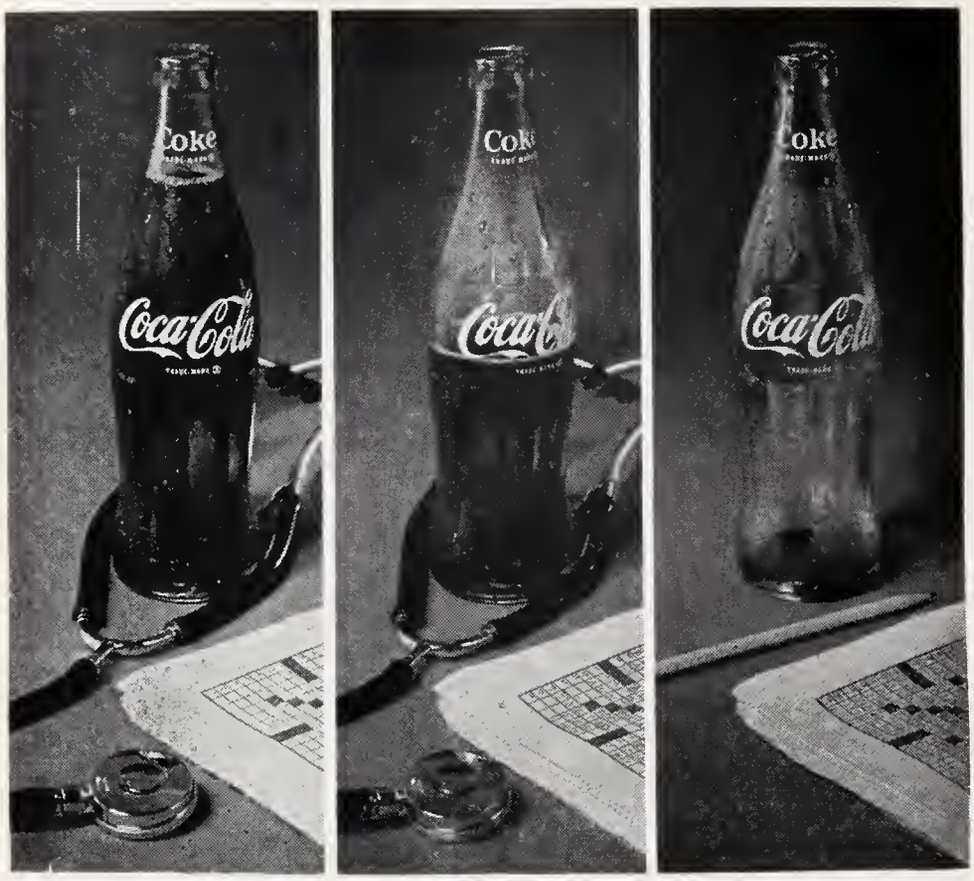
Dr. Howard has held executive positions with the AMA since 1948 when he was appointed assistant general manager. He had previously served as a division director in the Massachusetts Department of Public Health, assistant director of a US Army preventive medicine program, and chief of the US Health Mission to Peru.

"Drugs, Generic Names, and the 90th Congress" will be the topic discussed by C. Joseph Stetler, LL.B., of Washington, D. C., president of the Pharmaceutical Manufacturers Association. Mr. Stetler has served various agencies of the federal government and as general counsel and director of the Legal and Socio-Economic Division of the AMA. He joined the staff of the Pharmaceutical Manufacturers Association in 1963 as executive vice-president and general counsel. He became president of the association in 1965. He is co-author of the following books: *Doctor, Patient and the Law*, and *Handbook of Legal Medicine*. He is a member of the Chicago, Illinois, District of Columbia, and American Bar associations.

MR. STETLER

Two main speakers will highlight the afternoon sessions of the Conference. They are the Hon. Omar Burleson of Anson, US Congressional Representative from the 17th District of Texas, and the Hon. Ben Barnes, speaker of the Texas House of Representatives. Rep. Burleson will speak on "Issues Facing the 90th Congress," and Speaker Barnes will present "Issues Facing the 60th State Legislature."

REP. BURLESON



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David Lipsher, Ph.D.
John Gossett, Ph.D.
Dale R. Turner, Ph.D.

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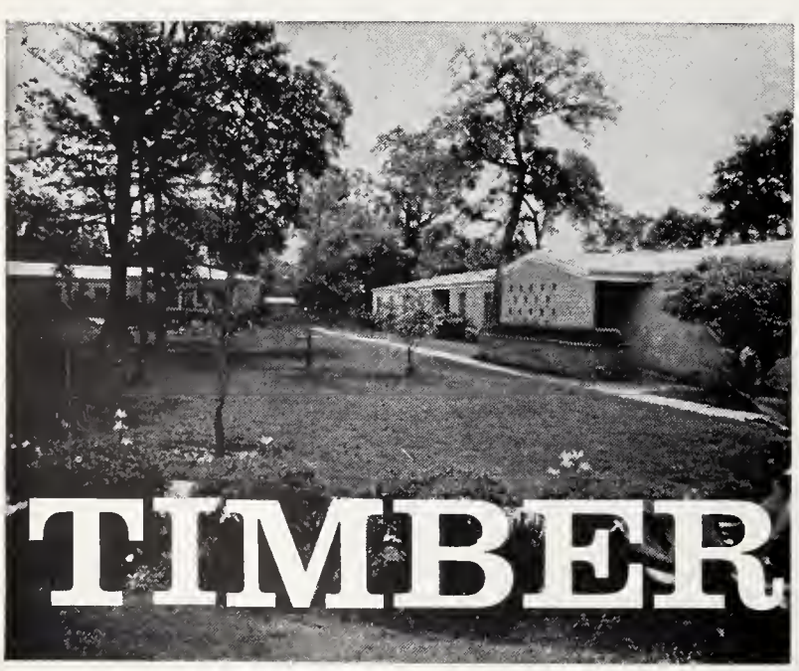

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A panel discussion will also highlight the program of the Legislative Conference afternoon session. The panel participants will consider the subject, "Medical and Health Issues Before the 1967 State Legislature." Presiding officer will be Dr. Howard R. Dudgeon, Jr., of Waco, President-elect of TMA. Speakers on the panel and their speech subjects will be Dr. Charles Max Cole of Dallas, chairman, TMA Special Committee to Study Title XIX, "Implementation of Title XIX in Texas"; Dr. Charles A. LeMaistre of Austin, vice-chancellor for health affairs at the University of Texas, "The Need to Educate More Physicians in Texas"; Dr. Robert W. Grace of Dallas, member of the TMA Committee on Nursing, "The Need for More Nurses: Proposed Amendments to the Nurse Practice Act"; Dr. P. C. Palasota of Abilene, chairman of the TMA Committee on Mental Health, "Mental Health and Mental Retardation"; and Philip R. Overton, LL.B., Austin, general counsel to TMA, "Laboratory Licensing Bill, Licensing of Paramedical Groups, Other Issues of Medical and Health Interest."

A discussion, question, and answer period will follow the presentations by panel members.

Officers Symposium Planned

A Symposium for County Medical Society Officers will take place at 9 a.m., Jan. 21. Presiding over the session will be Dr. Walter Walthall of San Antonio, chairman of the TMA Board of Councilors.

Speakers participating in the symposium and their discussion topics are C. Lincoln Williston of Austin, executive secretary, TMA, "Stimulating Greater Participation in County Medical Societies"; Dr. Jack M. Partain of San Antonio, past president, Bexar County Medical Society, "A Practical Approach to Improving County Medical Society Participation and Operation —Use of the County Medical Society Management Survey"; Dr. John C. Meadows of San Antonio, chairman, Ad Hoc Committee To Review the Survey Report and To Make Recommendations, Bexar County Medical Society, "Implementing the Survey Recommendations"; and Sid E. Cockrell, Jr., of San Antonio, executive director, Bexar County Medical Society, "The Impact of the Management Survey on the County Medical Society."

These presentations will be followed by a discussion period in which questions may be asked by the audience.

Orientation Program Set

Also meeting at 9 a.m. on Jan. 21 will be new and transfer members of TMA.

Presiding over the Orientation Program will be Dr. Wendell H. Hamrick of Houston, vice-speaker of the TMA House of Delegates. Participating speakers and their subjects will be Dr. Elliott Mendenhall of Dallas, vice-chairman, TMA Board of Trustees, "An Accounting of Stewardship: What Happens to Your Dues"; Dr. James H. Sammons of Highlands, vice-chairman, TMA Board of Councilors, "Medical Ethics—Obligations of the Physician to His Colleagues and to the Profession"; Dr. Walter Walthall of San Antonio, chairman, TMA Board of Councilors, "Doctor-Patient Relations"; and Sam V. Stone, Jr., LL.B., of Austin, office of the general counsel to TMA, "Legal Aspects of Medicine; Malpractice—How to Avoid It."

Other Activities Scheduled

Other scheduled activities to take place on the weekend of the Conference on Legislation include TMA committee and council meetings, meeting of the Woman's Auxiliary, a luncheon, and a hospitality hour.

Approximately 40 TMA committees and councils will meet to conduct Association business during weekend activities beginning on Friday night, Jan. 20, and ending with the Executive Board meeting on Sunday morning, Jan. 22.

The Woman's Auxiliary will meet separately at the TMA Headquarters Building at 9 a.m. Jan. 21, and jointly with the TMA members attending the Conference on Legislation after 11 a.m. Saturday.

A luncheon, catered by Walter Jetton of Fort Worth, will be served at noon on Saturday, Jan. 21, to the attending TMA members and guests, compliments of TMA.

Texas Employers' Insurance Association will sponsor a hospitality hour at the Colonnade Room of the Commodore Perry Hotel at 6:30 p.m. on Jan. 21.

TMA Woman's Auxiliary To Meet Jan. 21 in Austin

The Woman's Auxiliary to the Texas Medical Association will meet in Austin on Jan. 21 at the TMA Headquarters Building.

The Auxiliary will meet separately from 9 to 10:45 a.m. Saturday and jointly with members of TMA from 11 a.m. to 5 p.m.

Speakers for the 9 a.m. session will include Dr. Edmund M. Fountain of Houston, previously a neurosurgeon on the *S.S. Hope* in Ecuador, in Peru, and in Africa; and Mrs. Sibyl Hamilton of Dallas, city councilwoman and wife of Dr. C. F. Hamilton, TMA member. Dr. Fountain will speak on "Project HOPE," and Mrs. Hamilton will speak on "The State of Affairs." A panel discussion on health careers will follow the speeches.

Auxiliary members will be served a complimentary luncheon with TMA members in the basement of the Headquarters Building at noon Jan. 21.

International Medical Assembly Of Southwest Texas To Meet

The International Medical Assembly of Southwest Texas will hold its 31st annual session at the Gunter Hotel in San Antonio, Jan. 23-25, 1967.

Seventeen guest speakers will be on the program. They include Dr. David M. Little, Jr., Hartford, Conn.; Dr. Leo H. Criep, Pittsburgh, Pa.; Dr. A. D. Dennison, Jr., and Dr. Frank Vellios, both of Indianapolis, Ind.; Dr. Morton H. Maxwell, Los Angeles; Dr. Demetrio Sodi Pallares, Mexico City; Dr. Priscilla White, Boston; Dr. D. E. Cannell, Toronto, Ontario, Canada; and Dr. William J. Mellman, Philadelphia.

Other guest speakers are Dr. William Preston Wilson and Dr. Goe Welton, both of Durham, N. C.; Dr. John R. Hodgson, Rochester, Minn.; Col. Daniel C. Campbell, Jr., M.C., Scott Air Force Base, Ill.; Dr. William D. Logan, Jr., Atlanta, Ga.; Dr. Owen H. Wangensteen, Minneapolis, Minn.; Dr. David Hume, Richmond, Va.;

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The scientific program is acceptable for 15 accredited hours by the American Academy of General Practice.

Social events for the physician and his wife will include for the first time a post-convention trip to Mexico City.

Dr. John H. Bohmfalk, 227 E. Hildebrand, San Antonio, is president of the assembly.

Southwest Allergy Forum Set in Galveston Jan. 19-21

The scientific program of the 1967 Southwest Allergy Forum Meeting will emphasize the practical aspects of the practice of allergy. The meeting will take place Jan. 19-21 at the Flagship Hotel in Galveston.

Scientific papers to be presented include "Allergic Reactions in Patients with Immunologic Deficiencies," Dr. Armond S. Goldman, Galveston; "Hypnosis as an Adjunct for Treating Allergic Patients," Dr. Thomas M. Tyndall, Beaumont; "Present Status of Testing for Penicillin Hypersensitivity," Dr. John P. McGovern, Houston; "A Further Consideration of Insect Sting Allergy," Dr. Boen Swinny, San Antonio; "Report of Progress of Allergy Committee for Study of Repository Therapy," Dr. Mason Lowance, Atlanta, Ga.; "The Role of Regional Factors in Some Allergic Dermatoses Observed in Mexico," Dr. Jose Luis Cortes, Mexico City; and "Immunologic Studies on Mammalian Epidermals," Dr. Lloyd Crawford, Memphis, Tenn.

Panel presentations on the program will include "Practical Consideration in Management of Pulmonary Problems," "Medical Economics," "Socialized Medicine," and "Mold Allergy."

Optional social activities will include tours of the Astrodome in Houston, the NASA Manned Space Flight Center at Seabrook, and the Sea Arama at Galveston.

Plans have also been made for a post-convention scientific meeting at the Grand Bahama Hotel and Country Club on the West End of the Bahama Islands.

AMA To Sponsor January Socio-Economics Congress

The effective organization and delivery of health services will be considered at the First National Congress on the Socio-Economics of Health Care, Jan. 22-23, 1967, at the Palmer House in Chicago.

The Congress is sponsored by the American Medical Association Council on Medical Service and the AMA Division of Socio-Economic Activities.

The Congress will bring together authorities from medicine, health care administration, social science, education, community planning, and other disciplines to report on new issues, developments, and techniques in the organization, delivery, and financing of health care services.

Through a series of presentations and discussion sessions, conference participants will explore current health status of the population, impact of medical and social changes on patterns of health care, the changing role of the hospital and its medical staff in the community, new methods in training and utilization of health manpower, and financing of health services.

Texas Radiologists To Hold Annual Meeting Jan. 27-28

The Texas Radiological Society will hold its annual meeting in Fort Worth at the Green Oaks Inn, Friday and Saturday, Jan. 27 and 28.

Seven speakers, four of them from out-of-state, have been announced as participants on the program.

Speakers and their topics will include Dr. Malcolm A. Bagshaw, Palo Alto, Calif., "Definitive External Radiation Therapy of Carcinoma of the Prostate," "Intra-arterial Chemotherapy Combined with External Therapy of Advanced Carcinoma of the Head and Neck," and "Intra-arterial and X-ray Therapy in the Treatment of the Lymphomas"; Dr. Charles M. Nice, New Orleans, "Barium Bronchography in Infants," Experimental and Clinical Observations in Small Bowel Roentgen Studies," and "Roentgen Findings in Collagen Diseases"; Dr. Melvin M. Figley, Seattle, Wash., "Interesting Aspects of Mediastinal Radiography," "Further Experience with Pulmonary Embolism," and "Hemodynamic Effects of Angiocardiography"; and Dr. John G. McAfee, Syracuse, N.Y., "A Comparison of Radioisotopes Scanning and other Neuroradiological Methods in the Diagnosis of Cerebral Lesions," "Diagnosis of Hepatic Lesions by Radioisotope Scanning," and "Future Medical Applications of Radioisotope Imaging."

Also scheduled to speak are Dr. Joseph H. Jackson, Jr., Dallas, "The Association of Congenital Cardiovascular and Renal Anomalies"; Dr. Carole Simmons, Houston, "The Osseous Manifestations of Leukemia in Children"; and Dr. Lacy H. Williams, Galveston, "The Use of Pulmonary Scans in the Diagnosis of Pulmonary Embolic Disease."

Cerebral Palsy Meet Planned

The Continental Hotel in Houston will be the site of the 1967 annual meeting of United Cerebral Palsy of Texas Jan. 7-8.

Business meetings will take place on Saturday, Jan. 7, including those of the Corporation, the Board of Directors, and the Medical-Professional Advisory Board. A workshop with the theme "The Cerebral Palsied and His Family" is planned for Sunday, Jan. 8.

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Imagination Can Bring

C. LINCOLN WILLISTON

Imaginative planning and communicating can go a long way toward drawing a larger number of the physicians in your county society into your overall program, says C. Lincoln Williston, Executive Secretary of the Texas Medical Association, who visits many county medical societies each year and plans many programs for Texas physicians. He offers some provoking and realistic thoughts on how county medical society officers can stimulate greater participation in their activities.

STIMULATING participation in medical society affairs is a most difficult challenge. There are no quick remedies, but there are sound principles and positive ideas which can be used effectively by medical society officers and executives.

Stimulating participation starts at the top with the society officers. It is their responsibility to create real enthusiasm for the society and its projects, first among committee members and then to broaden the circle among the members.

The keys to greater participation are imagination, planning, organization, implementation, involvement of the membership, and much effort by key leaders. A broad-based program which is important and meaningful to physicians will insure greater membership participation. Activities need to be both exciting and realistic. The value of a professional society can be measured only in terms of services rendered to its members and to the public. Physicians always will look for a direct return on their dues investment. Once the society has demonstrated its ability to be of service in time of need and to resolve problems efficiently and effectively, the members can be expected to reciprocate by participating. Most physicians will respond if asked to serve and if provided with a good reason.

Yet, stimulating participation is really not that easy. The problem of getting a large percentage of members "into the act" always will be a major challenge. Individual doctors do, however, respond to various techniques which can be employed to arouse interest.

The secret of success lies in the selection of the proper stimulant to incite interest and in the providing of carefully planned aids to assist the physician in achieving a specific objective. To select the proper stimulant, it is necessary to know as much as possible about the individual's professional practice, his community and social interests, his church background, his political party affiliations, and his hobbies. With that information on hand, the task then is to match the assignment with the doctor's interests and his abilities. Simply stated, the first cardinal principle is to carefully match the physician's interest, his knowledge of the job to be done, and the abilities and contacts he possesses that will aid in accomplishing the objective.

Orienting the New Physician

The process of stimulating interest in medical society affairs should be initiated even before the new physician reaches his community. One of the most significant developments which has helped to build medical organizations is the presentation of special programs for medical students. Many county and state societies are sponsoring medical student day programs for the senior classes at schools within their jurisdictions. These programs are designed to provide the student with an insight into the purposes of medical societies and their activities as well as to furnish him with practical information which will make his introduction to medical practice a little easier and smoother. This is a sound approach in building for the future.

County society officers have their greatest opportunity to stimulate participation by courting the new physician just as soon as he reaches his community. It has been said that a first impression is a lasting one, and so it is with medical society participation. New physicians should be greeted personally by the president and the secretary. There is merit, too, in following up with a letter of welcome, along with appropriate information and brochures about the society. Each new physician should be interviewed personally, if possible, and information should be secured about his interests and his needs. In this way, the new member will learn that the society does care about him. Some societies effectively employ a so-called "buddy" system. They ask physicians who sign application endorsements

Them Out

to look out for the new member and to bring him to meetings. This will insure the new member's appearance as well as that of his endorser.

No matter how small or large the medical society, a comprehensive orientation program for new members is a must. Many societies now present a series of indoctrination lectures on medical practice, with emphasis on ethics, etiquette, and local customs. In some societies, the physician must complete the series before his application can be acted upon. In other societies, the new member is obliged to fulfill the requirement within the first 12 to 24 months.

The orientation program has proved to be an effective technique in developing a more active society and a better informed membership. It is a contributing factor to increasing the utilization of society services. It will make those who attend more aware of their responsibilities to the profession and to the public. Finally, the orientation provision of required attendance can help establish habit patterns which will lead to greater participation.

Use of Communications

The effective use of communications is an extremely important part of stimulating participation. Once a program is developed, it obviously must be presented to the membership in an imaginative manner. The same principle applies to problems, whether they are intramural in nature, relate to hospitals, to academic affairs, to the community, or to government. Physicians will not become interested if they are not made aware of the problems, the projects, and the working mechanisms of the society. The use of the various media of communication, therefore, is basic to stimulating more interest. If problems and projects can be communicated adequately, they should result in greater understanding and in membership interest.

Maintaining good communications is a continuous, day-after-day, year-round assignment. The members must be kept informed regularly so that they feel that they are part of the team. This will require the use of every media of communication which is available —the journal or bulletin, the newsletter, announcements at meetings, hospital bulletin boards, and direct mail.

Most important, officers and executives should not overlook the most effective media of all—personal communications. It is amazing, yet true, that some physicians never seem to get the message. Journal articles, letters, bulletin notices, promotional mailings, and even telephone calls frequently go unnoticed. Personal contact, however, will hit the mark.

Active participation generally can be stimulated only by personal contact between society leaders and those whose interest they are seeking. In some societies, the project chairman will invite a selected member into his home for cocktails. He takes that opportunity to outline the project, its objectives, and the plan of action. Many societies feel that this is one of the finest techniques of all to command the physician's attention, to arouse his interest, to let him know that he is personally wanted, and to secure his participation.

Life Blood of the Society

The organizational key to stimulating participation is the development of a sound committee structure. Committees, in many ways, are the life blood of the society. The society's achievements and its prestige generally will rise or fall with these groups. By themselves, society officers can produce relatively little. However, when their energies and talents are swelled a hundred fold through committee appointments, the expanded organization can move mountains.

Committees obviously represent the most useful technique for achieving a broad base of membership involvement. At the same time, the committee appointment can make the individual physician satisfied with his membership, giving him an opportunity for service to the profession. His degree of satisfaction will be directly proportional to the effort which he puts into it.

Any president should continually seek out new "blood" for society committees and responsibilities. One of the best ways to gain the participation of the new member is to appoint him to a committee. At the same time, officers should encourage long-standing members who have not been active to become involved and to make a contribution to the society. Nothing

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will kill participation more completely than the membership feeling that the same old gang is running the show.

Some societies routinely put every new member on a committee. Others circulate a roster of committees with their functions to all members every year or two, and invite physicians to express their preference. This makes it easy for members to offer their services and it provides the society with a team of volunteers to do the work.

In metropolitan societies with large memberships, it obviously is not possible to appoint each physician to a committee. Nevertheless, imaginative officers and executives are finding outlets for all new members and for those who are willing to work. Some societies appoint many new members to the speakers bureau. It also is possible to appoint newly-elected members to special community projects, such as society immunization programs, manned exhibits at health fairs, and science fair judging in the local schools.

Perhaps one word of caution should be sounded on committee appointments. There is a tendency to overload an energetic, capable physician with many responsibilities. Rather than the use of the same old workhorse, it is far better to limit every physician to one appointment, and to spread the assignments among more members.

The principle of stimulating participation doesn't end with the mere appointment of committees. Nothing is more frustrating to a physician than to be named to a committee that never meets, or to one that has nothing to do. Similarly, half-hearted, cursory approaches to a problem will quickly destroy committee effort and physician participation. It is vitally important to outline specifically the committee's duties as well as its aims and goals. At the same time, the committee should be given considerable latitude to develop its own ideas. Officers also have a responsibility to provide the committee with administrative assistance and other resources which might be necessary.

Committees should be given an opportunity to report regularly on their progress, problems which they have encountered, and their accomplishments. In addition, the accomplishments of each physician and the committee should be recognized. Rather than to merely express appreciation, it is better to cite or to commend the physician for a job well done. Like every one else, physicians are pleased to be recognized by their professional society, and they likely will be willing volunteers for future assignments.

Improving Attendance

Attention should be given to the problem of attendance at regular meetings of medical societies. No one needs to be convinced of the need for improving participation in meetings. Only a relatively few societies can boast of attendance which enables them to perform at peak efficiency to fulfill their obligations to the profession.

There is a correlation between the numerical membership of the society and the percentage of attendance at meetings. As a general principle, the larger the membership of the society, the smaller is the percentage of attendance. Surveys have revealed that the larger societies also have a higher percentage of "deadwood" (members who never attend a meeting and who contribute very little besides payment of dues).

A survey of county societies has yielded a list of factors that contribute to good attendance. County societies in Texas which reported better-than-average attendance were surveyed. In addition, ideas and suggestions were provided by 25 medical executives throughout the nation who are regarded as highly knowledgeable in attendance stimulation.

Again there is no magic formula which will guarantee good attendance for every society. Nevertheless, those who have been successful have postulated that there are seven primary factors which merit attention:

1. The most important element is the presentation of outstanding programs and the conduct of timely, meaningful, yet uncomplicated, business meetings. Simply stated, physicians will attend if there is sufficient interest in the meeting; otherwise, they will stay home or find something better to do.

Here, one brief observation might be offered. Many who are knowledgeable agree that there is nothing like the "pocketbook" issues for stimulating meeting attendance. Even as remote as the society's immunization program in the community may seem, it could have a direct bearing upon the fees

charged by the physician in his practice. Most physicians are vitally interested in these matters and would like to have some voice as to how those issues are resolved.

Presidents should make certain that there is always one so-called headline item on the agenda for every business session. Rambling and lengthy reports should be eliminated, preferably by requiring those who are to present them to prepare them in writing. There should be emphasis upon encouraging, even soliciting, discussion from the floor. Officers should endeavor to stage meetings for those members who attend.

2. A second factor in good attendance is food and fellowship. As one county society secretary pointed out: "We have a cocktail hour and a steak at the best restaurant in town. Attendance automatically takes care of itself." A medical society executive put it even more bluntly when he wrote: "The fellowship hour is the blood and guts of stimulating attendance."

3. Concurrent or joint meetings of the medical society and its auxiliary are highly recommended. More and more societies are including the wives in as many meetings as is practical. There is no doubt that joint or concurrent meetings can be a factor in stimulating attendance by both the physician and his wife. This same general principle also applies to joint meetings with lawyers, the clergy, and other professional groups.

4. Advance notification of the meeting, with particular citation of important policy matters which are to be discussed, is important. An effective plan used by many so-

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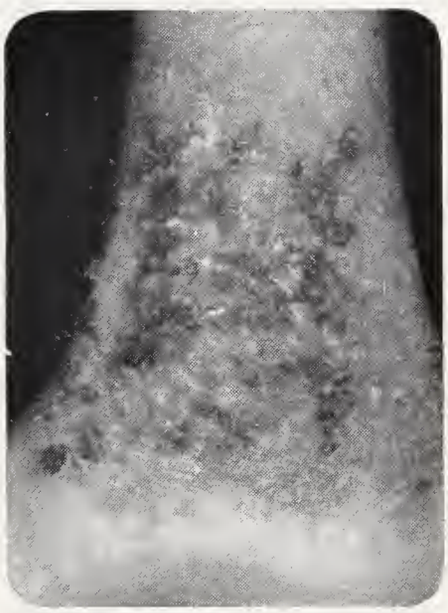
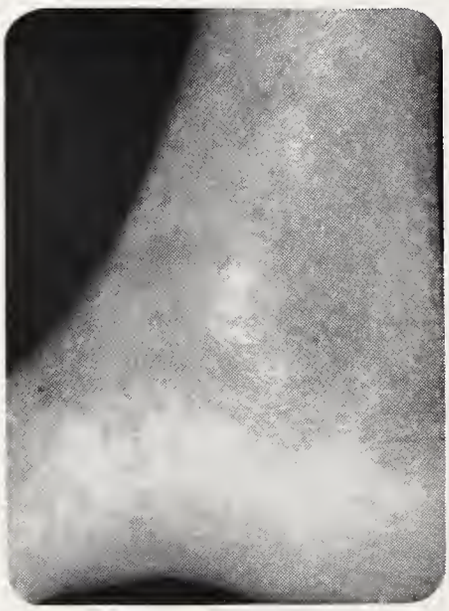


Before treatment



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cieties is to send an initial notice 10 to 14 days in advance of the meeting, with a follow-up reminder just prior to the program.

5. Meetings must be scheduled at a central location which will permit most members to attend without hardship.

6. Good relationships between physicians should be developed. When each doctor strives to promote better professional relations, the society is bound to profit. Many societies are blessed with a feeling of friendship and group interdependence. When the general attitude is wholesome, that in itself will stimulate attendance as well as participation in society affairs.

7. Personal initiative must be displayed by officers of a society. As in all phases of stimulating participation, responsibility rests with the society's key officers. A society cannot hope for good attendance without the imagination and the devoted attention of officers in arranging and conducting outstanding meetings.

Heart Disease, Cancer, and Stroke Planning Studies Now Under Way

[Editor's Note: At its Oct. 2 meeting the Texas Medical Association's Executive Board recommended that TMA representatives who serve on the state coordinating committee on the heart disease, cancer, and stroke program confer with Dr. Charles A. LeMaistre, vice chancellor for health affairs at the University of Texas, in order to ascertain the present status of the regional medical programs in Texas.

Responding to this directive, Dr. John F. Thomas of Austin, Dr. Byron P. York of Houston, and Dr. O. Brandon Hull of Lubbock conferred with Dr. LeMaistre in the chancellor's office at the University of Texas on Oct. 13. Accompanying them were Dr. Grover L. Bynum, Jr., of Austin, a member of the Committee on Cardiovascular Disease, and C. Lincoln Williston, TMA Executive Secretary. The subsequent report which follows has been submitted to members of the Executive Board, to the Council on Scientific Advancement, and to the Committees on Cancer and Cardiovascular Disease, as requested.]

Present Status of Program

In the summer, a planning grant submitted by the state coordinating committee was approved by the Division of Regional Medical Programs, National Institutes of Health. The grant totaled $1,271,013 for the first year. Dr. LeMaistre was designated as program coordinator and the Texas Medical Center, Inc., Houston, as fiscal agent. The planning grant is designed to assist in the planning and development of regional medical programs. Dr. LeMaistre reported that the first monies received from the grant are being utilized.

Separate planning studies are now being undertaken at Baylor University, Methodist Hospital, the University of Texas Medical Branch, Southwestern Medical School, South Texas Medical School, M. D. Anderson Hospital and Tumor Institute, the University of Texas Division of Continuing Education of the Graduate School of Biomedical Sciences at Houston, and the University of Texas Dental Branch.

The planning studies are expected to be continued by most centers for at least two years. Operational grants, designed to assist in the establishment and operation of the regional medical programs, will probably not be received before September, 1967, at the earliest, and probably not before the summer of 1968.

State Coordinating Committee

No meetings of the state coordinating committee have been held since June 14. At that time, representatives of TMA reported uniform agreement that the resources and cooperation of the medical and dental schools, medical and dental societies, hospitals and related institutions, voluntary health agencies, and state agencies were vitally needed to produce an effective regional program. TMA was promised an opportunity to take an active role and to carry out specific responsibilities in both planning and operational grants. TMA representatives also understood that all responsible organizations and institutions would have appropriate representation and would participate on the state coordinating committee.

No meetings of the state coordinating committee have been held during the past four months. While the state committee may continue to have some role in planning phases of the regional program in Texas, attention in the future seems more likely to be focused on a regional advisory group.

The state coordinating committee plans to meet soon. One of the primary purposes of the meeting will be to discuss the composition of the regional advisory group and to suggest names for appointment.

Regional Advisory Group

Public Law 89-239 specifies that an applicant for a planning grant must designate a regional advisory group. The law also specifies that the advisory group must approve an application for an operational grant. The advisory group must include practicing physicians, medical center officials, hospital administrators, representatives from appropriate medical societies, other health professions, voluntary health agencies, and representatives of other organizations, institutions, and agencies and members of the public familiar with the need for the services provided under the program. The group is intended to be broadly representative of the geographic areas and of the social groups who will be covered by the regional medical program.

The regional advisory group is expected to provide overall advice and guidance to the grantee in the planning and operation of the program. Texas Medical Association representatives believe that the advisory group should be actively involved in the review, guidance, and coordinated evaluation of the ongoing planning and operating functions; that it should be constituted to encourage cooperation among the institutions, organizations, health personnel, state and local health agencies, and with the state Hill-Burton agencies; that it should be concerned with a continuing review of the relevance of planning and operational activities in accordance with the objectives of the regional medical program;

and that it should review the effectiveness of these activities in attaining the objective of improved patient care.

TMA representatives previously had expressed the view that a regional medical program could attain maximum effectiveness only through the active support and cooperation of all of the state's medical and dental schools, hospitals and related institutions, medical and dental societies, voluntary health agencies, and state agencies which are interested in, and which are in position to contribute to the regional medical program. We continue to believe that all of these institutions and associations should be provided with an opportunity to take an active role and to be given specific responsibilities in both planning and operational phases of the program.

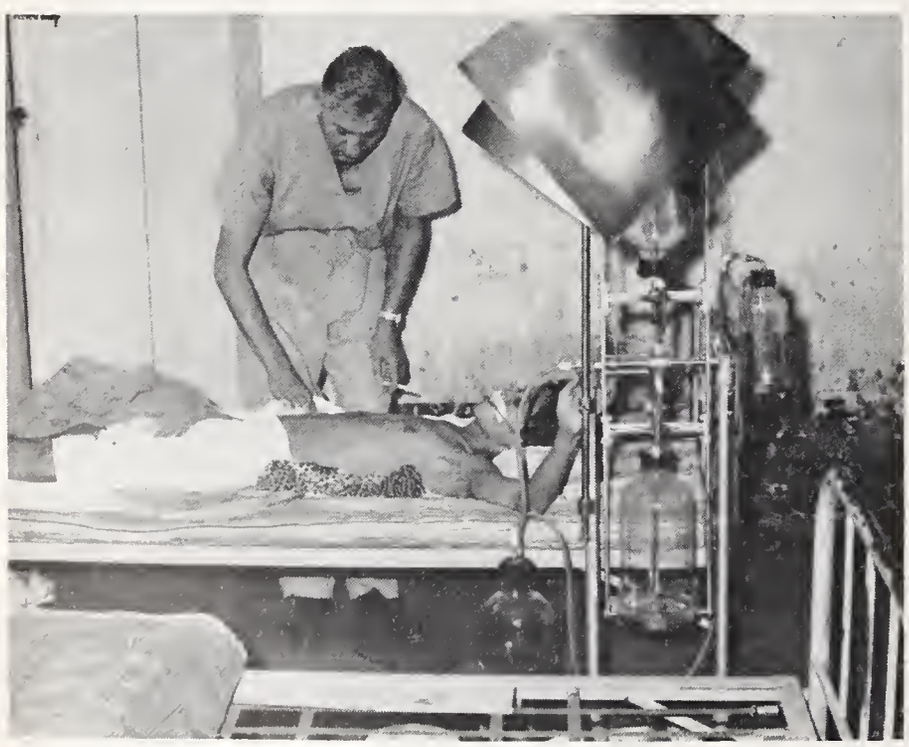
TMA representatives are now looking forward to the appointment of a regional advisory group and the selection of individuals to serve as members. The appointment of the regional advisory group, if made with broad representation, will satisfy the objectives which representatives of the TMA have sought for the past year in the development of the regional program on Heart Disease, Cancer, and Stroke in Texas.

Witch Doctors, Rare Diseases Add To Mekong Delta Medical Problems

Robert Franken, M.D.

[Editor's Note: The following article is an addition to a *Texas Medicine* continuing chronicle of Texas physicians' contribution to the war effort in Vietnam. Dr. Robert Franken, San Angelo surgeon, is one of a growing list of Texas doctors who have given two months volunteer medical service to the civilian population of South Vietnam. He is one of twelve physicians who were honored by Texas Medical Association on Oct. 1 for this volunteer service. He presents here some of his experiences and observations gathered during his tour of duty in Phu Vinh, Vinh Binh Province, South Vietnam, June 20-Aug. 20. The September issue of *Texas Medicine* contained similar accounts of five doctors who have served in similar capacities in South Vietnam. The editors of *Texas Medicine* would like to receive still other reports from physicians returning from Vietnam.]

MANY UNIQUE problems face the American physician in practice among the civilian population of Vietnam.



Dr. Robert Franken of San Angelo works with an injured South Vietnamese boy in the intensive care unit of Vinh Binh Provincial Hospital, Phu Vinh, where he served two months volunteer medical duty in the Mekong Delta region of Vietnam. The patient had been shot through the abdomen, and his colon was perforated. The wound was two days old when the boy was brought in. He did not survive.

Nearly all diseases were far advanced when seen by American physicians. The basic reason for this is the presence of witch doctors. The people have great faith in the Cambodian and Chinese witch doctors and always go to them first. Their methods of treatment leave definite indications. Vertical scarification marks on the throat show that the patient has been treated for an upper respiratory infection or goiter. Hundreds of small round or oval scars on any area of the body are signs that pain and swelling have been under therapy. These scars are formed by the injection of a drop of some sort of acid solution with a sharp splinter of bamboo. I have seen them around swollen joints, on the face, neck, chest, and back.

A great round red spot reveals that the patient has had a heated suction cup applied to an affected area. My own cook showed up one morning with one of these marks, two inches in diameter, in the center of his forehead. He thought I was a pretty good doctor, but he went first to the Cambodian witch doctor for his headache. He cured it too.

The witch doctors frequently slip into the hospital wards at night to treat their patients. One learns to accept this after a while; but it is good form to act very angry, call the patient "stupid" and "very bad," and threaten to send him home. They say they are very sorry and won't do it again; but, of course, they always do.

Certain diseases rarely seen in the United States, such as typhoid fever, cholera, plague, tetanus, and malaria, are common. Rare nutritional diseases especially kwashiorkor and noma, are frequently seen. Tuberculosis in a rather unusual form is endemic; and goiter, both toxic and colloid, is widespread. The high incidence of goiter in a fish-eating population is puzzling until one remembers that the iodine in the soil has been leached out through centuries of pouring rain.

Hookworm, roundworm, and amebic infestation is almost universal; and this applies also to American

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References: 1. Editorial: JAMA *191*:592 (Feb. 15) 1965. 2. Meilman, E., in Moyer, J.H.: Hypertension, Philadelphia, W.B. Saunders Company, 1959, p. 395.

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Side Effects: Hydroflumethiazide: Purpura plus or minus thrombocytopenia, hyperuricemia, leukopenia, hyperglycemia, glycosuria, malaise, weakness, dizziness, fatigue, paresthesias, muscle cramps, skin rash, epigastric distress, vomiting, diarrhea and constipation. *Reserpine:* Depression, peptic ulceration, diarrhea, Parkinsonism, nasal stuffiness, dryness of the mouth and, with overdosage, agitation, insomnia and nightmares. *Protoveratrine A:* Nausea, vomiting, cardiac arrhythmia, prostration, excessive hypotension and bradycardia. (Treat bradycardia with atropine and hypotension with vasopressors.)
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personnel who remain in the country for any length of time. Worms of varying sizes and descriptions were present in the intestinal tract of all but two of the 36 persons on whom I performed abdominal operations.

Cancer of the penis, rectum, breast, and stomach were all rarely seen and were usually inoperable when encountered.

The Vietnamese people almost never complain of pain, even with the most dreadful injuries. Even the children show little reaction to pain. Morphine and demerol were available to us but were rarely used.

Most of the wounds among civilians were caused by land mines, and the typical injury was a foot blown to pieces. The patients were brought in by their families and friends, often from miles away, on foot through the mud and water of rice paddies. There was always at least one tourniquet in place and sometimes two or even three at different levels on the leg. Sometimes these had been in place for as long as 48 hours; and then, of course, amputation was necessary.

Napalm burns were next in frequency, and many of the victims of these injuries were children and old women. Huge keloids and contractures were characteristic in those who survived this type of burn. There were a large number of arm and hand injuries in children caused by hand grenades placed in desks and waste baskets in schoolrooms by the Viet Cong.

Nearly all fractures were compound, comminuted, grossly contaminated, and accompanied by extensive soft-tissue destruction. High velocity bullets, artillery shells, mines, and the vicious little C.B.U.'s (cluster bomb units) produced tremendous internal damage to extremities and also, very frequently, to the lungs. Shock was almost universally present in our war casualties, and our operative mortality rate on these patients was staggering.

I have read in other accounts that Viet Cong patients could be kept in the same ward with South Vietnamese without danger. This was not our experience. I tried mixing the two only once, and the Viet Cong prisoner was promptly strangled during the night. After that I placed all V.C. wounded in the prison ward.

Houston Selected To Host International Cancer Congress

The Tenth International Cancer Congress, sponsored by the International Union Against Cancer, will be held in Houston in 1970. An attendance of 5,000 to 8,000 members is expected.

Dr. R. Lee Clark, director, surgeon-in-chief, and professor of surgery at the University of Texas M. D. Anderson Hospital and Tumor Institute, issued the invitation to the organization last spring.

The Ninth International Cancer Congress, recently held in Tokyo, voted on proposed sites in four countries for the next Congress. Houston, earlier selected as the choice of the United States, was chosen to host the international event.

The International Union Against Cancer is made up of scientists and physicians from 65 nations and maintains an office in Geneva.

Dr. Murray M. Copeland of Houston, chairman of the US committee of the union, was one of two Americans selected to the organization's council. The council, composed of delegates appointed by its national members, is the supreme governing body of the Union.

Dr. Copeland, associate director of education at M. D. Anderson, is a past president of the American Cancer Society.

Newly elected president of the congress, Dr. N. N. Blohkin of the Soviet Union, noting considerable competition among world cities for the site of the next congress, said, "I must congratulate our American colleagues for a victory."

In recent years, the congresses have met in Paris (1950), Sao Paulo (1954), London (1958), and Moscow (1962). The last US meeting was in St. Louis in 1947.

Eighteen staff members from The University of Texas M. D. Anderson Hospital and Tumor Institute presented 23 papers at the ninth congress.

Personals

Dr. James R. Schofield has been appointed dean of academic affairs at Baylor University College of Medicine at Houston. He has been associate dean at Baylor since 1959.

Dr. James E. Peavy of Austin was elected president of the American Association of Public Health Physicians at the association's meeting on Nov. 1 in San Francisco. Dr. Peavy is the Texas State Commissioner of Health.

Dr. John L. Matthews of San Antonio was presented the Beverly Myers Achievement award by the American Board of Opticianery at its meeting in October.

New officers for the Harris County Academy of General Practice for 1967 are **Dr. Philip A. Belleggie**, president; **Dr. Jack A. Haley**, vice-president; **Dr. Max M. Diamond**, president-elect; **Dr. Charles R. Younkin**, secretary; **Dr. George O. Zenner, Jr.**, treasurer; and **Dr. J. D. Gerdes**, member of the board of directors.

Welfare Commissioner John H. Winters Dies

John H. Winters, who had been commissioner of public welfare in Texas for 23 years, died Tuesday, Nov. 29, in an Austin hospital. He had been ill for several months.

Mr. Winters had been a great help to Texas Medical Association on numerous occasions. C. Lincoln Williston, executive secretary of TMA, said of him: "John Winters was a dedicated public servant, and he will be missed by those who worked with him. He was a friend of Texas Medicine, and he deserves great credit for developing the Kerr-Mills program in this state which was one of the finest such programs in the nation."

Burton G. Hackney of Brownfield, chairman of the State Board of Public Welfare for eight years was named to succeed Mr. Winters as commissioner. Announcement of his appointment was made Monday, Dec. 5, by the other two members of the board, Will Bond of Hillsboro and W. Kendall Baker of Houston.

Assistant Commissioner Herbert C. Wilson was named acting commissioner until Mr. Hackney is able to get his personal affairs in order. Mr. Wilson had assumed heavy responsibilities in the department during Mr. Winters' illness.

Medicare Extended Benefits Program Evaluated by Medical Leaders Involved

On Jan. 1, 1967, the extended care benefits of the national Medicare program will go into effect. These benefits will provide relatively short-term skilled care for the recently hospitalized elderly patient who continues to need full-time skilled nursing care. This nursing care must, however, be provided by an extended care facility which has met the standards for participation in the Medicare program.

Medicare will pay the full cost of covered services furnished by a participating extended care facility for further treatment of a condition for the first 20 days and all but $5 a day for 80 additional days in each spell of illness, or period of time during which benefits are payable for inpatient hospital services and extended care services.

Services covered by the extended care Medicare program include: (1) room and board in a semi-private room, or a private room if medically necessary; (2) physical, occupational, or speech therapy; (3) nursing services, except private duty nursing; (4) drugs, biologicals, supplies, appliances, and equipment usually furnished to patients by the extended care facility; (5) medical social services; (6) other services necessary to the health of the patient ordinarily furnished by the extended care facility; and (7) services of interns and residents-in-training of a teaching hospital with a transfer agreement with the extended care facility.

Services not covered include physician's services not covered by Medicare hospital insurance; (2) services in a facility operated primarily for the treatment of mental illness or tuberculosis, (3) private duty nursing; (4) personal comfort or convenience items; (5) private room, unless medically necessary; and (6) any services not covered as an inpatient hospital service.

The extended care facility will complete and send a claims form to the organization chosen by the government to pay these claims. Any payment due is made to the extended care facility, not to the patient.

To receive extended care services covered under Medicare, a patient must show his health insurance card when admitted to the facility. He will also be asked to sign a special Medicare claims form which the facility will use to get its payment from the insurance carrier.

Key figures in the implementation of the extended care benefits portion of Medicare in Texas are Drs. Robbie C. Jordan, chief of Medical Care Administrative Services Section, the Texas State Department of Health; Mr. Herbert Wilson, Assistant Commissioner of Public Welfare, and Dr. Joseph T. Phillips, medical director, Texas State Department of Public Welfare; and Mr. John M. Crawford, Jr., executive director, Texas Nursing Home Association. Drs. Jordan and Phillips and Mr. Crawford present pertinent comments with regard to the implementation of this medical care program.

HEALTH DEPARTMENT EVALUATING FACILITIES

Dr. Robbie C. Jordan,
Chief of Medical Care Administrative Services Section
Texas State Department of Health

Medicare is the abbreviated term for medical care services as defined in Title XVIII of Public Law 89-97. This law provides for the designation of a state agency to assist the Department of Health, Education, and Welfare in qualifying providers of health services. Its duty is to appraise the potential provider and furnish consultation for those who wish to participate.

The Texas State Department of Health was designated by Gov. John Connally as the state agency, and the Commissioner of Health, Dr. James E. Peavy, created the Medical Care Administration Services Section. This new section's responsibility in the Medicare program is to work with providers of service, do "on-the-site" surveys, and recommend to the Secretary of Health, Education, and Welfare the certification of those facilities which meet the standards prescribed by this law.

Hospitals, laboratories, and home health agencies wishing to participate, began providing service on July 1, 1966. The extended care facilities will begin their participation on Jan. 1, 1967.

The extended care facility is a new category of health care conceived by this same law which created Medicare. It means an extension of care following hospitalization. It is a bridge between the patient's hospital care and his return to his place of residence. To qualify for extended care coverage, a patient must have been in a hospital for at least three days. Admission must be within 14 days after dismissal from a hospital.

The "Blue Book," *Conditions of Participation for Extended Care Facilities*, was developed by the Secretary of Health, Education,

and Welfare. To comply with the conditions of participation, the primary requisites to be met are statutory. Generally, these call for each patient to be under the care of a physician; for the facility to provide continuous skilled nursing care; for each institution to have written patient care policies, a transfer agreement with a hospital, and a utilization review plan; and for clinical records to be maintained on each patient. Also, each facility must be licensed as a nursing home or a hospital by the State of Texas, and in no way present a hazard to the patient's health and safety.

Most nursing homes that are licensed with registered professional nurses as supervisors can qualify under Medicare, because they meet these requirements with little difficulty. There have been 301 applications for participation. We have completed 217 surveys to date. There are 82 surveys not yet completed. Two surveys have not yet been assigned. Most facilities need only to follow a transfer agreement, patient care policies, etc. to complete their work up. Approximately 150 nursing homes will be certified in Texas providing about 12,500 beds.

Some hospitals are designating a distinct part of their physical plant as an extended care facility. A distinct part of a hospital, or a nursing home, can be defined as a wing, a floor, a ward, or any identifiable section of the facility.

The State Department of Health had a deadline of Nov. 15 to complete the survey, process it, and send the recommendation to the Secretary of Health, Education, and Welfare. A perpetual service of the section is to furnish consultation on any phase of the program. In addition to the initial survey of a facility, re-surveys must be conducted at scheduled intervals.

Although change is usually accepted slowly and cautiously, it is hoped that the extended care facility will be accepted by the medical profession and be able to take its place in the Medicare program.

NURSING HOME PARTICIPANTS ESTIMATED

John M. Crawford, Jr.,
Executive Director
Texas Nursing Home Association

As Jan. 1 approaches, I am frequently asked, "How many nursing homes will qualify as extended care facilities?" My estimation is approximately 170.

With the cooperation of physicians, with more understanding of the program on the part of nursing home administrators, and, hopefully, with a future increase of the number of nursing and paramedical personnel, the number of participating homes should increase.

Basically, there are three reasons why more nursing homes are not applying for Medicare participation, or are not being approved:

1. The tremendous shortage of registered professional and licensed vocational nurses is prohibiting many homes from meeting the necessary staffing requirement. Many homes cannot secure the services of a registered nurse as the supervising nurse due to the lack of supply.

Texas averages 166.8 registered nurses per 100,000 persons. This places our state 46th in the nation. The national average is 282.2 registered nurses per 100,000 persons, with some states, such as New Hampshire, having as many as 470.

The availability of acceptable licensed vocation nurses (LVN's) is complicated by the fact that the LVN's must be graduates of state approved schools. Since the Texas LVN licensing law went into effect, 35,940 licenses have been issued. Of these, 13,282 are by waiver and thus do not meet these requirements. Many of the waivered LVN's are employed in Texas nursing homes and are doing an admirable job in nursing our aged citizens.

Relatedly, the shortages of ADA dietitians, social workers, and speech, physical, and occupational therapists present another staffing hurdle that is hard to overcome.

2. The purposes and functions of the modern nursing home do not readily lend themselves to Medicare extended care services. The nursing home of 1966 is established and staffed for long-term chronic care, not for short-term acute convalescence as provided for by Medicare.

The man on the street (and many newspaper reporters) do not yet realize that an extended care facility and a nursing home are not necessarily the same thing. Many excellent nursing homes are not applying for participation because of the philosophies and practices of the institution. These facilities have rehabilitative, social, and dietary programs set up for the 12 to 18 month resident and are staffed accordingly. Many extended care beneficiaries may only need to remain in a Medicare facility for 7-21 days.

Thus, the dedication of the nursing home to the maintenance arts does not necessarily correspond to the curative arts needed and demanded by Medicare.

3. Basically, the nursing home profession provides a proprietary function, as does the practicing physician. Nationally, 87 percent of all nursing homes are proprietary, as compared to approximately 8 percent of hospitals. In Texas, less than 10 percent of the licensed facilities and less than 9,000 of the 43,000 beds are nonprofit.

The reimbursement formula for provider services, although recently improved, is not generally attractive to proprietary organizations. Furthermore, if the nonprofit institution must be free standing (which is becoming more prevelant) and support its own operations, then the reimbursement formula is not adequate for these institutions either.

As I stated in an editorial in *Texas Medicine* in March, 1966, "The role of the physician will and must continue to grow as nursing homes evolve into their rightful place as an integral part of the total health team. The physician is the key figure in determining utilization of health services."

Those homes that participate in Medicare now or in the future, and those homes that provide services under Title XIX of the Social Security Act will lean heavily on the physician for many services, particularly in the development of patient care policies and in utilization review. A 40-member Medical Care Advisory Committee has been appointed to help develop a Title

XIX program for Texas—16 of these members represent Texas Medical Association and Texas Nursing Home Association.

With your help, nursing homes—with or without Medicare—will be recognized as an important part of the total health team. It is the objective of the nursing home profession to make available to the elderly the best nursing care available.

WELFARE PROGRAM TO BE IN OPERATION

Dr. Joseph T. Phillips,
Medical Director
Texas State Department of Public Welfare

The Vendor Payment Program (popularly known as Kerr-Mills program) helps provide nursing home care for the recipients of Old Age Assistance when they need qualified skilled nursing care. This program has been in effect since 1962 and will be in effect in Texas until Title XIX is implemented by the Texas Legislature.

The current Vendor Payment Program makes payments of a stipulated amount directly to the vendor and not to the patient. Nursing homes must meet certain standards to qualify for participation in this program. There must be a registered nurse or a licensed vocational nurse in charge of the nursing services provided in the nursing home. Also, a certain nurse-to-patient ratio must be maintained according to the classification of the home such as: intensive care, moderate care, or minimum care.

The nursing homes choose the class under which they wish to qualify and are then evaluated according to the Texas State Department of Public Welfare standards for each class. The homes ask to participate in the program, and a free choice of doctor, pharmacy, and other needs are maintained in every area.

The Vendor Payment Program is a state-federal program and is administered by the Texas State Department of Public Welfare. Any discontinuance or amendment of this program must be decided by the Legislature. Title XIX must also be acted upon by the state legislature in order to be put into effect.

Title XVIII (Medicare) on the other hand is a completely federal program and needs no state legislative action. The program is therefore administered by the federal government. The extended care facility (nursing home care) benefits portion of Medicare (Title XVIII) will go into effect on Jan. 1, 1967.

However, the Vendor Payment Program will also remain in effect until the Texas Legislature takes action regarding Title XIX. A person over 65 years of age may *not* qualify for benefits under both programs until the Legislature has acted.

Title XIX provides for medical care for the medically indigent only.

Postgraduate Courses

Bedside Diagnosis–Part IV, Philadelphia, Jan. 11-13.—This postgraduate course is one of seven courses being offered by the Department of Medicine of Hahnemann Medical College and Hospital, Philadelphia, during a seven-month period.

For further information, write Dr. Bernard L. Segal, Department of Medicine, Hahnemann Medical College, 230 N. Broad St., Philadelphia 19102.

Current Coneepts in Blood Disease, Miami Beach, Fla., Jan. 23-27. The American College of Physicians (ACP) will present a five-day course through the University of Miami School of Medicine at the Fontainebleau Hotel, Miami Beach, Fla.

Pathogenesis, clinical features, and management of various hematologic diseases will be presented. Congenital and acquired disorders of red blood cells, leukocytes, platelets, and blood coagulation will be reviewed. Selected advances in hematologic technique will be demonstrated.

Tuition fee for ACP members is $60; for nonmembers, $100. Only ACP members are accepted in courses immediately upon receipt of application. Nonmembers are placed on waiting lists in the order in which applications are received, and notified three weeks after the release of the course outline whether there is sufficient room for them in the course.

Requests for information and registration forms should be addressed to Dr. Edward C. Rosenow, Executive Director, American College of Physicians, 4200 Pine Street, Philadelphia 19104.

Bioehemical Lesions in Internal Medieine, St. Louis, Feb. 2-10.—Another American College of Physicians course will be presented through Washington University School of Medicine. This course will emphasize the importance of biochemical considerations as they relate to the etiology, pathogenesis, clinical phenomena, and therapy of various disorders encountered in internal medicine. The application of these considerations to genetics, metabolism, hematology, allergy, arthritis, gastroenterology, and renal, cardiovascular, pulmonary, infectious, and malignant disease will be illustrated by a series of lectures. Various biochemical tools will be demonstrated in laboratory sessions.

Information concerning tuition fees and course registration is the same as in *Current Coneepts in Blood Disease* preceding.

Mierobiology, Atlanta, Ga.—A series of laboratory refresher short courses is being presented during an 11-month period by the Laboratory Branch of the Communicable Disease Center of the US Public Health Service.

Courses with registration closing dates through January are Laboratory Methods in Systemic Mycoses, Feb. 13-24 (closing date Jan. 3, 1967); Laboratory Methods in General Medical Bacteriology, and Laboratory Methods in Arbovirology, both Feb. 27-March 10 (both with closing dates on Jan. 16).

Information and application forms are available from the Training Office, Laboratory Branch, Communicable Disease Center, Atlanta, Ga. 30333.

against the usual gram-negative urinary pathogens

Why use five...where **one** will do?

In a recent 217-patient hospital study,[1] urinary tract infections were treated with a variety of widely prescribed antimicrobial agents including: a sulfonamide (40 patients), chloramphenicol (20 patients), nitrofurantoin (33 patients), nalidixic acid (30 patients), tetracycline (27 patients), colistimethate sodium (22 patients)...and 2 combinations of 5 agents each (45 patients). The 2 combinations were selected to afford maximal theoretical antibacterial coverage against the usual urinary pathogens. They were (1) tetracycline, chloramphenicol, nitrofurantoin, rlstocetin and polymyxin B; and (2) tetracycline, chloramphenicol, erythromycin, nitrofurantoin and colistimethate sodium.

This clinical study shows that the two combinations of antibiotics were not superior to some of their single components. The authors point out that antibiotic antagonism often negates theoretical advantages of multiple therapy. Coly-Mycin Injectable (colistimethate sodium) was one of the single components that was shown to be equal to the combinations and eradicated bacteriuria in two-thirds of the patients.

Theoretical choice of multiple antibacterial therapy has been shown to be no more effective than one well-chosen agent which also offers least patient exposure to possible side reactions, toxicities, allergic manifestations and higher drug costs.

1. McCabe, W. R., and Jackson, G. G.: New England J. Med. 272:1037, 1965.

in gram-negative urinary tract infections often the single well-chosen agent

Coly-Mycin® Injectable
(colistimethate sodium)

Indications: Especially indicated for the treatment of severe acute and resistant chronic urinary tract infections due to sensitive strains of gram-negative organisms. Also indicated in respiratory tract, surgical, wound and burn infections and in septicemia due to sensitive organisms. Particularly indicated when any of these infections are caused by sensitive strains of *Pseudomonas aeruginosa.*

Adverse Reactions: Occasional reactions such as circumoral paresthesias, tingling of the extremities, pruritus, vertigo or dizziness may occur. Reduction of dosage may alleviate symptoms. Therapy need not be discontinued, but such patients should be observed with extra care.

Warning: Patients should be cautioned not to drive vehicles or use hazardous machinery while on therapy.

Precautions: In cases of impaired or suspected renal impairment, use with greater caution and reduce dosage in proportion to extent of impairment. Transient elevations of BUN have been reported. As a routine precaution, appropriate blood studies should, therefore, be made during prolonged therapy.

As with all polypeptides, the possibility of muscular weakness, including apnea, due to inadvertent overdosage or normal dosage in the presence of impaired renal function, should not be overlooked. In cases of apnea, medication should be promptly discontinued and assisted respiration given until serum levels fall and normal breathing is restored.

Other antibiotics, such as kanamycin, streptomycin, dihydrostreptomycin, polymyxin, and neomycin, may also have varying neurotoxic or nephrotoxic potential. They should be used with great caution concomitantly with Coly-Mycin Injectable (colistimethate sodium).

For deep intramuscular injection only.

Dosage: By the I.M. route only, in 2 to 4 divided doses ranging from 1.5 to 5 mg./Kg./day (0.7 mg. to 2.3 mg./lb./day). Average adult dose is 2.5 mg./Kg./day (1.1 mg./lb./day). In the presence of bacteremia, septicemia, or other serious infection, greater than average doses may be required; however, maximum daily doses should not exceed 5 mg./Kg. (2.3 mg./lb.) where renal function is normal.

Not recommended against Proteus.

Colistin is also available (as colistin sulfate) in: Coly-Mycin® Pediatric for Oral Suspension (not for systemic use), and Coly-Mycin® Otic with Neomycin and Hydrocortisone.

Full information is available on request.

CI-GP-69-R2

WARNER-CHILCOTT Morris Plains, New Jersey

Speakers, Television, Curbstone Topics Announced for Centennial Annual Session

Plans for the 1967 Texas Medical Association Centennial Annual Session are reaching final stages. Three general meeting speakers have been selected. Four closed-circuit color television programs have been scheduled. Ten Curbstone Consultations have been set up.

Among numerous general arrangements will be a special concert by the Dallas Symphony Orchestra, alumni and fraternity functions, sports tournaments, and a memorial service honoring deceased members of TMA and the Woman's Auxiliary.

In addition, a total of 32 out-of-state speakers have accepted invitations to participate in the meeting. Also, several special events to celebrate the 100th Annual Session of TMA have been planned. Among these special features will be a commemorative painting by artist Robert Thom and a special issue of *Texas Medicine* relating the histories of various facets of medicine in the state.

General Meeting Speakers

Three outstanding medical leaders will give general meeting presentations during the 1967 Annual Session. They are Dr. Sydney S. Gellis, chairman, Department of Pediatrics, Tufts University School of Medicine, Boston; Dr. Robert I. Wise, head, Department of Medicine, Jefferson Medical College, Philadelphia; and Dr. Robert J. Coffey, chairman, Department of Surgery, Georgetown University School of Medicine, Washington, D.C.

Dr. Gellis will discuss "Controversial Aspects of Immunization Procedures." Dr. Wise will speak on "Guide Lines in the Selection of Antibiotics in the Treatment of Bacterial Infections." Both Dr. Gellis and Dr. Wise will speak on Friday morning, May 5.

Dr. Coffey will present, "The Diagnosis and Treatment of Chronic Pancreatitis" on Saturday morning, May 6.

Television Presentations

Four color closed-circuit television programs will also be highlights of the Annual Session. These television presentations will be made possible through the sponsorship of Smith Kline & French Laboratories of Philadelphia.

Cases will be presented from Parkland Memorial Hospital and will be beamed to the theater of the Dallas Memorial Auditorium.

Dr. Vernie A. Stembridge of Dallas, television chairman for the Annual Session, has selected topics of interest to a large number of physicians. Four Dallas physicians have agreed to serve as moderators. These doctors are presently working with panelists and compiling data for the programs. Scientific content of the programs is being supervised by Dr. Stembridge, the moderators, and members of the Council on Annual Session.

The schedule of television presentations is as follows:

1. "Detection and Management of Psychosomatic Disorders," Thursday, May 4, during the Texas Academy of General Practice Seminar. The program presents three cases. Dr. Jerry M. Lewis will moderate a two-hour discussion from 2-4 p.m.

2. "Shoulder-Neck Problems," Friday, May 5, 11:15 a.m.-12:15 p.m., during the general meeting. Dr. Charles F. Gregory will moderate.

3. "Intra-Uterine Transfusions and the Care of the Neo-nate," Friday afternoon, May 5, before a joint session of the sections on general practice, obstetrics and gynecology, and pediatrics. Dr. Reuben H. Adams will serve as moderator.

4. "Common Dermatological Problems," Saturday morning, May 6, during the general meeting session. Dr. J. B. Shelmire, Jr., will moderate the discussion session.

Curbstone Consultations

A popular Annual Session feature in previous years has been the Curbstone Consultations. These will once again be presented during the 1967 meeting. Ten, informal, unrehearsed, round-table discussions will be conducted on topics of general medical interest. The Curbstone Consultations are conducted as person-to-person conversational discussions of cases and problems.

Moderators for the sessions have been selected for each table to stimulate comments from physicians visiting the tables. Physicians will be encouraged to bring case histories and to exchange opinions. Beer, coffee, and soft drinks will be served.

Topics and moderators selected are

1. "Stroke," Dr. David D. Daly, Dallas, chairman, Department of Neurology, Southwestern Medical School;

2. "Skin Tumors—Clinical Diagnosis and Treatment," Dr. J. Fred Mullins, Galveston, chairman, Department of Dermatology, the University of Texas Medical Branch;

3. "Cancer of the Thyroid," Dr. Edgar C. White, Houston, head, Department of Surgery, M. D. Anderson Hospital and Tumor Institute;

4. "Management of Thromboembolic Disease," Dr. Fred B. Aurin, Fort Worth, surgeon;

5. "Sensitivity Chest Disease,"

Dr. John P. McGovern, Houston, allergist;

6. "Respiratory Insufficiency," Dr. John W. Middleton, Galveston, professor of internal medicine, the University of Texas Medical Branch;

7. "Adolescent Amenorrhea," Dr. Harwin B. Jamison, Dallas, obstetrician and gynecologist;

8. "Management of Chronic Infantile Diarrhea," Dr. David M. Mumford, Houston, clinical instructor of pediatrics, Baylor University College of Medicine;

9. "Management and Treatment of the Post-Coronary Patient," Dr. David McMahon, Jr., San Antonio, internist; and

10. "Evaluation of the Patient Complaining of Sinus Disease," Dr. Claude D. Winborn, Dallas, otolaryngologist.

General Arrangements

A special concert will be presented for the Centennial Annual Session registrants by members of the Dallas Symphony Orchestra. The concert will take place at 8:15 p.m. Saturday, May 6.

The special concert is one of the results of work by the General Arrangements Committee, a group of Dallas physicians who have been arranging certain special features for the Annual Session. Dr. Don G. Harrel is chairman of the General Arrangements Committee. Vice-chairman is Dr. L. S. Thompson, Jr.; and executive officer is Millard J. Heath, executive officer, Dallas County Medical Society.

Other committee activities include arrangement of fraternity and alumni parties, golf and tennis tournaments, a memorial service for deceased members of the Association and the Woman's Auxiliary, and a breakfast for women physicians.

Alumni functions are planned for Saturday evening, May 7, preceding the symphony concert. Alumni groups meeting and local chairmen are the University of Texas Medical Branch, Dr. A. Ben C. Dowdey; Tulane University School of Medicine, Dr. Theodore P. Votteler; Southwestern Medical School, Dr. Norman Kaplan; and Baylor University College of Medicine, Dr. Herbert W. Beutel, Jr.

Fraternity parties and class reunions also will be scheduled from 6 to 8 p.m. Saturday.

Dr. Reuben Adams serves as general chairman for alumni and fraternity events.

Golf and tennis tournaments are being planned to take place on Thursday, May 4. If interest justifies it, a skeet tournament will also be held. Drs. Robert Romero, Halcuit Moore, and Albert O. Loiselle are chairmen of sports activities.

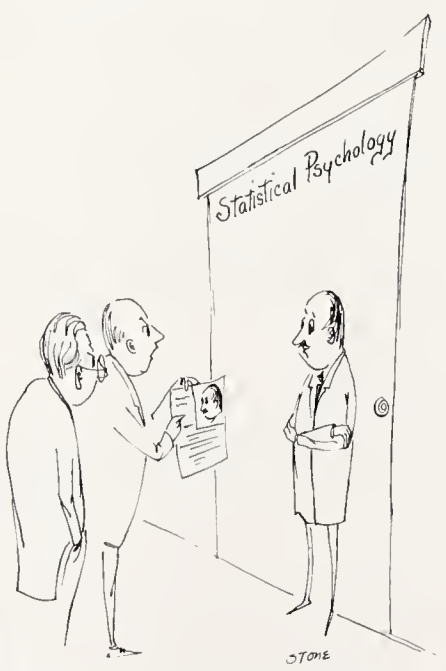
Women physicians who are members of TMA will convene for a breakfast buffet at 7 a.m. Saturday, May 6, at the Adolphus Hotel. Dr. Alice Smith is chairman for this event.

The memorial service for deceased members of TMA and the Woman's Auxiliary will take place at 11:15-11:45 a.m. Sunday, May 7, in the Embassy Ballroom of the Statler Hilton Hotel. Dr. C. E. Van Cleave, Jr., is local chairman of arrangements for the service.

Other local general arrangements chairmen are Dr. David Vanderpool, entertainment; Drs. Walter Patton and Guy K. Driggs, first aid; Dr. Robert K. Portman, Housing; Dr. Elgin W. Ware, Jr., information; Dr. P. E. Luecke, Jr., Public Lectures; Dr. Frederick J. Bonte, publicity; Dr. John Stack, scientific exhibits; and Dr. Alice Smith, technical exhibits.

Housing Accommodations

Scientific meetings and exhibits will be concentrated at the Dallas Memorial Auditorium, and some scientific meetings will be held in the Adolphus Hotel.



"We don't like this report on 'What Is Normal.'"

The Statler Hilton Hotel has been designated as headquarters for business affairs and for sessions of the House of Delegates. The Adolphus Hotel will be the headquarters for scientific activities. The headquarters for the Woman's Auxiliary will be the Sheraton-Dallas Hotel. Rooms at the Statler Hilton and the Adolphus have been reserved for TMA officers, members of boards, councils, and committees, delegates and alternate delegates, and officers of specialty and related groups. Rooms are also available for physicians and their wives at the Sheraton-Dallas Hotel. Other hotels with room blocks reserved to accommodate TMA members are the Baker Hotel of Dallas, the Marriott Motor Hotel, and the Inn of the Six Flags, Arlington.

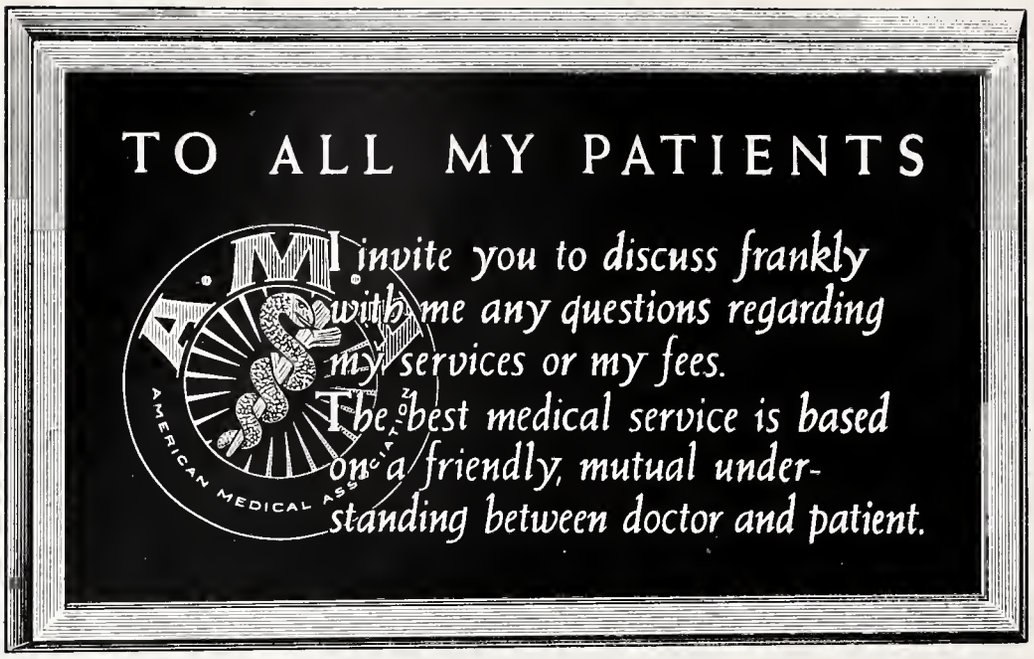
Physicians should write to the facility of their choice at an early date in order to secure reservations.

Dr. R. M. Tenery Elected To AMA Council Post

Dr. Robert Mayo Tenery, Waxahachie, was elected to the American Medical Association's Council on Constitution and By-laws at the 1966 Clinical Convention at Las Vegas on Nov. 28-30.

Dr. Tenery was elected by the House of Delegates to fill a vacancy on the AMA Council. His term will continue through 1969. He defeated Dr. Joseph M. Ribar, Fairbanks, Alaska, in a special election.

Dr. Tenery has taken an active role in the AMA and the Texas Medical Association for many years. He has served as a member of the AMA Investigating Jury since 1964. In 1965, he was a special consultant to the staff of the Department of Medical Ethics. Dr. Tenery is a Past President of the Texas Medical Association. He presently serves as a member of the Association's Council on Constitution and By-laws, and as chairman of the Special Committee on Liaison with the Texas Hospital Association.


TO ALL MY PATIENTS
I invite you to discuss frankly with me any questions regarding my services or my fees. The best medical service is based on a friendly, mutual understanding between doctor and patient.
AMERICAN MEDICAL ASSOCIATION

Doctor, Do Your Patients Understand Your Fees?

Patients hesitate to ask about fees. But they **want** to know. They naturally worry about what their medical costs will be. And if they don't feel free to talk about it, serious problems—misunderstanding, resentment and even deep hostility—can arise, threatening the all-important rapport between you and your patients.

Open discussion before treatment prevents such embarrassing problems. It paves the way for mutual understanding and good will.

An AMA Fee Plaque, prominently displayed in your waiting room, serves as an open invitation for your patients to discuss medical costs and financial problems with you. It shows that you care. Just seeing the message can give many financially worried patients peace of mind.

The AMA Fee Plaque works hard to prevent doctor-patient misunderstandings. And it works well.

Suitable for wall or desk display, the plaque measures 6" x 10½". The lettering is white on a dark brown background and the frame is durable beige plastic.

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AMA Clinical Meeting Delegates Vote To Seek Medicare Changes

Issues related to the Medicare program—certification of patients, utilization review, and reimbursement of charges for professional services on the basis of a valid statement rather than a receipted bill—commanded the attention of the Texas Delegation at sessions of the American Medical Association House of Delegates during the 1966 Clinical Convention at Las Vegas on Nov. 28-30.

Attention also was devoted to the new Title XIX program, which provides federal financial assistance to the states for health care for those who qualify for categorical aid as well as for the medically needy.

Texas delegates shared the general concern of the medical profession about the Medicare requirement to certify and to recertify the medical necessity for the admission or continuing hospitalization of a patient, pointing out that it places an unnecessary burden upon the physician. Testimony presented emphasized that the present requirements for certification and recertification do not contribute to the quality of medical care. It was urged that an admission record or an adequate progress note be accepted to satisfy the legal requirements.

After evaluating testimony, the House of Delegates determined that the AMA should endeavor to bring about the repeal of those provisions of Public Law 89-97 (the Social Security Amendments of 1965) which require physician certification of medical necessity for hospitalization. The AMA will be available to counsel with fiscal intermediaries and the American Hospital Association in the development of amendments to the law which will be professionally acceptable and administratively workable.

In another action the AMA House of Delegates urged state medical societies to seek the passage of state legislation which would provide a physician who serves on a utilization review committee with immunity from litigation arising from acts of the committee. A number of state legislatures already have granted this protection to physicians.

Under Public Law 89-97, utilization review committees are not empowered to discharge patients from hospitals. The discharge continues to be the decision of the physician responsible for the patient's care, with the utilization review committee determining only the continued fiscal responsibility of the federal government. Although it was pointed out that exposure to vexatious litigation may be slight, the AMA urged state societies to seek legislation to provide the committee member with desired immunity.

The AMA House of Delegates also devoted attention to a desired change in the Medicare law to permit the reimbursement of charges for professional services to be rendered on the basis of a valid statement by the physician to the patient, rather than a receipted bill. The present language of the law stipulates that payment for physicians' charges will be made by the fiscal intermediary on the basis of a receipted bill, or on the basis of the assignment of benefits by the patient to the physician.

The AMA has been urged to seek remedial action to amend Public Law 89-97, establishing that an agreement for payment between the patient and physician constitutes valid evidence of service rendered.

The AMA House of Delegates also devoted considerable attention to the new Title XIX program for medical assistance programs to the blind, the permanently and totally disabled, dependent families with children, the aged, and the medically needy. Many states already have implemented Title XIX programs, while the Texas Legislature will consider it when it convenes in January.

At the Las Vegas meeting, it was pointed out that free choice of physician is not guaranteed in Title XIX, Public Law 89-97, even though this is a provision insuring it for beneficiaries under Title XVIII (Medicare).

Physicians stated that the AMA should support the historical principle of the patient's right to a free choice of physician. They also pointed out that a patient should have a freedom of choice of a medical facility and should not be required to receive care at a facility not of his choosing. After hearing testimony on this subject, the House of Delegates requested the AMA to make a major effort to amend Title XIX of Public Law 89-97 to include these principles in the law.

The AMA House of Delegates also took a firm position in support of an amendment to the Social Security Act permitting the direct billing of patients under Title XIX by providers of services.

The Department of Health, Education, and Welfare has interpreted the present legislation governing Title XIX programs to be on a "vendor payment" basis. Federal reimbursement presently is not available for payments made by patients to physicians. This is in contrast to Title XVIII (Medicare) in which the physician has the option of billing the patient directly or taking an assignment of benefits from the patient.

At hearings on this subject, it was pointed out that a stated aim of Title XIX legislation was to eliminate the "second class" status of the needy. By not permitting reimbursement for direct payment, Title XIX is, in effect, denying beneficiaries the provision of care on the same basis which is available to those who do not need help.

Culminating these discussions, the AMA House of Delegates went on record in support of an amendment to Public Law 89-97, Title XIX, which would permit payments without assignments for medical care rendered to the patient by the provider of services. If the amendment is enacted by the 90th Congress, physicians would have the option of billing the patient directly or accepting an assignment.

Wait, Annabelle. Mommy hasn't told the doctor yet that you *need* a shot.

Maybe she does, maybe she doesn't. But if her problem is nasal congestion due to cold or allergy, consider the medication that combines convenience and effectiveness—Novahistine Melet.

Melet is especially suitable in the treatment of children. It's a pleasant tasting, chewable tablet that leaves no aftertaste or gritty, undissolved particles in the mouth. It provides decongestant action almost as quickly as a liquid, but assures the accurate dosage of a tablet.

Because Melets are so convenient to carry and so easy to take at any time, adults, too, will appreciate this modern dosage form.

Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution adults who operate machinery or motor vehicles that drowsiness may occur. Each Melet contains phenylephrine hydrochloride 10 mg. and chlorpheniramine maleate 2 mg.

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Advances in Drug Research

Antidepressant Agents Cause Varied Results In Treatment of Mentally Depressed Patients

Jaime N. Delgado, Ph.D.
Frank P. Cosgrove, Ph.D.
Eugene I. Isaacson, Ph.D.

[Editor's Note: This is the first part of an article to be completed in the January issue of *Texas Medicine*. The authors are on the faculty of the College of Pharmacy at the University of Texas, Austin.]

PERHAPS more human suffering has resulted from mental depression than from any other single disease.[1] In 1963 the late President John F. Kennedy in a special message to Congress on mental illness and mental retardation, said "Mental illness and mental retardation are among our most critical health problems. They occur more frequently, affect more people, require more prolonged treatment, cause more suffering by the families of the afflicted, waste more of our human resources, and constitute more financial drain upon both the public treasury and the personal finances of the individual families than any other single condition."[2]

There are two principal types of psychotherapeutic agents: the tranquilizers and the antidepressants. The major types of antidepressant agents will be considered here.

Although antidepressants have been introduced into medical therapy rather recently, they are now used in the treatment of more than four million patients a year in this country. There is little doubt that many millions more with mild depression never seek treatment because they fail to recognize the need for help. The inability to diagnose certain types of depression and to effectively get all persons with obvious cases to undergo therapy allow the occurrence of 20,000 known suicides annually in the United States.[1]

Duboin also states that two million persons in this country are estimated to have made at least one suicidal attempt.[3]

The five most recognized symptoms of depression are (1) feeling of sadness, (2) fatigue, (3) loss of interest in social environment, (4) self-neglect, and (5) insomnia.[1]

The important antidepressants may be structurally classified as follows: (1) β-phenethylamine congeners, (2) α-alkyltryptamines, (3) hydrazine derivatives, (4) cyclopropylamines, (5) propargylamines, (6) iminodibenzyl derivatives, and (7) derivatives of 10, 11-dihydrodibenzo-cycloheptene.

Beta-Phenethylamine Congeners

The modern chemotherapeutic attack on mental depression began in the early 1950's when the medicinal chemist began making molecular modifications of the amphetamine molecule in order to overcome some of the shortcomings of this drug. Pipradol (Meratran) and methylphenidate (Ritalin) are representative examples of antidepressants which were the result of this research. These drugs may be classified as mild antidepressants which produce their effect principally through psychomotor stimulation.[4]

Alpha-Alkyltryptamines

When the phenyl group of amphetamine was replaced by an indole ring, a class of moderately potent antidepressant agents classified as the α-alkyltryptamines resulted. One of the most important changes brought about by this replacement was the development of a significant antidepressant effect with the virtual elimination of the potent psychomotor stimulant effects seen with amphetamine. Etryptamine and MP-809 are protypes of this group. The former was found to be effective in the treatment of neurotic depression; however, it was removed from the market when it was suspected of causing blood dyscrasias.[4] The latter was also observed, using multiblind technique, to be a potent antidepressant agent in neurotic depressive states.[5]

Hydrazine Derivatives

Much interest in preparing and testing the aralkylhydrazines and hydrazides was brought about in 1957 when it was found that iproniazid (Marsilid), once used in the treatment of tuberculosis, elevated the mood and restored vitality in patients. The central stimulation or antidepressant activity of iproniazid was found to take place by an indirect mechanism. This drug inhibits the enzyme monoamine oxidase (MAO) which is known to catalyze the degradation of certain chemical mediators having the α-arylethylamine structure. Perhaps the most important neurohormones which MAO affects are epinephrine, norepinephrine, and serotonin. Some investigators believe that by decreasing the rate of destruction of these amines, the MAO inhibitors prolong the biographical half-life of these neurohormones, the end result being an increase in the

central sympathetic tone within the hypothalamus and an aroused behavioral pattern.[6]

When it was reported that iproniazid induced hepatocellular jaundice, many groups began research programs designed to produce MAO inhibitors which might have less toxic characteristics. It was soon observed that the following structural modifications of iproniazid had much to do with the physiological activity of the parent compound[6]:

1. Any substitution on the terminal amine group reduces the antidepressant and MAO inhibitory activity.
2. Any substitution other than the methyl on the alpha carbon of the side chain reduces the antidepressant potency.
3. Lengthening or decreasing the two carbon side chains or any substitution on the aromatic ring reduces the antidepressant activity and the brain MAO specificity.

Investigations based on the rationale of the previous structure activity relationships led to the development of pheniprazine (Catron), isocarboxazid (Marplan), mebanazine (Actomol), phenelzine dihydrogen SO_4 (Nardil), and nialamide (Niamid). Although all of these hydrazine-derived MAO inhibitors have been shown to be potent antidepressants, pheniprazine (Catron), which was believed to be the most potent and specific antidepressant, exhibited a severe, irreversible toxic reaction of the retina which necessitated its withdrawal from the market.[6] Marjerrison clinically found that this drug and similar compounds which have an MAO inhibitory property produced sporadic hallucinations in psychiatric patients. It was found to produce an overall effect on the quality of visual imagery (an increase of structural detail; vividness and clarity) but not on the amount.[7]

Cyclopropylamines and Propargylamines

Further structural alterations of the sympathomimetic amines resulted in two nonhydrazine-derived MAO inhibitors, namely tranylcypromine (Parnate) and pargyline (Eutonyl).[4]

It has been stated that there are three properties of the MAO inhibitors (particularly the hydrazine derivatives) which can restrict their range of therapeutic application: (1) they are long acting, (2) they have cumulative effects, and (3) they potentiate many other drugs.[2] The phenylcyclopropylamine compound, tranylcypromine (Parnate), is reported to be more acceptable than the phenylalkylhydrazines since it has a more rapid onset of action, a lack of cumulative effect due to a shorter duration of action, a lesser intensity of action restricting its use mainly to the nonpsychotic depression, and a greater safety with respect to hepatotoxicity and visual disturbances.[4]

Pargyline (Eutonyl) a propargylamine, containing an acetylenic moiety in the side chain of the phenylalkylamine structure, is a highly potent MAO inhibitor. Chemically pargyline is N-benzyl-N-methyl-2-propynylamine, and it is reported to be readily soluble but unstable in aqueous solution.[8] Sutynick and associates found it was an effective agent in the treatment of hypertension; but its action is primarily of an orthostatic type.[9] Because it is also effective in lowering blood pressure in moderate and severe essential hypertension, this drug is used extensively as an antihypertensive agent. Pargyline's MAO inhibiting properties are similar to those of the phenylalkylhydrazines; however, from the viewpoint of hepatotoxicity it is reported to be a much safer drug.[4]

Pargyline compares favorably with guanethidine SO_4 and α-methyldopa as an antihypertensive agent. Although the blood pressure lowering is achieved primarily with these drugs via orthostatic hypotension, some decrease in high blood pressure is seen in the supine position. The potent autonomic side effects generally experienced with the ganglionic blocking type of antihypertensive agents are less frequent with Eutonyl.[4] Sutynick and associates pointed out that great care must be used with pargyline in establishing the proper maintenance dose, for neither the hypotensive effect nor the amount of drug required to produce this effect could be predicted.[9]

Discussion of other antidepressant agents, some observed adverse reactions to antidepressants, and conclusions reached, along with a complete bibliography, will appear in the January, 1967, issue of Texas Medicine.

Texas Holds Sixth US Rank In Major Medical Coverage

Texas ranks first in the South and sixth in the US in number of persons with insurance company major medical expense protection. The Health Insurance Institute lists 2,435,000 persons in Texas as possessing such coverage.

The Midwest is the leading section in the country with 15,253,000 persons covered. The Northeast is second with 14,647,000, the South third with 12,284,000, and the West fourth with 9,762,000.

The five states ahead of Texas in medical coverage are California, 6,669,000; New York, 6,444,000; Illinois, 4,190,000; Pennsylvania, 2,789,000; and Ohio, 2,737,000.

The only Southern states besides Texas to have more than 1,000,000 persons covered are Florida, 1,383,000, and Georgia, 1,131,000.

At the end of 1965 the total number of persons with major medical coverage in the US was 51,946,000. The Institute said benefits of over $1 billion were paid under these policies.

Dallas Birth Defects Center In Second Year of Operation

The Department of Pediatrics, the University of Texas Southwestern Medical School, Dallas, with financial assistance from Texas Chapters of the National Foundation—March of Dimes has a Birth Defects Center which is in its second year of operation.

The center functions in three major areas: (1) diagnosis of the condition of the patient, (2) research into problems presented, and (3) training of students and resident staff in awareness and recognition of birth defects.

Any child under 15 years of age who has a birth defect may be referred to the center for evaluation.

Patients able to meet medical expenses may be accepted on a sliding fee basis. Many children seen in the Center qualify for financial aid from state and local agencies. Plans for this can be made on an individual need basis.

The Center is an integral part of the University of Texas Medical School System, thus all other services are available for consultation.

Southwest Clinic Association

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*Diplomate of The American board of his specialty

**Required training for Certification by the American Board of his specialty has been completed.

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Commission Rules Blood a 'Product', Non-Profit Blood Bank a 'Business'

[Editor's Note: The Federal Trade Commission ruled on Oct. 26 that a nonprofit Kansas City blood bank was guilty of restraining trade. The decision, in effect, places nonprofit blood banks under the jurisdiction of the FTC, subjects the process of distributing blood to the regulation accorded commercial products, and seems to give preference to the enterprises of commercial blood banking firms. Medical authorities have expressed fear that the results of this FTC decision will be blood that is more expensive for recipients and at the same time is of poorer quality than that presently distributed by nonprofit blood banks.]

The Federal Trade Commission released an opinion on Oct. 26 upholding an FTC hearing examiner's conclusion that the nonprofit Community Blood Bank of the Kansas City Area, Inc., was guilty of conspiring with the Kansas City Hospital Association, its members, and 14 hospital pathologists in restraining the exchange, sale, and distribution of human blood.

This long-awaited decision by the FTC is the result of charges filed with that agency by two commercial blood banks in the Kansas City Area claiming that the nonprofit blood bank, along with certain pathologists and the hospital association, was involved in a conspiracy in restraint of trade whereby the commercial blood banks were unable to sell their blood. These allegations have become known as the "Kansas City Case" and were discussed briefly in this column in the August issue of *Texas Medicine*.

Following the filing of the charges of conspiracy, evidence was taken at a hearing during the summer of 1963 by an FTC hearing examiner. In an initial decision, Hearing Examiner Edward Bennett ruled that a conspiracy did exist between the various "defendants" and that they were hampering and restraining trade. Implicit in the examiner's findings of restraint of trade was the determination that human blood was a commodity or a product and that the nonprofit, charitable blood bank was under the jurisdiction of the FTC.

The hearing examiner's decision was appealed to the full Federal Trade Commission and was argued before the FTC on Dec. 10, 1964. After holding its decision in abeyance for almost two years, the FTC, in a three to two decision, has adopted the hearing examiner's conclusion.

In writing the majority opinion, FTC Commissioner Paul Rand Dixon has held that a blood bank incorporated under state not-for-profit statutes and exempted from federal income taxation is nevertheless under the jurdiction of the FTC by virtue of the Federal Trade Commission Act of 1914. This act defines a corporation as "any company or association incorporated or unincorporated, which is organized to carry on business for its own profit or that of its members." The commissioner holds that only those organizations or corporations operated for "religious, charitable, educational, and other specified purposes" and which provide that no part of their income inures to the benefit of any private person would be excluded from the jurisdiction of the FTC. It seems that even though no person receives any portion of the profit, if any, which might be evolved from a charitable blood bank's operation, the mere fact that the blood bank might take in more than it spends in operating is enough for the FTC to find the blood bank to be a "profit-making corporation" notwithstanding the fact that such a "profit" is reinvested in improved facilities and service.

Thus, by perhaps straining the Congressional intent as expressed in the Federal Trade Commission Act, the FTC determined that it has jurisdiction over the operations of a nonprofit blood bank. Having made this determination, one is not surprised to then read that the Commission does not feel the process of acquiring and processing blood to constitute the practice of medicine and, therefore, the operation of the blood bank not to be a legitimate attempt by the medical profession to regulate medical matters.

In his opinion Commissioner Dixon does recognize that blood banks may be generally supervised by physicians and that medical skills may be utilized at various points along the line. However, the Commissioner finds that the procedures of drawing, processing, storing, and delivering blood, although technical, are routinely performed by persons who are not physicians. Thus, the acts involved in blood banking do not, in the opinion of the Commission majority, constitute the practice of medicine. Consequently, the Commission finds in its opinion that the acts of acquiring, processing, and supplying whole blood to hospitals when performed by licensed blood banks are parts of a "business" rather than parts of the practice of medicine.

Perhaps the most significant and far-reaching finding contained in the FTC opinion is that whole human blood is considered to be a "drug" and therefore a "product" for the purposes of Section 5 of the Federal Trade Commission Act. Based on these deductions, we find the Commission determining that whole blood is a product and not a medical service, a product presumably subject to all of the expressed and implied warranties of wholesomeness and fitness for human use which every other product is subject to. No sane person would argue that the highest degree of skill and care should not be exercised in every facet of collecting and processing blood, yet even the most extreme care and the most thorough tests are unable to detect such infections as serum hepatitis

virus in blood. Court imposed liability on blood banks for failure to test for and detect every possible impurity may, in the end, serve to force withdrawal of the nonprofit blood banks leaving only those truly in the *business* of selling blood. At the same time, the cost of whole human blood would be driven up several hundred percent over present patient costs.

Also, in finding that a conspiracy existed between the blood bank and the hospitals, Commissioner Dixon's opinion admits that there was no direct evidence of a conspiracy, but holds that there was sufficient evidence for this conspiracy to be inferred. Motive for such a conspiracy was "proved" in part by the fact that the parties believed that commercial blood-banking was morally wrong and that commercial blood banks do not supply blood of equal quality with nonprofit banks. Additional "proof" of conspiracy was that various meetings took place between the hospital association, the blood bank, and the society of pathologists and "affirmative steps were taken by the parties" to prevent the commercial blood banks from establishing donor clubs.

In their dissenting opinions, Commissioners Philip Elman and John R. Reilly held that the nonprofit blood bank was not within the Commission's jurisdiction. Further, they held that the record does not show the existence of a conspiracy. In addition, Commissioner Reilly did not feel that the proceeding was warranted by the public interest.

Earlier this year, attorneys for the Community Blood Bank of the Kansas City Area, Inc., indicated that if the Federal Trade Commission opinion went against the Community Blood Bank, then the blood bank, hospital association, and hospital pathologists would appeal that decision to the US Circuit Court. Thus, if this is still their intent, it is possible that the United States Supreme Court will settle whether or not the transfusion of blood is a sale of a commodity or the providing of a service.

Whatever the final disposition of the case, it is regrettable that an organization founded by physicians and hospitals to fill a most necessary and humane function should be placed in the position of defending itself against conspiracy charges brought by corporations organized to sell blood as a business and answerable only to stockholders or owners demanding a profit.

—PHILIP R. OVERTON, LL.B., and SAM V. STONE, JR., LL.B., Austin.

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Book Notes

Conflict in Society. Anthony De Reuck and Julie Knight, Ciba Foundation Symposium, 467 pp., illus., Boston, Little, Brown and Co., 1966, $13.50.

TWENTY-THREE world leaders in the fields of sociology, anthropology, social psychology, psychiatry, ethnology, systems analysis, political science, history, and international law share their ideas on the genesis and resolution of conflict in man.

By discussing the etiology of group conflict at a number of levels, it is hoped that it may eventually be possible to elucidate some of the sociological processes leading to war. The essence of the human problems is somehow to learn to combine material progress with survival.

The authors' approach to this topic of world concern is multi-disciplinary, and is, therefore, well rounded and well informed.

It All Started With Hippocrates. Richard Armour, 136 pp., illus., New York, McGraw-Hill Book Co., 1966, $3.95.

ONLY A WELL INFORMED medical historian could turn facts into a book of nonsense. Medical greats and firsts are well documented with footnotes and illustrations which are good for a laugh on every page. ". . . To remove a bone from the throat, for example, the physician would recite a long incantation, completely without meaning. . . ." The footnote to this reference reminds the reader that this long recitation sounded more like the commencement address at the medical school than a remedy.

Any one interested in medical history or spending a few hours with a delightful author should not miss reading *It All Started With Hipproerates.*

Handbook of Clinical Laboratory Data. Henry C. Damm, and John W. King, eds., 469 pp., Cleveland, Ohio, The Chemical Rubber Co., 1965. $12.50.

THIS BOOK is a collection of medical laboratory data grouped in eight main areas: histology, clinical laboratory values, endocrinology, chemistry, toxicology, hematology, blood banking, and microbiology.

It is a non-exhaustive compilation of miscellaneous and current information on laboratory and other values and norms. It is worthy in that it puts together in a single volume data which are available in the scattered literature of research and medical journals.

A large number of tables pervades the volume. Some of these are well planned and easy to use. Others are more confusing, less well organized, and need some proofreading.

There is an occasional mixing of subjects within specific areas, but the indexing is good and facilitates finding the misplaced matter. A seeming departure from the objectives of the book is noted when outlines of therapy are introduced in the chapter on acid-base balance.

Generally, however, the publication should be very useful to medical students and physicians. The reference listing at the end of almost each item would be more useful to the specialist and researcher.

—A. Q. DaSilva, M.D., Austin.

Practical Manual for Clinical Laboratory Procedures. Henry C. Damm, and John W. King, eds., Cleveland, Ohio, The Chemical Rubber Co., 1965. $12.50.

THIS loose-leaf collection gives the medical laboratory procedures

relating to blood banking, hematology, chemistry, histology, microbiology and urinalysis.

The procedures are listed alphabetically within each of the main sections. The description of each procedure includes a brief discussion of the general principle on which it is based, the normal values, the type of specimen necessary for the examination, a list of the equipment and reagents required, and the method involved. Notes pertaining to quality control, sources of error, calculations, results, and their significance, as well as references, are also included whenever needed.

The compilation is comprehensive and designed primarily for the medical technologist and the medical technology student.

The publishers intend to release supplements to the *Manual*, which will be available without charge to those who acquire the book. These will be printed in loose-leaf form to be inserted in the proper section within the plastic-covered folder of the *Manual*. According to the publishers, the "inserts" will keep the owner currently informed of newly developed techniques.

—A. Q. DaSilva, M.D., Austin.

Neurological Surgery of Trauma. Colonel John Boyd Coates, Jr., ed., 604 pp., illus., Washington, D.C., Office of the Surgeon General, Department of the Army, 1965. $6.25.

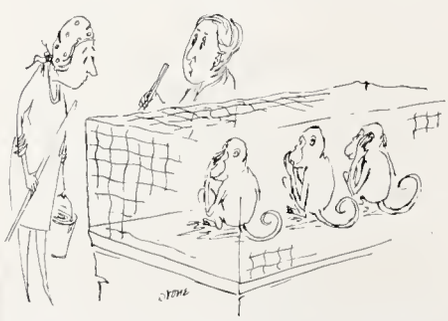
THE AUTHORS have written of experiences gained in World War II and Korea in the management of injuries to the nervous system. *Neurological Surgery of Trauma* has many virtues, including a reasonable price. Unfortunately, it has some shortcomings.

The use of many authors has made the volume uneven in its text. Obviously, some of the chapters are very carefully and thoroughly researched and documented. These have a most authoritative and precise ring. The chapters on organization, on spinal cord injuries, and on rehabilitation are noteworthy in this regard. Clearly the authors of these chapters have considerable personal interest and experience in the area covered by the chapters.

Other chapters are of historic or philosophic interest only. The contrast between the two chapters on closed head injury and differential diagnosis of surgical lesions and those immediately following, point this out. There are no data included in the chapter on differential diagnosis of surgical lesions: Not even the incidence of the various types of surgical lesions encountered are indicated. Furthermore, no mention is made of arteriography as a diagnostic tool. Only the sketchiest and most general statements cover the management of closed head injuries, and no information is given about prognosis or what factors may influence it.

The chapters on penetrating wounds of the brain are very well done and carefully documented. The line drawings accompanying them are clear and informative. Precise methods of operative and supportive care are given. The section on spinal cord injury is the most thoroughly documented section of the book, covering in detail the wounds of the cord virtually segment by segment. Clear and lucid indications for surgery are given. The technique of proper care is illustrated, and the problems the paraplegic faces are carefully and systematically enumerated.

The section on peripheral nerves is also well done. Of particular interest is the graphic illustration of the function of each individual peripheral nerve. Both motor and sensory testing is shown. This is of great value in examination of a patient. Since most physicians have little knowledge of peripheral neuroanatomy, this single feature is worth the price of the book. Unfortunately, data regarding recovery after peripheral nerve lesions are rather sketchily presented.



"Some sort of communications experiment, I think."

The book is printed on excellent paper with clear type. No major editorial or typographic errors are present. The index is excellent, but the bibliography leaves much to be desired, consisting of articles written 20 years ago or before; a rather notable exception is the section on rehabilitation of the paraplegic where a lengthy and complete bibliography is appended.

In general, this book has much to commend it. It is not a definitive treatise on all aspects of injuries to the nervous system. However, it can be read with profit by practitioners of the specialty. It should be a part of the library of any physician who is called on to treat patients with injuries to their nervous system. It will furnish him with sound guidelines of patient care.

—Kemp Clark, M.D., Dallas.

Of Molecules and Men. Francis Crick, 99 pp., Seattle, Wash., University of Washington Press, 1966, $3.95.

THIS WORK is based on the fifth series of John Danz Lectures delivered at the University of Washington. The series has been noted for the broad scope of its consideration of man. In this volume, the reader is brought to the actual borderline between the living and the nonliving.

Dr. Crick has the faculty of finding relevant material in unlikely places. This characteristic combined with an unusual clarity of expression, makes his book a lively delight for the lay reader. His descriptions of the structure of nucleic acid of which the genetic material

is formed, of the way in which genetic information is stored in the molecule, and of how "copying" takes place, have startling beauty and simplicity.

New Films In TMA Library

Surgery of Varicose Veins and Ulceration. Color, 22 minutes, 1964, Dr. John Mullany, director.

THE CAUSES of varicose veins and associated ulceration are briefly explained. Diagnostic measures are illustrated. Corrective surgical techniques are demonstrated, and examples of postoperative results are shown.

A Renal Biopsy. Color, 6 minutes, 1963, Dr. Nancy Thornton, director.

THIS FILM shows the technique of renal biopsy in a pediatric patient. Preoperative sedation, positioning, and actual techniques are described or shown. The same technique is applicable for adults.

Recent Deaths

DR. V. R. MOORE

Dalhart general practitioner, Dr. Victor Rhodes Moore, 67, died on Oct. 19, 1966. He had been in a Dalhart hospital since Oct. 10 following a series of heart attacks. He had practiced in Dalhart since 1932.

DR. MOORE

The son of William C. and Mima Rhodes Moore, he was born on Feb. 3, 1889, in Junction City, Kan. He attended schools in Parsons, Kan., and received a bachelor of arts degree from the University of Kansas in 1923. In 1928 he received his medical degree from the University of Louisville School of Medicine. He served both an internship and a residency at Louisville General Hospital in Louisville, Ky.

In 1918 he served as a private in the US Army.

Dr. Moore had been a member of Central Methodist Church for many years and sang in the church choir. He had also served on the official board of the church. He was a member of the Lions Club, Knife and Fork Club, Dalhart Chamber of Commerce, and Phi Beta Pi medical fraternity.

A past president and secretary of Dallam-Hartley-Sherman-Moore Counties Medical Society, he belonged to the Texas Medical Association, Panhandle District Medical Society, American Medical Association, Aerospace Medical Association, and American Academy of General Practice. He was medical advisor for the Dalhart draft board for 24 years, and had been medical examiner for the Dallam and Hartley Counties health office. He had been aviation examiner for the Federal Aviation Agency for 15 years.

Survivors include his wife, the former Sophia M. Utermann of Alma, Kan., whom he married July 8, 1925, in New Haven, Conn.; one daughter, Mrs. Marilyn Drendel, and four grandchildren, of Reno, Nev.

DR. B. H. VAUGHAN

Port Arthur physician, Dr. Ben H. Vaughan, 76, died Sept. 23, 1966, there. He had practiced in Port Arthur since 1918.

Born Sept. 14, 1890, in Paintsville, Ky., he was the son of C. N. and Elizabeth Melvin Vaughan. He attended schools in Russell, Ky., and Valparaiso University in Valparaiso, Ind. In 1917 he received his medical degree from the Chicago College of Medicine and Surgery. He did postgraduate work at a New York Hospital.

DR. VAUGHAN

A diplomate of the American Board of Otolaryngology, he was a fellow of the American College of Surgeons, and a member of the Jefferson County Medical Society, Texas Medical Association, American Academy of Ophthalmology and Otolaryngology, Texas Society of Ophthalmology and Otolaryngology, and Texas Ophthalmological Association. He belonged to the Methodist church. He was a Rotarian and a Mason. He had served the Jefferson County Medical Society as president in 1930. He also was a past president of the Rotary Club.

His hobby was fishing.

On Sept. 28, 1916, Dr. Vaughan married Pearl Walter of Blaine, Ky., in Ashland, Ky.

Survivors include his wife of Port Arthur; a daughter, Mrs. Mildred Hidalgo of Falls Church, Va.; two brothers, Oscar Vaughan of Port Arthur, and Charles V. Vaughan of Middletown, Mo.; and three grandchildren.

DR. G. F. MIDDLEBROOK

Dr. George F. Middlebrook, 82, died Aug. 14, at his home in Nacogdoches. He had practiced medicine for more than half a century.

Born on Nov. 20, 1883, in Willisville, Ark., he was the son of the Rev. and Mrs. T. A. Middlebrook. He grew up in Hope, Ark., and was graduated from high school there. He received his medical degree in 1907 from Washington University School of Medicine in St. Louis, Mo. He did postgraduate work at Tulane University School of Medicine in New Orleans.

Dr. Middlebrook first came to Texas in 1911 to serve as physician for a lumber company in Caro. On Aug. 10, 1916, he married the former Miss Annie Lou Carter of Caro. They celebrated their golden

DR. MIDDLEBROOK

wedding anniversary the week before Dr. Middlebrook died.

After leaving Caro, he practiced in Louisiana and East Texas. In 1931, Dr. Middlebrook moved to Nacogdoches. He went into semi-retirement in 1947. For several years he continued to practice medicine in addition to running a dairy and producing beef cattle.

Elected to inactive membership in the Texas Medical Association in 1959, he belonged to the Nacogdoches County Medical Society and the American Medical Association. The son of a minister, Dr. Middlebrook was a lifetime member of the Baptist church and had served in official positions in several congregations. In recent years he had been a member of the Lilly Grove Baptist Church near his farm home.

He is survived by his wife; one son, George F. Middlebrook, Jr., of Nacogdoches; four daughters, Mrs. J. Norris Tucker and Mrs. Stephen Sakach, both of Houston, and Mrs. N. G. Bright and Mrs. Edwin W. Gaston, Jr., both of Nacogdoches; a brother, Otto Middlebrook of Athens, La.; four sisters, Mrs. Ruth Carrigan of Hope, Ark., Mrs. J. H. Briley of Alexandria, La., Mrs. C. C. Nash of Dallas, and Mrs. Ruby Berry of El Dorado, Ark.; and 12 grandchildren.

DR. W. R. LEON

Dr. William Robert Leon, a Fort Worth internist, died Sept. 17, 1966, after suffering a heart attack at a high school football game. He was 48.

The son of Harry Cecil and Harriett Shine Leon, he was born May 28, 1918, in Kansas City, Mo. He married Violet M. O'Brien of St. Louis, Mo., on April 27, 1943, in St. Louis.

Dr. Leon attended schools in Tulsa, Okla., Villanova University in Villanova, Pa., and St. Louis University, in St. Louis, Mo. He received his medical degree from St. Louis University School of Medicine in 1942. He interned at St. Louis City Hospital in St. Louis. He served a residency at the same hospital and at St. John's Hospital, also in St. Louis. He practiced a short time in St. Louis after serving in the US Army. In 1944, he began practice in Fort Worth.

Dr. Leon had been president of the Physicians' Building, Inc., medical director of Mid-States Security Life Insurance Company, and director of Mid-States Escrow Agency. He served as chairman of the Fort Worth Foundation for Medical Education and Research.

He was a member of the Tarrant County Medical Society, Texas Medical Association, American Medical Association, and the Fort Worth Club of Internists. He belonged to Phi Beta Pi medical fraternity and Rivercrest Country Club. He was a member of Holy Family Catholic Church.

Dr. Leon's hobbies were model railroading, tropical fish, photography, and coins.

Survivors include his wife; three sons, William Robert Leon, Jr., David Terrance Leon, and Richard Cecil Leon; two daughters, Merri Diane Leon and Aleta Marie Leon; and his parents, Mr. and Mrs. Harry Cecil Leon, all of Fort Worth.

DR. LEON

DR. C. D. HENRY

Dr. Colvern D. Henry, 66, died Aug. 20, 1966, in San Antonio. Cause of death was cardiac arrest. He was born Sept. 2, 1899, in Lamasco, Tex., the son of Squire Morgan and Sarah Alice Davenport Henry.

Dr. Henry attended schools in Groom, Goodnight, and Southland, Tex., and Texas Christian University. He was graduated from the University of Oklahoma in 1924 and received his medical degree from the University of Oklahoma School of Medicine in 1926. He interned at Parkland Memorial Hospital in Dallas and practiced in Lockney and Plainview before moving to San Antonio in 1932. In 1942 he graduated from Aviation School of Medicine at Randolph Air Force Base.

Dr. Henry served in the US Army as a corporal during World War I. He also served in the US Army Medical Corps and the US Army Air Force during World War II. He was a flight surgeon with flying status during World War II. He belonged to the US Air Force Reserves and retired as a lieutenant colonel.

In 1960 Dr. Henry was Chief of the Examiners Training & Control Branch, Medical Certification Division, Bureau of Aviation Medicine, Federal Aviation Agency, in Washington, D.C.

A past president of the Civil Aerospace Aviation Medical Association and of the Texas Air-Medics Association, he was a member of the Bexar County Medical Society, Texas Medical Association, American Medical Association, American Academy of General Practice, National Aeronautic Association, and Aerospace Medical Association. He also belonged to the Quiet Birdmen, Civil Air Patrol, Aircraft Owners and Pilots Association, Flying Physicians Association, Texas Private Fliers Association, and OX5 Club of America.

Active in local affairs, he was a member of the San Antonio Chamber of Commerce, and the Downtown Lions Club. He was a York Rite and Scottish Rite Mason and was a past master of Prospect Hill Lodge No. 1247 AF & AM. He belonged to the San Antonio Consistory, the San Antonio Commandery No. 7, Knights Templar; Prospect Hill Chapter No. 817, Order of Eastern Star; San Antonio Chapter 381, Royal Arch Masons; and Alzafar Temple Mystic Shrine.

Besides holding a commercial rating as an airplane pilot, Dr. Henry pursued the hobbies of hunting, fishing, golf, and vegetable gardening.

Dr. Henry and Bertha Louise Wertheim were married Jan. 15, 1955, in Boerne, Tex. She survives as do three daughters, Mrs. Jack Buckner, Mrs. B. K. Owen, and Miss Mary Alice Henry, and three grandchildren, all of San Antonio; a brother, Charles M. Henry, Rich-

DR. HENRY

ards-Gebaur AFB, Mo.; and a sister, Mrs. Lou Emma Peck, Stamford, Conn.

DR. P. W. DAY

Dr. Proctor Wilson Day, 68, died on Oct. 22 in San Antonio. Cause of death was congestive heart failure and arteriosclerotic heart disease.

Born May 2, 1898, in Oakman, Ala., he was the son of Byrd W. and Della Brown Day. He attended schools in Nixon, Tex., Southwest Texas State College, and the University of Texas. He was graduated from the University of Texas Medical Branch at Galveston in 1922. He was an instructor of anatomy at the Medical Branch in 1922 and 1923. After serving an internship and residency at San Francisco Hospital in San Francisco, Calif., he served another residency in surgery at California State Prison, in San Quentin, Calif., in 1924 and 1925. From 1927 until 1956, he was the chief medical officer at Folsom Prison Hospital in Represa, Calif.

During World War II, Dr. Day was a commander in the US Navy at Treasure Island, Calif., and in Hawaii.

He was a member of Bexar County Medical Society, Texas Medical Association, the American Medical Association, and Industrial Medical Association. He was an elder of Central Christian Church in San Antonio. A Scottish Rite Mason, he belonged to the Alzafar Temple, San Antonio Consistory, San Antonio Rotary Club, and Texas Ex-Students Association.

On Aug. 31, 1932, Dr. Day and Mabel Taylor were married in Berkeley, Calif. She survives, as do four daughters, Mrs. Joan Wooley, Arlington, Mrs. Nancy Liles, Austin, and Diane Day and Sandra Day, both of San Antonio; two brothers, Ike H. Day, Devine, Tex., and Byrd W. Day, San Antonio; and one sister, Mrs. Will Ezell, Nixon.

DR. DAY

DR. W. O. LUEDEMANN

Dr. William O. Luedemann, 90, died on Oct. 3, 1966, in a San Antonio hospital. He had lived in San Antonio since his retirement in 1960.

One of six children of Simon and Emma Windell Luedemann, he was born Feb. 16, 1876, in Washington County near Brenham. He attended Blinn College and Baylor University in Waco. He taught school for two years before entering St. Louis College of Physicians and Surgeons. He received his medical degree in 1889. He practiced briefly in Cedar, Tex., and took postgraduate work in Chicago before moving to Schulenburg in 1901. He practiced there for 59 years, until his retirement.

He was a member of Colorado-Fayette Counties Medical Society and the American Medical Association. Dr. Luedemann was elected to inactive membership in the Texas Medical Association in 1959.

In 1902 he and Miss Alvina Clara Schaefer were married. She survives as do a son, Dr. Waldo Luedemann, San Antonio, and a daughter, Mrs. Jewel White of Houston. A daughter, Mrs. Beulah Young, preceded him in death in 1948.

DR. J. F. McVEIGH

A past president of Tarrant County Medical Society, Dr. Joseph F. McVeigh, 73, died Aug. 25, 1966, in a Fort Worth hospital.

Dr. McVeigh, an internist, was awarded the Gold Headed Cane of the Tarrant County Medical Society in 1961. The cane annually goes to a physician who is considered to be a "doctor's doctor." He is selected by members of the county society on the basis of professional skill, personal integrity, and friendship.

A native of Fort Worth, he was born Jan. 11, 1893, the son of William M. McVeigh, Sr., and Blanche Fielding McVeigh. He attended Fort Worth schools and received a bachelor of arts degree from the University of Texas in 1913. To earn money for medical school expenses he taught chemistry and zoology at Austin High School for two years. After graduating from the University of Texas Medical Branch, he interned at St. Vincent Charity Hospital in Cleveland, Ohio. He returned to Galveston to teach anatomy at the medical branch. He did postgraduate work at Barnes Hospital in St. Louis, Mo.

Dr. McVeigh returned to Fort Worth in 1924 and helped establish the Coffey Clinic. He practiced there until he began his work at St. Joseph's Hospital. At St. Joseph's he was a past chief of staff and medicine, and was dean of the St. Joseph's School of Nursing. A portrait of him now hangs in the entrance of the school, a gift of the alumnae of the St. Joseph's School of Nursing. Dr. Hugh Beaton of Fort Worth established a scholarship fund for the nursing school in Dr. McVeigh's honor.

In addition to the presidency of his county society, Dr. McVeigh was a past president of the Texas Heart Association, Texas Club of Internists, and a past vice-president of the Texas Medical Association. He belonged to the American Medical Association, American College of Physicians, American Heart Association, Texas Academy of Internal Medicine, and Fort Worth Club of Internists.

Dr. McVeigh was a member of All Saints Episcopal Church of Fort Worth. He was a Mason, served as vice-chairman and as a member of the executive committee of the United Fund in Fort Worth, and belonged to River Crest Country Club.

On Jan. 12, 1924, Dr. McVeigh married Dorothy Gillett of Georgetown, in Georgetown.

Survivors include his wife; a daughter, Mrs. Judy Cordell, both of Fort Worth; a sister, Miss Blanche McVeigh of Fort Worth; and two brothers, William M. McVeigh of Dallas, and John K. McVeigh of Anna.

DR. McVEIGH

DR. G. N. LANCASTER

Dr. Gus Nyat Lancaster, 83, died on Sept. 2, 1966, in a Granbury hospital. He was born on Feb. 20, 1884, in Thorpe Springs, the son of Mary Duval and Dr. J. R. Lancaster.

Dr. Lancaster attended schools in Galveston and Dallas, and received his medical education at the University of Texas Medical Branch and Baylor University College of Medicine then located in Dallas. After interning at Harris Hospital in Fort Worth, he began practice in Granbury where he remained until his death.

A member of Erath-Hood-Somervell Counties Medical Society, he was also a member of the Texas Medical Association and the American Medical Association. He belonged to the First Christian Church of Granbury.

Dr. Lancaster is survived by his wife, the former Claude Knider; a daughter, Mrs. Delyghte Landers of Granbury; a son, Lanny Lancaster in the US Air Force; and two sisters, Mrs. Fred Walker of Cleburne, and Mrs. Will Lyle of Dallas.

DR. O. W. GIBBONS

Dallas surgeon, Dr. Olin Welborn Gibbons, 77, died Oct. 9, 1966, in St. Paul's Hospital, Dallas.

Born Sept. 1, 1889, in Waxahachie, he was the son of Luther L. and Sallie Couch Gibbons. He received his bachelor of arts degree from Trinity University then located in Waxahachie. In 1919, he received his medical degree from the University of Texas Medical Branch in Galveston. He interned at St. Paul's Hospital in Dallas. He was a past president of the medical staff and a former chief of surgery there. Dr. Gibbons had practiced in Dallas since 1920 and was in active practice at the time of his death.

DR. GIBBONS

A fellow of the American College of Surgeons, he was a member of Dallas County Medical Society, Texas Medical Association, American Medical Association, Dallas Southern Clinical Society, and Southwestern Surgical Congress.

Dr. Gibbons was a Presbyterian. He belonged to the Dallas Country Club, Dallas Athletic Club, and Delta Sigma Phi fraternity.

He enjoyed stock farming.

Dr. Gibbons and Marguerite Elms were married Sept. 6, 1922, in Dallas. She survives as do three sons, Olin W. Gibbons, Jr., D.D.S.; William E. Gibbons, M.D., and Raymond Robert Gibbons, M.D., all of Dallas; a daughter, Mrs. Ray Bowen of Baton Rouge, La.; three sisters, Misses Opal Gibbons, Elsie Gibbons, and Betty Gibbons, all of Waxahachie; and one brother, Couch Gibbons of Grand Prairie.

DR. C. H. NASH, JR.

Lubbock obstetrician-gynecologist, Dr. Charles Henry Nash, Jr., 49, died unexpectedly Oct. 4, 1966.

He was born on Aug. 25, 1917, in Memphis, Tenn., the son of Charles Henry and Edna Lee York Nash.

Dr. Nash attended schools in Amarillo, and the University of Texas where he received his bachelor of arts degree in 1939. In 1942 he was graduated from the University of Texas Medical Branch in Galveston. After interning at Indianapolis City Hospital in Indianapolis, Ind., he served a residency at John Sealy Hospital in Galveston. He was an instructor at George Washington University in 1949 and was appointed associate medical officer of Gallinger Municipal Hospital and chief medical officer in 1950. Both institutions are in Washington, D.C.

He had practiced in Galveston and then moved to Lubbock in 1952. While in Galveston, he had been an instructor and chief resident in obstetrics and gynecology at the University of Texas Medical Branch.

Dr. Nash served as a captain in the US Army Medical Corps. from 1943 to 1946.

He belonged to the Lubbock-Crosby-Garza Counties Medical Society, Texas Medical Association, American Medical Association, American College of Obstetricians and Gynecologists, and West Texas Ob-Gyn. He was a diplomate of the American Board of Obstetrics and Gynecology.

He is survived by his wife, Penny; two sons, Charles Henry Nash, III, and William Alan Nash; and a daughter, Mary Ann Nash, by a previous marriage, all of Lubbock.

DR. M. H. LATIMER

Dr. Mark H. Latimer, 66, died Oct. 2, 1966, in Memorial Baptist Hospital in Houston. He had lived in Houston since 1927.

Born in Meridian, Tex., on Mar. 9, 1900, he was the son of Henry Lloyd and Elizabeth Ellis Latimer. He attended schools in Meridian and received his associate in arts degree from Meridian Junior College, his bachelor of business administration from the University of Texas, and his medical degree from the University of Texas Medical Branch. Dr. Latimer interned at Jefferson Davis Hospital in Houston, and served a residency in surgery at Methodist Hospital in Houston. In 1946 he received a master of surgery degree from the University of Pennsylvania in Philadelphia. He had been an associate professor of surgery at Baylor University College of Medicine.

Dr. Latimer was a private in the US Army from 1918 to 1919.

A fellow of the American College of Surgeons, he belonged to the Harris County Medical Society, Texas Medical Association, American Medical Association, and the Southwestern Surgical Congress. He had been president of the medical staff of Memorial Baptist Hospital and of the Postgraduate Medical Assembly of South Texas.

DR. LATIMER

Dr. Latimer was a member of River Oaks Baptist Church.

He is survived by his wife, the former Charlotte Knowd, whom he married on March 28, 1929, in Temple.

DR. E. D. RICE

Tyler physician, Dr. Elbert Dean Rice, 85, died Sept. 25, 1966, in a local hospital. He had been named the General Practitioner of the Year for 1960 by the Texas Medical Association.

The fifth of seven children, Dr. Rice was born about four miles south of Tyler. He obtained his early schooling at Rice community school, and attended high school in Tyler. He worked two and one-half years as a rural mail carrier to earn money for college. He was graduated from Westminster College in Tehuacana, Tex., in 1909, and the University of Louisville School of Medicine in 1913. He served an externship at Louisville General Hospital his senior year in medical school.

On June 3, 1913, Dr. Rice and Blanche Chapin were married in Holder, Ill. They moved to Hubbard, Tex., where he practiced until October, 1914. They returned to Holder, and he practiced with his father-in-law for two years. In 1916 Dr. Rice and his family returned to Texas, and he began his 50-year practice in Tyler.

Dr. Rice served as a first lieutenant in the US Army Medical Corps during 1918 and 1919.

He had served as county health officer for about four years, had been president of Smith County Medical Society in 1921 and 1955, and had served on the Selective Service Examination Board during World War II.

Dr. Rice was a member of the Marvin Methodist Church where he was on the Board of Stewards and a member of the choir for over 40 years. He was a member of Saint John's Masonic Lodge and was both a Scottish Rite and York Rite Mason.

An honorary member of the Texas Medical Association, he belonged to Smith County Medical Society, American Medical Association, Texas Academy of General Practice, and the American Academy of General Practice.

His wife died in February of this year. He is survived by a son, Dr. Robert D. Rice of Tyler; one brother, Clifton Rice of Del Rio, and three grandchildren.

DR. RICE

DR. H. G. STEPHENSON, JR.

Dr. Henry G. Stephenson, 45, died July 29, 1966, in San Angelo.

Born Sept. 8, 1920, in Fort Worth, he was the son of Henry Grady and Elsie Huffman Stephenson. He attended schools in Fort Worth, and received a bachelor of science degree from Abilene Christian College in 1943. In 1947 he received a bachelor of arts degree from Texas Christian University. He was graduated from the University of Texas Medical Branch in 1951. He served in the US Navy from 1943 to 1946, and in 1951 he returned to the Navy and served as a lieutenant in the Medical Corps until 1953. While in the Medical Corps, he interned at the US Naval Hospital in Pensacola, Fla. He practiced in Fort Worth, Harlingen, and Weslaco before serving a residency in psychiatry at the Austin State Hospital. He then practiced in Dell City and San Angelo.

Dr. Stephenson belonged to the Tom Green Eight Counties Medical Society, Texas Medical Association, and the American Medical Association. He was a member of Nu Sigma Nu medical fraternity. He was an Episcopalian.

Dr. Stephenson is survived by his wife of Dell City, whom he married Sept. 5, 1948, in Fort Worth. Other survivors are three sons, Michael Owen, Henry Buckland, and David Grady Stephenson, all of Dell City; two daughters, Rebecca Ann and Lizabeth Diane Stephenson, Dell City; his father, Henry G. Stephenson, Sr., his mother, Mrs. Elsie Stephenson, both of Fort Worth; a brother, Billy Charles Stephenson, San Diego, Calif.; and two sisters, Mrs. Mary Nell Brothers, Fort Worth, and Mrs. Geraldine Beaver, Borger.

DR. T. T. FROST

Dr. Thomas T. Frost, 64, died on Oct. 26, in a Temple hospital. He had lived in Temple the past four years where he was chief of laboratory services at the Veterans Administration Hospital.

Dr. Frost was born on Oct. 17, 1902, in Cleveland, Ohio. He was the son of Huron Jackson and Anna Adele Romp Frost. He attended Adelbert College and Western Reserve University and received a bachelor of arts degree in 1926. His medical degree was received from Western Reserve University School of Medicine in 1929. After interning at Grasslands Hospital in Valhalla, N. Y., he did a residency in pathology at City Hospital in Cleveland, Ohio.

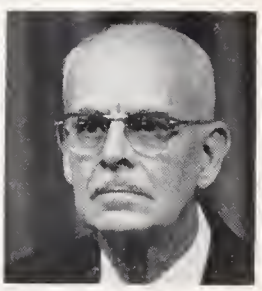
From 1934 to 1936, Dr. Frost was an instructor in pathology at Ohio State University College of Medicine. He practiced in Winston-Salem, N. C., Indianapolis, Ind., and Phoenix, Ariz., before joining the Veterans Administration service. He practiced in Veterans Administration hospitals in Whipple, Ariz., and North Little Rock, Ark., before moving to Temple in 1962.

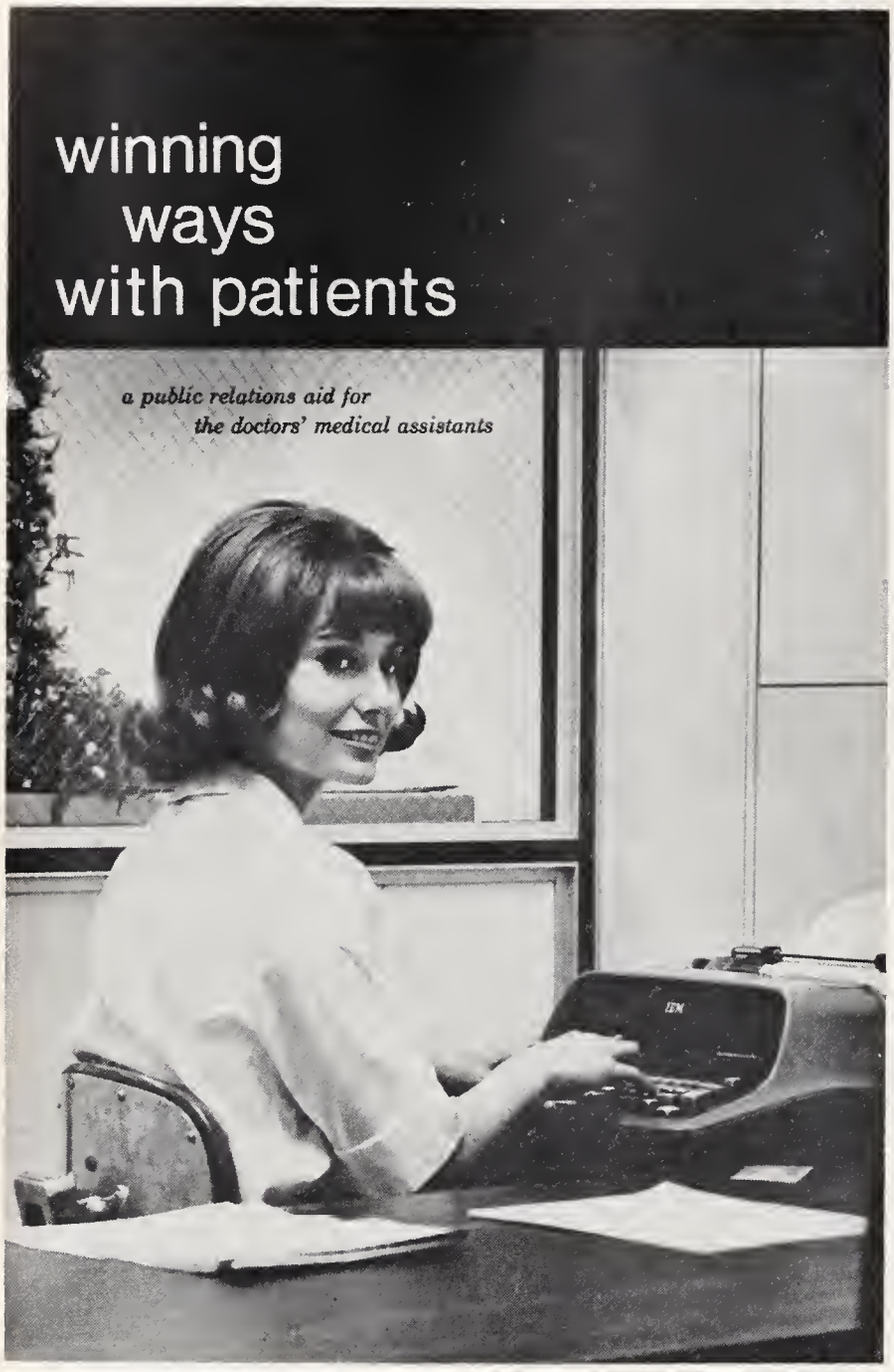
A member of Bell County Medical Society, he belonged to the Texas Medical Association, the American Medical Association, and Sigma Xi honorary fraternity. A diplomate of the American Board of Pathology, he was a fellow of the College of American Pathologists and the American Society of Clinical Pathologists. He was the author of numerous scientific articles, and was listed in *Who's Who in the South and Southwest.*

He was a member of Christ Episcopal Church.

On Jan. 3, 1954, Dr. Frost and the former Juanita C. Corbitt were married in Las Vegas, Nev. She survives, as do a daughter, Annamarie Frost, and a son, Thomas T. Frost, Jr., all of Temple.

DR. FROST





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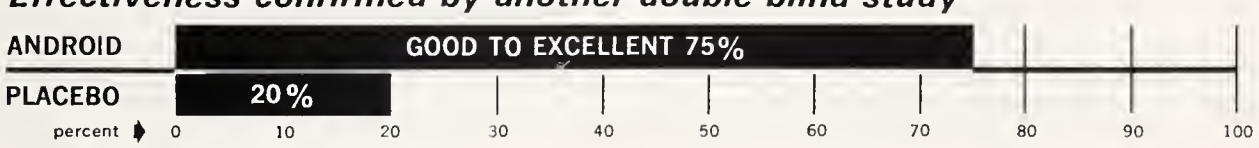

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COMING MEETINGS

American Medical Association, Atlantic City, June 18-22, 1967. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 60610, Exec. Vice Pres.

JANUARY

American College of Radiology, Los Angeles, Jan. 31-Feb. 4, 1967. Mr. W. C. Stronach, 20 N. Wacker, Chicago 60606, Exec. Dir.

Southwest Allergy Forum, Galveston, Jan., 1967. Dr. W. L. Marr, Sealy-Smith Prof. Bldg., Galveston 77550, Pres.

Texas Medical Association, Austin, Legislative Conference, Jan. 21, 1967; Dallas, Annual Session, May 4-7, 1967. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Texas Radiological Society, Fort Worth, Jan. 27-28, 1967. Dr. H. C. Sehested, Medical Arts Bldg., Fort Worth 76102, Sec.

Texas Society of Pathologists, Inc., Austin, Jan. 28-29, 1967. Dr. Jack L. Smith, 3155 Stagg Drive, Beaumont 77701, Sec.

Fifth District Society, San Antonio, Jan. 25, 1967. Dr. Walter Walthall, 1114 South Texas Bldg., San Antonio 78205, Councilor.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1967. S. E. Cockrell, Jr., 202 W. French Pl., San Antonio 78212, Exec. Sec.

National and Regional

American Academy of Allergy, Palm Springs, Calif., Feb. 18-22, 1967. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 53202, Exec. Sec.

American Academy of Dermatology, Inc. Dr. Stanley E. Huff, 636 Church St., Evanston, Ill., Sec.-Treas.

American Academy of General Practice, Dallas, Sept. 15-23, 1967. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 64112, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 29-Nov. 3, 1967. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn. 55901, Exec. Sec.

American Academy of Pediatrics, San Francisco, April 3-5, 1967. Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill. 60201, Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 7-9, 1967. Dr. R. B. Wilson, 200 First St. S. W., Rochester, Minn. 55902, Exec. Sec.

American Association of Plastic Surgeons, Toronto, Can., May 18-21, 1967. Dr. Clarence W. Monroe, 715 Lake St., Oak Park, Ill. 60301, Sec.

American Association for Thoracic Surgery, New York City, April 17-19, 1967. Ada Hanvey, 311 Carondelet Bldg., West, 7730 Carondelet Ave., St. Louis 63105, Adm. Asst.

American Cancer Society, Inc., New York City, Oct. 18-20, 1967. Mr. Charles R. Ebersol, 219 E. 42nd St., New York 10017, Sec.

American College of Allergists, New Orleans, March 22-24, 1967. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo. 80302, Treas.

American College of Chest Physicians. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 60611, Exec. Dir.

American College of Gastroenterology. Mr. D. Weiss, 33 W. 60th, New York 10023, Exec. Dir.

American College of Obstetricians and Gynecologists, Jackson, Miss., Oct. 19-21. Mr. D. F. Richardson, 79 W. Monroe, Chicago 60603, Exec. Sec.

American College of Physicians, San Francisco, April 10-14, 1967. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 19104, Exec. Dir.

American College of Surgeons. Dr. Samuel P. Harbison, 55 E. Erie St., Chicago, Ill. 60611.

American Congress of Physical Medicine and Rehabilitation, Miami Beach, Fla., Aug. 27-Sept. 1, 1967. Mr. Creston C. Herold, 30 N. Michigan, Chicago 60602, Exec. Dir.

American Dermatological Association, Colorado Springs, May 28-June 1, 1967. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn. 55901, Sec.

American Gastroenterological Association, Colorado Springs, Colo., May 25-27, 1967. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C., 27103, Sec.

American Gynecological Society, Phoenix, May 4-6, 1967. Dr. G. W. Douglas, 550 First Ave., New York 10016, Sec.

American Heart Association, San Francisco, Oct. 20-24, 1967. Mr. Rome A. Betts, 44 E. 23rd, New York 10010, Exec. Dir.

American Hospital Association, Chicago, Aug. 21-24, 1967. Mr. James E. Hague, 840 N. Lake Shore Dr., Chicago 60611, Sec.

American Medical Women's Association. Mrs. Gertrude F. Conroy, 1790 Broadway, New York City 10019, Exec. Dir.

American Neurological Association, Atlantic City, June 12-14, 1967. Dr. M. D. Yahr, 710 W. 168th, New York 10032, Sec.

American Proctologic Society, New Orleans, April 17-19, 1967. Miss Harriette Gibson, 320 W. Lafayette, Detroit 48226, Adm. Sec.

American Public Health Association, Inc. Dr. B. F. Mattison, 1790 Broadway, New York 10019, Exec. Dir.

American Society of Anesthesiologists, Las Vegas, Sept. 30-Oct. 4, 1967. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., 60608, Exec. Sec.

American Society of Clinical Pathologists, Las Vegas, Feb. 26-March 3, 1967 (Interim). Dr. William D. Dolan, Arlington Hospital, Arlington, Va., 22203, Sec.

American Surgical Association, Colorado Springs, May 11-14, 1967. Dr. Harris B. Shumacker, Jr., 1100 W. Michigan, Indianapolis 46207, Sec.

American Thoracic Society, Pittsburgh, Pa., May 21-24, 1967. F. W. Webster, 1790 Broadway, New York 10019, Exec. Sec.

Association of American Physicians and Surgeons, Inc., Houston, Oct. 5-7, 1967. Mr. H. E. Northam, 185 N. Wabash, Chicago 60601, Exec. Dir.

Gastrointestinal Research Forum. Dr. Frank Brooks, 3400 Spruce St., Philadelphia, Pa., 19104, Sec.

Gastrointestinal Research Group. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn. 55901, Sec.

International College of Surgeons, April 30-May 5, 1967. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago 60610, Sec.

Medical Society of United States & Mexico, Guadalajara, Mexico, Jan. 25, 1968. Dr. Robert Witzemann, 222 W. Osborn Rd., Phoenix, Ariz., 85013, Sec.

Radiological Society of North America. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb. 68104, Sec.

South Central Association of Blood Banks, Jackson, Miss., March 30-April 1, 1967. Miss Norma M. Bender, 3600 Florida St., Baton Rouge, La. 70806, Sec.

Southern Medical Association. Robert F. Butts, 2601 Highland, Birmingham, Ala. 35205, Exec. Dir.

Southern Psychiatric Association. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southern Surgical Association. Dr. George Yeager, 314 Medical Arts Bldg., Baltimore 21201, Sec.

South Texas District, American College of Surgeons. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio 78205, Sec.

Southwestern Medical Association, El Paso, Feb. 8-10, 1968. Bob Reid, 310 N. Stanton St., El Paso 79901, Exec. Sec.

Southwestern Society of Nuclear Medicine. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas 75219, Sec.

Southwestern Surgical Congress, Phoenix, Ariz., April 10-13, 1967. Dr. Jack A. Barney, 301 Pasteur Building, Oklahoma City, Okla., 73103, Sec.

State

Private Clinics and Hospitals Association of Texas. A. A. Hubbard, Alvarado Community Hospital, Alvarado 76009, Sec.

Texas Academy of General Practice, Austin, June 12-14, 1967. Mr. D. C. Jackson, 1905 N. Lamar, Austin 78705, Exec. Sec.

Texas Academy of Internal Medicine. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth 76104, Sec.

Texas Air-Medics Association, Dallas, May 6-7, 1967. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health. R. W. Shafer, Jr., 4004 N. Lamar Blvd., Austin 78756, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Galveston, March 4, 1967. Dr. James T. Downs, 3707 Gaston Avenue, Dallas 75246, Sec.

Texas Association of Public Health Physicians, Dallas, May 4, 1967. Dr. S. J. Lerro, Box 95, La Marque 77568, Sec.

Texas Chapter, American Academy of Pediatrics. R. L. Mathis, 4119 Montrose Blvd., Houston 77006, Sec.

Texas Chapter, American College of Chest Physicians, Dallas, May 7, 1967. Dr. Paul Richburg, 6011 Harry Hines, Dallas 75235, Sec.

Texas Dermatological Society, Dallas, May 6-7, 1967. Dr. A. C. Ressmann, 714 Medical Arts Bldg., San Antonio 78205, Sec.

Texas Diabetes Association, Dallas, May 7, 1967. Dr. Holbrooke Seltzer, Veterans Administration Hospital, 4500 S. Lancaster, Dallas 75216, Sec.

Texas Division, American Cancer Society, Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 78731, Exec. Sec.

Texas Heart Association, Houston. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Hospital Association, Houston, May 20-24, 1967. O. Ray Hurst, 6225 US Highway 290 E., Austin 78723, Exec. Dir.

Texas Industrial Medical Association, Dallas, May 4, 1967. Dr. C. O. Bruce, P. O. Box 817, Port Neches 77615. Sec.

Texas Neuropsychiatric Association, Dallas, April 7, 1967. Dr. Irvin M. Cohen, 641 Hermann Prof. Bldg., Houston 77025, Sec.

Texas Ophthalmological Association, Dallas, May 5-6, 1967. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio 78205, Sec.

Texas Orthopaedic Association, Dallas, May 6, 1967. Dr. L. Ray Lawson, 2828 Lemmon Ave., Dallas 75204, Sec.

Texas Otolaryngological Association, Dallas, May 5-6, 1967. Dr. C. F. Engelking, 224-34 W. Beauregard, San Angelo 76902, Sec.

Texas Pediatric Society, Houston, Sept. 28-30, 1967. Dr. C. E. Gilmore, 811 Bonham, Paris 75460, Sec.

Texas Physical Medicine and Rehabilitation Society, Dallas, May 4, 1967. Dr. Vincent J. Kitowski, 1333 Moursund Ave., Houston 77025, Sec.

Texas Proctologic Society, Dallas, May 4, 1967. Dr. T. Melton, 636 Hermann Professional Bldg., Houston 77025, Sec.

Texas Public Health Association, San Antonio, Feb. 26-March 1, 1967. J. N. Murphy, Jr., P. O. Box 5192, Austin 78731, Exec. Sec.

Texas Rehabilitation Association, Dallas, May 4, 1967. D. Preston, 3700 Worth, Dallas 75210, Sec.

Texas Rheumatism Association. Dr. Homer R. Goehrs, 801 W. 34th St., Austin 78705, Sec.

Texas Society of Anesthesiologists, Dallas, May 6-7, 1967. Dr. Gerald Mullikin, Longview, Sec.

Texas Society on Aging. Mrs. Edith DeBusk, Rio Grande Bldg., 251 N. Field, Dallas 75202, Sec.

Texas Society of Athletic Team Physicians, Dallas, May 5, 1967. Dr. W. Cone Johnson, Scott and White Clinic, Temple 76501, Sec.

Texas Society of Ophthalmology and Otolaryngology. Dr. Jack B. Lee, 409 Camden St., San Antonio 78215, Sec.

Texas Society of Plastic Surgeons, Dallas, May 6, 1967. Dr. J. R. Smith, 515 M&S Tower, San Antonio 78205, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 201 E. 14th St., Austin 78701, Exec. Sec.

Texas State Board of Medical Examiners. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 76102, Sec.

Texas State Division, International College of Surgeons, Dallas, May 4, 1967. Jack A. Crow, 1825 Hickory, Abilene 79601.

Texas Surgical Society, Fort Worth, April 2-4, 1967. Dr. Jesse E. Thompson, 1008 N. Washington, Dallas 75204, Sec.

Texas Traumatic Surgical Society. Dr. J. C. Long, 805 W. 8th, Plainview 79072, Sec.

Texas Tuberculosis Association, Fort Worth, April 6-8, 1967. Mrs. Pauline K. Matthis, 2406 Manor Rd., Austin 78722, Exec. Dir.

Texas Urological Society, Longview. Dr. Joseph Rappeport, 621 N. Fourth, Longview 75601.

United States-Mexico Border Public Health Association. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso 79901, Sec.

District

First District Society, Pecos, Feb. 4, 1967. Dr. Mario Palafox, 111 N. Fullam, El Paso 77905, Sec.

Third District Society, Lubbock, Oct. 14, 1967. Dr. Louis Devanney, 2209 W. 7th, Amarillo 79106, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin 78701, Sec.

Eighth District Society. Dr. Harry T. Barnes, Jr., 1015 Avenue G, Bay City 77414, Sec.

Ninth District Society. Dr. Bobby Jones, 218 Main Street, Houston 77002, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont 77701, Sec.

Eleventh District Society, Tyler, Spring, 1967. Dr. Lucy Mathis, 107 Redland Lane, Palestine 75801, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple 76501, Sec.

Thirteenth District Society, Fort Worth. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth 76104, Sec.

Clinics

Blackford Memorial Lectures. Dr. W. S. Fry, 2301 S. Austin Ave., Denison 75020, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 13-15, 1967. C. F. Hamilton, 1616 Medical Arts Bldg., Dallas 75201, Dir.

Oklahoma City Clinical Conference. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 73102, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1967. Irma B. Sherwood, 1430 Tulane Ave., New Orleans 70112, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 17-19, 1967. Dr. W. Tom Arnold, 6448 Fannin, Houston 77025, Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 Fifth Ave., Fort Worth 76104, Sec.

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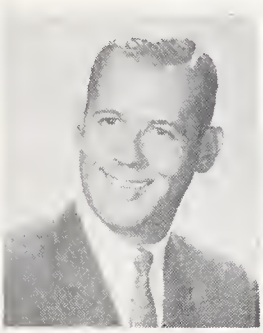

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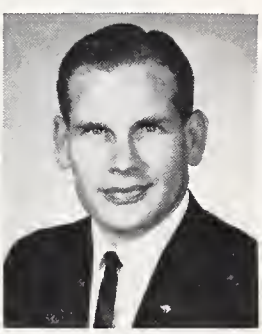

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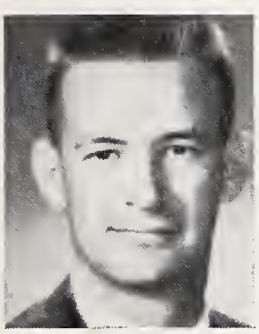

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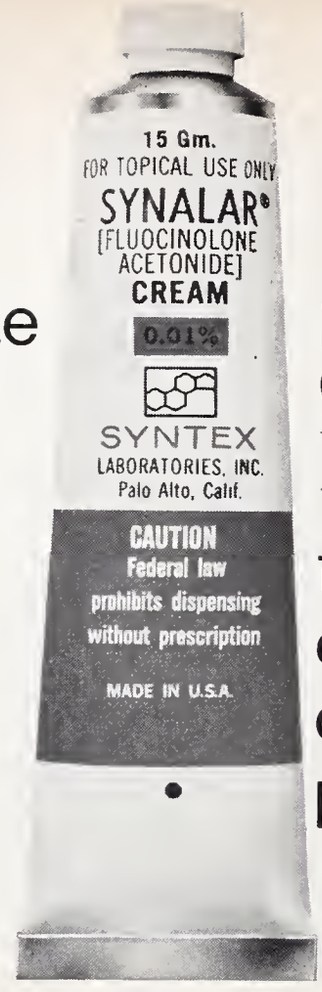

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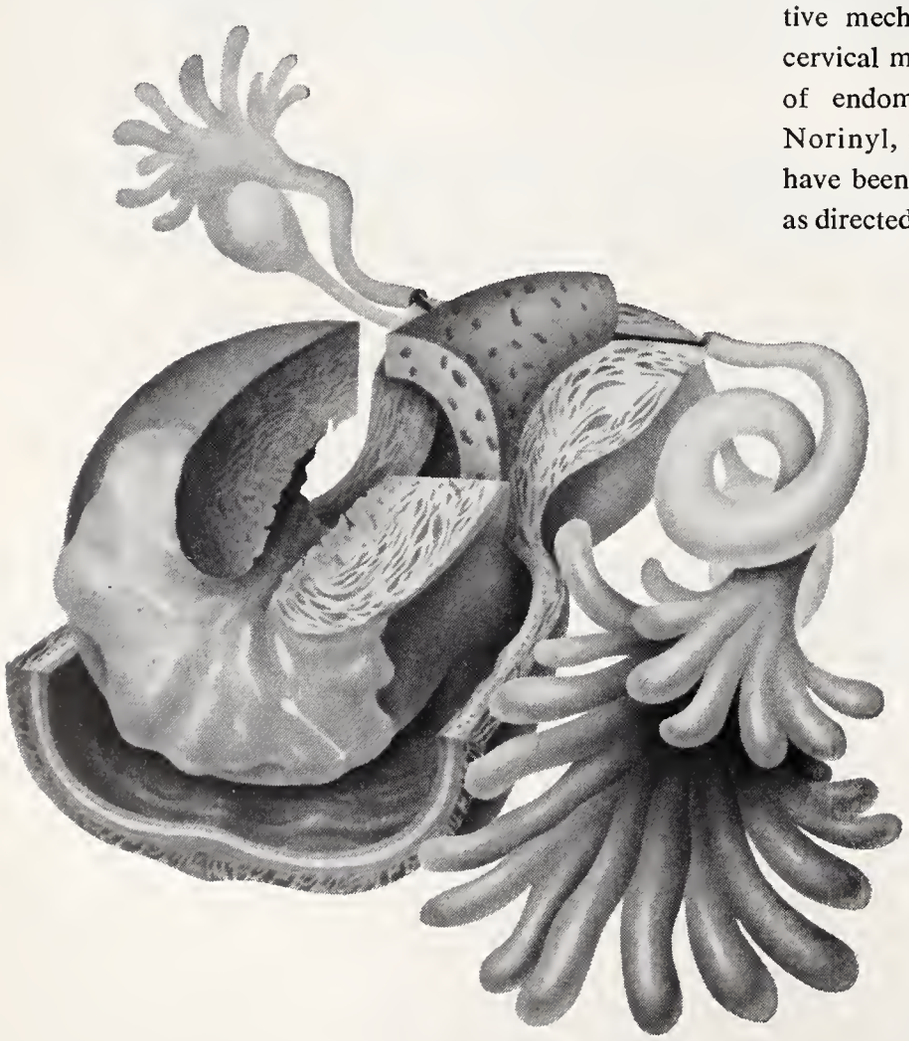

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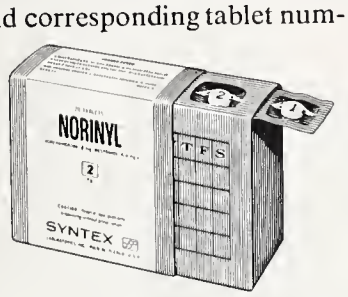

Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb. 29) 1964. 2. Brvans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Coldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Coldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Coldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Coldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

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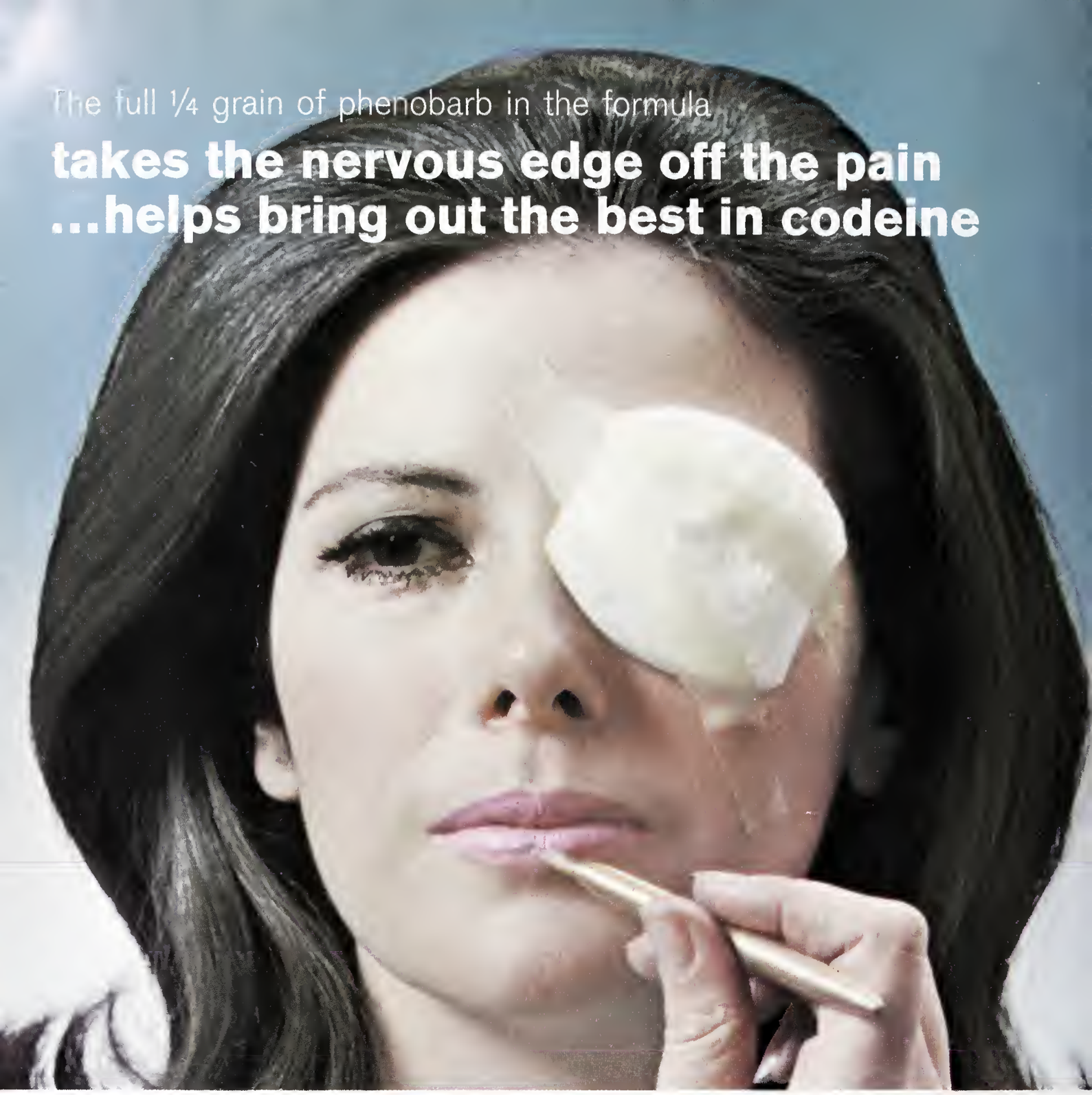

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.

Contraindications: Severe renal impairment; previous hypersensitivity.

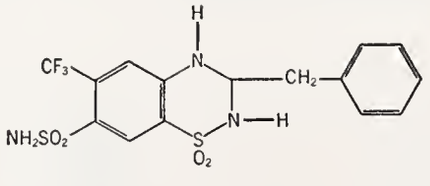
Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c̄ K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: **1.** Southworth, H.: Proc. Soc. Exper. Biol. & Med. *36*:58, 1937. **2.** Mann, T. and Keilin, D.: Nature *146*:164, 1940. **3.** Pitts, R. F., and Alexander, R. S.: Am. J. Physiol. *144*:239, 1945. **4.** Schwartz, W. B.: New England J. Med. *240*:173, 1949. **5.** Friedberg, C. K., in Moyer, J. H., and Fuchs, M.: Edema Mechanisms and Management, Philadelphia, W. B. Saunders Co., 1960, p. 259. **6.** Cumming, J. R.; Tabachnick, E., and Seelig, M., in Moyer, J. H., and Fuchs, M.: op. cit., p. 254. **7.** Werko, L., in Moyer, J. H., and Fuchs, M.: op. cit., p. 188. **8.** Beyer, K. H., Jr., in Moyer, J. H., and Fuchs, M.: op. cit., p. 274. **9.** Maren, T. H., and Wiley, C. E.: J. Pharmacol. & Exper. Therap. *143*:230, 1964. **10.** Earley, L. E., and Orloff, J.: Ann. Rev. Med. *15*:149, 1964. **11.** Fuchs, M., and Mallin, S. R., in Moyer, J. H., and Fuchs, M.: op. cit., p. 276. **12.** Ford, R. V., in Moyer, J. H., and Fuchs, M.: op. cit., p. 290. **13.** cited in Fuchs, M., and Mallin, S. R. (ref. 11): op. cit., p. 283.



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*Need in human nutrition not established.
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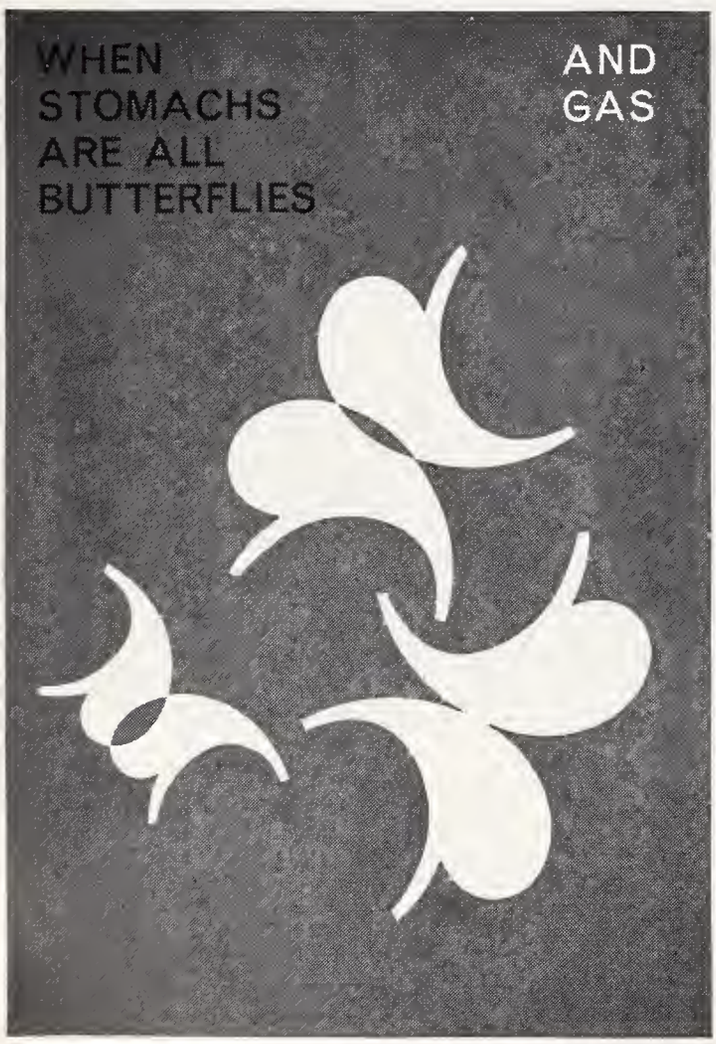

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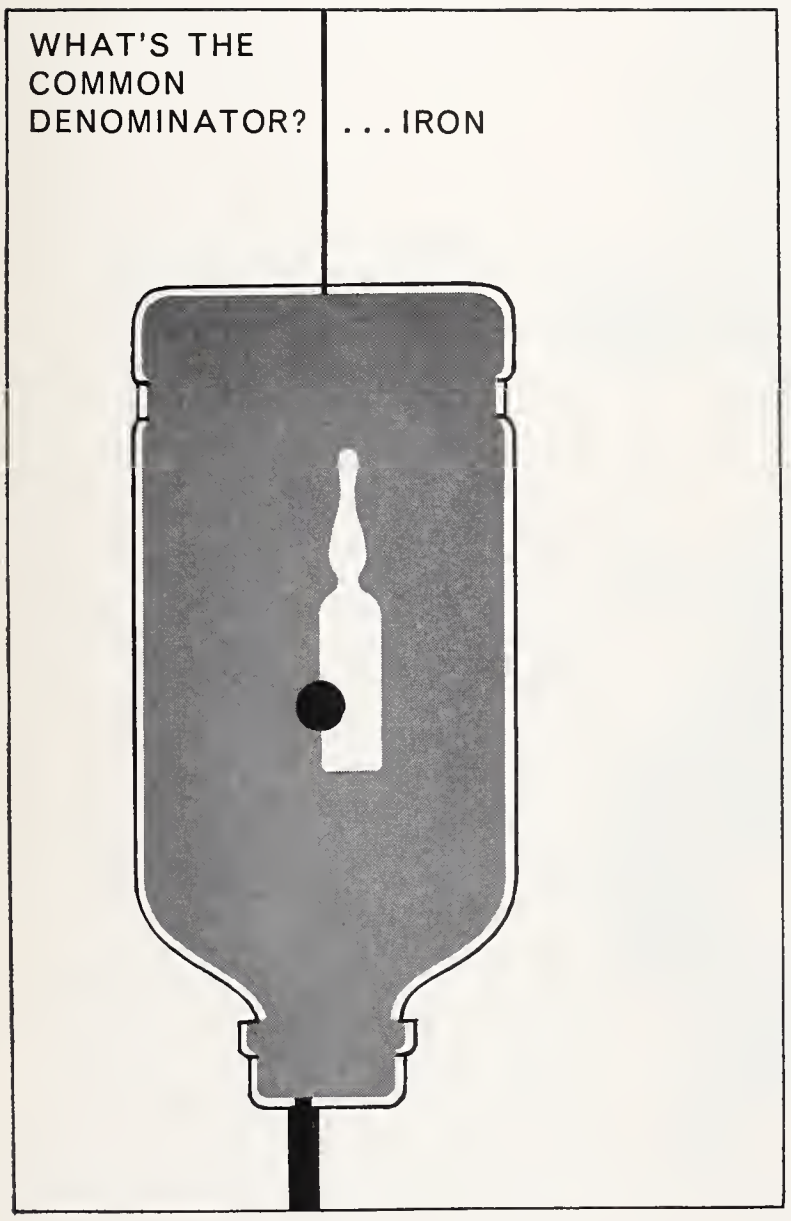

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SIDE EFFECTS: Local and systemic side effects are few. Staining of the skin may occur. Excessive dosage, beyond the calculated need, may cause hemosiderosis. Although allergic or anaphylatoid reactions are not common, occasional severe reactions have been observed, including three fatal reactions which may have been due to Imferon (iron dextran injection). Urticaria, arthralgia, lymphadenopathy, nausea, headache and fever have occasionally been reported.

PRECAUTIONS: If sensitivity to test doses is manifested, the drug should not be given. Imferon (iron dextran injection) must be administered by deep intramuscular injection only. Inject only in the upper outer quadrant of the buttock, not in the arm or other exposed area.

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1. Riese, J. A.: Amer. J. Gastroent. 28:541 (Nov.) 1957

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IMMEDIATELY AVAILABLE: Fully-equipped clinic of recently deceased physician and surgeon. Patients records and same personnel available. Doctor's widow will introduce. Reply to Lowry Clinic, P. O. Box 2005, Laredo, Texas.

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PHARMACIST DESIRES TO PURCHASE a strictly prescription pharmacy in clinic with several physicians, or lease space for same; if not in clinic at least in close proximity. Please reply to Box 117, Texas Medicine, 1801 North Lamar Blvd., Austin, Texas 78701.

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PRQ stands for "Patient Relations Quotient."

Surveys show that the public's opinion of physicians is not as high as it once was. There are areas that physicians can work on to improve the time-honored physician-patient relationship.

Here is a simple test that will help you determine how you handle 10 sensitive areas in the physician-patient relationship:

	T	F
1. My patients usually consider me a friend and confident, as well as the technician who cures their ills.	☐	☐
2. I always give my patients time to talk to me about their ills; I listen attentively, and I take the time to explain diagnosis and therapy in terms they can understand.	☐	☐
3. I take the time to explain tactfully when I am unable to make a house call.	☐	☐
4. I am concerned about my patients' problems in paying for health care; I usually have a frank discussion of fees; I have a fee plaque (available from the AMA—see page 120, this issue) on my wall inviting discussion.	☐	☐
5. I always itemize my bills so that my patients will know exactly what the charges are; I try to work tactfully with my patients to help them catch up on unpaid bills.	☐	☐
6. I train my office staff to schedule appointments so that I do not keep patients waiting more than a few minutes past the appointed time.	☐	☐
7. My reception room is bright and cheery, with up-to-date magazines; a rack of health education pamphlets; comfortable chairs, furniture and equipment; good lighting.	☐	☐
8. My receptionist is neat, cheerful, friendly; she handles telephone calls with a pleasant voice and interested manner; she greets patients graciously and explains to them the reason for my delay in keeping their appointment, if a delay is unavoidable.	☐	☐
9. I often meet my patients outside the office, through my participation in civic affairs, such as serving on the school board, joining a service club, working with the chamber of commerce, and generally conducting myself as a good citizen as well as a physician.	☐	☐
10. I keep abreast of new developments in medicine in my community by regular participation in the affairs of my county medical society, thus enabling me to give better care to my patients.	☐	☐

If you answered "true" to all 10, you're practically a saint. If you answered "true" to 7, your score is good, but could stand improvement. If you answered "true" to 5 to 7, you would be wise to improve your manners. If you answered "true" to less than three, you'd better look out in your waiting room—it may be empty!

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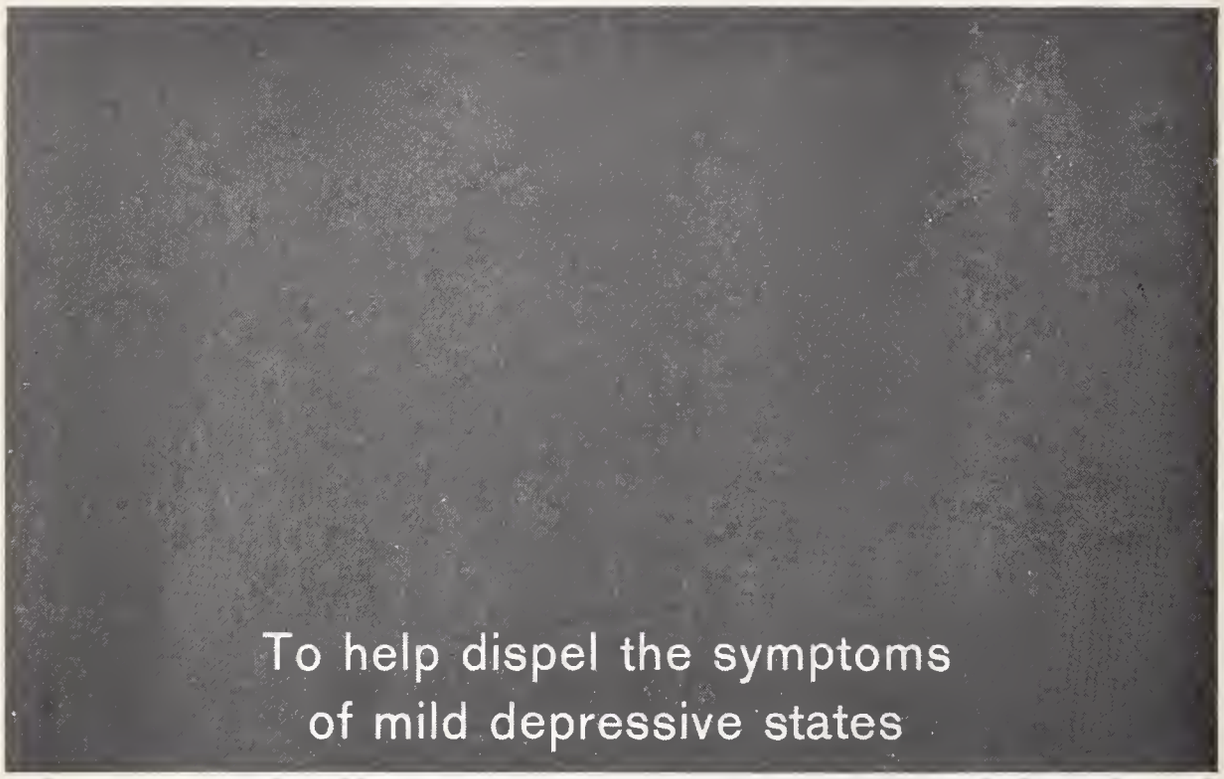

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